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Technical Support Document:

Control of Emissions of Hazardous Air Pollutants from Motor Vehicles and Motor Vehicle Fuels

Assessment and Standards Division
Office of Transportation and Air Quality
U.S. Environmental Protection Agency

Executive Summary

Today's action addresses emissions of hazardous air pollutants from mobile sources and their fuels. In this action, we identify those compounds that should be considered Mobile Source Air Toxics (MSATs), and examine the mobile source contribution to national inventories of these compounds and the impacts of existing and newly promulgated mobile source control programs. We also evaluate whether additional mobile source air toxics controls are technologically feasible at this time, and set new gasoline toxic emission performance standards which require refiners to maintain current levels of overcompliance with RFG and anti-dumping toxic emission performance requirements. Because the technology-forcing standards found in our Tier 2 motor vehicle emissions standards and gasoline sulfur control requirements (Tier 2) and our proposed heavy-duty engine and vehicle standards and on-highway diesel fuel sulfur control requirements (HD2007) would represent the greatest degree of toxics control achievable for vehicles and engines at this time, we do not set additional vehicle-based air toxics controls. Finally, because of our concern about potential health impacts of public exposure to air toxics, today's action establishes a process to continue to conduct research and analysis on mobile source air toxics. Based on the information developed through this research, we will conduct a future rulemaking, to be completed no later than July 1, 2004, in which we will revisit the feasibility and need for additional controls under 202(l)(2).

This Technical Support Document (TSD) describes in greater detail the technical reasoning behind each of the components of today's program. We begin with background information in support of the regulatory decisions for control of mobile source air toxics under Section 202(l)(2) of the Clean Air Act, including a description of air toxics and a review of Agency mobile source emission control programs that relate to mobile source air toxics control. This is followed by a discussion of how we identified our list of Mobile Source Air Toxics. Our selection approach, as described in Chapter 2, is based on identifying those compounds that we know are emitted by motor vehicles and comparing this list to EPA's Integrated Risk Information System (IRIS) database. IRIS is a database of compounds that presents EPA's consensus scientific judgment on the characterization of the potential adverse health effects that may result from exposure to various substances. This process resulted in a list of 21 compounds, including various volatile organic compounds (VOCs) and metals as well as diesel particulate matter and diesel exhaust gases (collectively DPM+DEOG). Chapter 3 contains important health and environmental information for each of those MSATs.

Chapter 4 examines the effectiveness of current and proposed controls in reducing on-highway emissions of these MSATs. Our analysis shows that the programs we currently have in place, including our reformulated gasoline (RFG) program, national low emission vehicle (NLEV) program, Tier 2, and our recently proposed HD2007 rule, are expected to yield significant reductions of mobile source air toxics. Between 1990 and 2020, these programs are expected to reduce on-highway emissions of benzene, formaldehyde, 1,3-butadiene, and acetaldehyde by 67 percent or more. In addition, we expect to see on-highway diesel PM

emission reductions of over 90 percent.

Chapter 5 reviews what we know about ambient concentrations and exposures associated with emissions of mobile source air toxics. We look at monitoring and modeled data on ambient concentrations of five of the 21 mobile source air toxics. These compounds are benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and diesel PM. We also review results of on-highway vehicle inhalation exposure assessment prepared by EPA. The exposure estimates for gaseous air toxics are compared to estimates of the highway vehicle contribution to modeled ambient concentrations. We also discuss what we know about inhalation exposures from micro-environmental sources.

Chapters 6 and 7 contain our analysis regarding additional toxics controls for vehicles and fuels. The analysis in Chapter 6, which focuses on vehicle-based controls, leads us to conclude that it is not appropriate at this time to propose more stringent standards than the technology forcing standards found in our Tier 2 rule and our proposed HD2007 rule. Chapter 7 provides our analysis for fuel-based controls. Our new toxic emissions performance requirement directs refiners to maintain the current levels of over-compliance with RFG and anti-dumping toxic emissions performance requirements. Because the proposed standards for each refiner are the same as the 1998-2000 toxics emission performance level for that refiner, the proposed standards are expected to impose only negligible costs.

Finally, in Chapter 8 we describe our current nonroad engine emission control programs and present our estimates of the impacts of these programs on future air toxics inventories. In this chapter, we also highlight the significant uncertainty and several of the data gaps that exist with respect to toxics emissions from nonroad engines.

List of Acronyms

µg/m^3	micrograms per cubic meter
AIRS	Aerometric Information Retrieval System
API	American Petroleum Institute
ASPEN	Assessment System for Population Exposure Nationwide
ATSDR	Agency for Toxic Substances and Disease Registry
CAA or the Act	Clean Air Act
CalEPA	California Environmental Protection Agency
CARB	California Air Resources Board
CASAC	Clean Air Scientific Advisory Committee
CEP	Cumulative Exposure Project
CG	conventional gasoline
CHAD	Consolidated Human Activity Database
CMB	chemical mass balance
CO	carbon monoxide
CPIEM	California Population Indoor Exposure Model
DHHS	Department of Health and Human Services
DOC	diesel oxidation catalyst
EPA or the Agency	U.S. Environmental Protection Agency
FAA	Federal Aviation Administration
FTP	federal test procedure
g/bhp-hr	grams per brake-horsepower-hour
GVWR	gross vehicle weight rating
HAP	hazardous air pollutant
HAPEM	Hazardous Air Pollutant Exposure Model
HAPEM-MS	Hazardous Air Pollutant Exposure Model for Mobile Sources
HC	hydrocarbon
HD2007	heavy-duty engine and vehicle standards and diesel sulfur controls
HD-FTP	heavy-duty federal test procedure
HDE	heavy-duty engine
HDV	heavy-duty vehicle
HLDT	heavy light-duty truck
I/M	inspection/maintenance

IARC	International Agency for Research on Cancer
ICAO	International Civil Aviation Organization
IRIS	Integrated Risk Information System
IUATS	Integrated Urban Strategy (also called Urban Air Toxics Strategy)
LDT	light-duty truck
LDV	light-duty vehicle
LEV	low emission vehicle
LLDT	light light-duty truck
MARPOL	International Convention on the Prevention of Pollution from Ships
MDPV	medium-duty passenger vehicle
MRL	minimum risk level
MSAT	mobile source air toxic
MTBE	methyl *tert* butyl ether
NAAQS	National Ambient Air Quality Standards
NATA	National Air Toxic Assessment
NESCAUM	Northeast States for Coordinated Air Use Management
NIPER	National Institute for Petroleum and Energy Research
NLEV	national low emission vehicle
NMHC	non-methane hydrocarbons
NMOG	non-methane organic gases
NOx	oxides of nitrogen
NPC	National Petroleum Council
NPRA	National Petrochemical & Refiners Association
NPRM	Notice of Proposed Rulemaking
NTE	not-to-exceed
NTI	national toxics inventory
OAQPS	Office of Air Quality Planning and Standards
OBD	on-board diagnostics
OMB	Office of Management and Budget
ORD	Office of Research and Development
ORVR	on-board refueling vapor recovery
OTAQ	Office of Transportation and Air Quality
PADD	Petroleum Administrative Districts for Defense
PAH	polycyclic aromatic hydrocarbon compounds

PCM	powertrain control module
PM	particulate matter
POM	polycyclic organic matter
PTD	product transfer document
R&D	research and development
REL	reference exposure level
RFA	Regulatory Flexibility Act
RfC	reference concentration for noncancer effects
RfD	reference dose for noncancer health effects
RFG	reformulated gasoline
RVP	Reid vapor pressure
SAB	Scientific Advisory Board
SBA	U.S. Small Business Administration
SBARP or the Panel	Small Business Advocacy Review Panel
SBREFA	Small Business Regulatory Enforcement Fairness Act
SCAQMD	South Coast Air Quality Management District (California)
SFTP	supplemental federal test procedure
SI	spark ignited
SIC	Standard Industrial Classification
SIGMA	Society of Independent Gasoline Marketers of America
SIP	State Implementation Plan
SOF	soluble organic fraction
SRP	scientific review panel
TAC	toxic air contaminant
TAP	Technical Analysis Plan
TEAM	Total Exposure Assessment Methodology study
THC	total hydrocarbons
Tier 2	tier 2 motor vehicle emission standards and gasoline sulfur controls
TOG	total organic gases
TSCA	Toxic Substances Control Act
TSD	technical support document
TWC	three-way catalyst
ULEV	ultra-low emission vehicles
VMT	vehicle miles traveled
VOC	volatile organic compound

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Chapter 1: Introduction

The purpose of this Technical Support Document (TSD) is to provide background information in support of the regulatory decisions for control of mobile source air toxics under Section 202(l)(2) of the Clean Air Act. Chapter 1 begins with a "roadmap" of the organization of this document. This is followed by a brief overview of air toxics (what they are, their general health and environmental effects, and their sources) and a summary of some of the Agency's air toxics studies. The remainder of Chapter 1 reviews our mobile source emission control programs that relate to mobile source air toxics control.

A. Roadmap of This Document

Unlike the provisions for the control of toxic air pollutants from stationary sources found in Section 112 of the Act, Section 202(l)(2) does not specify which compounds should be controlled. Chapter 2 of this TSD describes the approach we took to identify a list of 21 mobile source air toxics (MSATs). Chapter 3 provides background information about the potential health impacts of these compounds, including how they enter the environment and how they can affect human health.

Chapter 4 provides estimates of highway motor vehicle emissions ("emission inventories") of these compounds. We present baseline inventory information for 1996 that compares motor vehicle inventories to overall national inventories of emissions from both mobile and stationary sources. This chapter also contains our estimates of the reductions in emissions we can expect when our current and proposed on-highway emission control programs are fully phased-in. We estimate that by 2020 highway inventories of certain key MSATs will decrease by as much as 67 percent from 1990 levels. This chapter also describes the methods we used to estimate these emission inventories and reductions.

In Chapter 5 we review existing information on ambient concentrations of toxic compounds from both monitoring and modeling efforts. We also present estimates of the human exposure to highway motor vehicle emissions of these compounds. This chapter also describes the methods we used to estimate the ambient concentrations and exposure, along with associated limitations and uncertainties.

The next two chapters of this TSD contain our analysis of MSAT controls beyond those mobile source emission controls that are already in place. Chapter 6 addresses vehicle-based controls and concludes that our Tier 2 light-duty vehicle and our recently promulgated heavy-duty engine standards represent the most stringent controls feasible for motor vehicle emissions of air toxics at this time. Chapter 7 contains our analysis of fuel-based controls. In it we present our rationale for our gasoline toxic emission performance requirement. We also discuss the challenges to other types of fuel-based air toxics controls.

Finally, in Chapter 8 we describe our current nonroad engine emission control programs and present our estimates of the impacts of these programs on future air toxics inventories. In

this chapter, we also highlight the significant uncertainty and several of the data gaps that exist with respect to our estimates of toxics emissions from nonroad engines.

B. Brief Overview of Air Toxics

This section contains a brief overview of what air toxics are, their health and environmental effects, and their sources. This summary will help ensure that the remaining discussion is based on a common understanding of the nature of the air toxics problem.

- What are air toxics?

Air toxics, which are also known as "hazardous air pollutants" or HAPs, are those pollutants known or suspected to cause cancer or other serious health or environmental effects. They include pollutants like benzene, perchloroethylene, methylene chloride, heavy metals like mercury and lead, polychlorinated biphenyls (PCBs) and dioxins. 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 other geographic areas. Some can persist for considerable time in the environment and/or bioaccumulate in the food chain.

- What are the sources of air toxics?

There are literally millions of sources of air toxics, including: major stationary sources[1] such as large industrial complexes like chemical plants, oil refineries and steel mills; small (area) stationary sources[2] such as dry cleaners, gas stations, and small manufacturers; and mobile sources such as cars, trucks, buses, and nonroad vehicles such as construction and farm equipment.

- What health and environmental effects do air toxics cause?

Hazardous air pollutants can cause many adverse health effects. Many of these substances are known or suspected to be human carcinogens. Some of these chemicals are known to have negative effects on people's respiratory, neurological, immune, or reproductive systems. Some chemicals pose particular hazards to people with preexisting illnesses, or those of a certain age or stage in life, such as children or the elderly.

- What are the Urban HAPs?

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[1] Under §112(a)(1) of the Act, major stationary sources are sources that emit, or have the potential to emit, 10 tons per year or more of any one HAP or 25 tons per year or more of a combination of HAPs.

[2] Area sources are those stationary sources that are not major sources.

The urban HAPs are the 33 compounds that have been identified by the Agency in the Integrated Urban Air Toxics Strategy (IUATS), published July 19, 1999 (64 FR 137, 38706), as those HAPs posing the greatest threat to human health in the largest number of urban areas. These compounds are a subset of the 188 compounds listed in Section 112(b) of the Clean Air Act. The 33 urban HAPs are listed in Table I-1. The IUATS is described in greater detail in Section C.2., below.

Table I-1
List of Urban HAPs for the Urban Air Toxics Strategy

Acetaldehyde[1]	Coke oven emissions	Mercury compounds[1]
Acrolein[1]	1,2-dibromomethane	Methylene chloride (dichloromethane)
Acrylonitrile	1,2-dichloropropane (propylene dichloride)	Nickel compounds[1]
Arsenic compounds[1]	1,3-dichloropropene	Polychlorinated biphenyls (PCBs)
Benzene[1]	Ethyl dichloride (1,2-dichloroethane)	Polycyclic organic matter (POM)[1]
Beryllium compounds	Ethylene oxide	Quinoline
1,3-Butadiene[1]	Formaldehyde[1]	2,3,7,8-tetrachlorodibenzo-p-dioxine (and cogeners and TCDF cogeners)[1]
Cadmium compounds	Hexachlorobenzene	1,1,2,2-tetrachloroethane
Carbon tetrachloride	Hydrazine	Tetrachloroethylene (perchloroethylene)
Chloroform	Lead compounds[1]	Trichloroethylene
Chromium compounds[1]	Manganese compounds[1]	Vinyl chloride

[1] Included in our Mobile Source Air Toxics List (see Chapter 2)

- What are mobile source air toxics?

We use the term “mobile source air toxics,” or “MSATs,” to signify those air toxics emitted by nonroad engines and on-highway motor vehicles. Section 202(l) of the Act, which addresses controls for hazardous air pollutants from motor vehicles and motor vehicle fuels, does not specify which pollutants are to be evaluated as air toxics, other than benzene, formaldehyde, and 1,3-butadiene. As a result, we must develop a list of compounds to be addressed. Using the methodology described in Chapter 2, we identified 21 MSATs. These are listed in Table I-2 below.

Of our 21 MSATs, thirteen are also included on the list of urban HAPs for the Integrated Urban Air Toxics Strategy (see above). Of the remainder, all but one are specifically identified

in the CAA Section 112(b) HAP list. Diesel particulate matter and diesel exhaust organic gases (DPM+DEOG) is not included in these other two lists because this pollutant was not included by Congress in the Section 112(b) HAP list and, consequently, was not included in the group of pollutants that were considered for inclusion in the urban HAP list. Although not specifically listed in the CAA Section 112(b) list, and consequently with the Urban HAPs, DPM+DEOG is a particular type of emission which is composed of many listed HAPs, including chemicals that fall into the group of POM chemicals, as well as some HAP metals and volatile organic compounds. In the IUATS, we specifically recognized it as a pollutant of concern in urban areas.

Table I-2
List of Mobile Source Air Toxics (MSATs)

Acetaldehyde[4]	Ethylbenzene	Naphthalene
Acrolein[4]	Formaldehyde[4]	Nickel Compounds[1,4]
Arsenic Compounds[1,4]	n–Hexane	POM[3]
Benzene[4]	Lead Compounds[1,4]	Styrene
1,3-Butadiene[4]	Manganese Compounds[1,4]	Toluene
Chromium Compounds[1,4]	Mercury Compounds[4]	Xylene
Dioxin/Furans[2,4]		
Diesel Particulate Matter & Diesel Exhaust Organic Gases	MTBE	

[1] Although the different metal compounds differ in their toxicity, the on-road mobile source inventory contains emissions estimates for total metal compounds (i.e., the sum of all forms).
[2] This entry refers to two large groups of chlorinated compounds. In assessing their cancer risks, their quantitative potencies are usually derived from that of the most toxic, 2,3,7,8-tetrachlorodibenzodioxin.
[3] Polycyclic Organic Matter includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 100 degrees centigrade. A group of seven polynuclear aromatic hydrocarbons, which have been identified by EPA as probable human carcinogens (benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, 7,12-dimethylbenz(a)anthracene, and indeno(1,2,3-cd)pyrene) are sometimes used as surrogates for the larger group of POM compounds.
[4] Although the different metal compounds differ in their toxicity, the on-road mobile source inventory contains emissions estimates for total metal compounds (i.e., the sum of all forms).

- How are air toxics from mobile sources formed?

Mobile source air toxics come from four sources. First, some air toxics are present in fuel and are emitted to the air when the fuel evaporates 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, mobile source air toxics 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. DPM+DEOG emissions, as well as formaldehyde, acetaldehyde, and 1,3-butadiene, are also by-products of incomplete combustion. Third, some compounds, like 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. They can also be present in fuel additives.

C. Other Air Toxics Activities

This section describes the most important of these research and regulatory programs. The first of these is the study required pursuant to Section 202(l) of the Act. That section calls on EPA to study the need for and feasibility of controlling toxic air pollutants associated with motor vehicles and motor vehicle fuels. The study is to focus on those categories of emissions that pose the greatest risk to human health or about which significant uncertainties remain. The Act specifies that, at a minimum, the study focus on emissions of benzene, formaldehyde, and 1,3-butadiene. This study was completed in 1993, and is summarized below. An update of the inventory and exposure data was completed in 1999, and is also summarized below.

In addition, as we developed and prepared this mobile source air toxics rule, we worked in the context of two other important activities that are ongoing at the Agency. These are EPA's Integrated Urban Air Toxics Strategy (IUATS) development and the National Air Toxics Assessment (NATA) activities. Because these two programs are also important parts of our efforts to reduce toxics emissions from all sources, this section contains a brief summary of their key components as well as a description of the Report to Congress that was prepared pursuant to Section 112(k) of the Act. Interested readers are encouraged to visit EPA's Toxics website for more information about these programs (www.epa.gov/otaq/toxics.htm). Finally, EPA's Office of Research and Development has developed a draft Air Toxics Research Strategy (ATRS) to assist in improving our understanding of air toxics and to provide a framework to help manage air toxics research objectives and priorities. The ATRS is described briefly below.

1. Motor Vehicle Air Toxics Studies

In 1993, EPA released a study of motor vehicle-related air toxics in compliance with Section 202(l)(1) of the Clean Air Act.[3] 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, diesel particulate matter, 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

[3]EPA, 1993. Motor Vehicle-Related Air Toxics Study. Report No. EPA 420-R-93-005. This report can be accessed at http://www.epa.gov/otaq/toxics.htm.

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 LEV standards. The study also explored air toxics emissions from alternative fuel vehicles and nonroad engines, but for the baseline cases only and not for control scenarios. It should be noted that this study did not address whether to promulgate air toxics standards or suggest what those standards should be. That discussion was deferred to a future rulemaking under Section 202(l)(2). An evaluation of the costs or benefits of the control scenarios examined for on-highway engines and vehicles was also deferred to a future Section 202(l)(2) rulemaking.

We sought peer review of this study in 1994.[4] The comments from the peer review included suggestions for improving EPA's exposure modeling and risk assessment methodology. In response to these comments, EPA updated its exposure model for motor vehicle-related air toxics. Also, since 1993, significant new information on vehicle emission rates has been developed as part of the Auto/Oil program, the development of the Complex Model for reformulated gasoline, CARB test programs, and other sources, and much more is known about the impact of fuel properties on toxic emissions. Furthermore, EPA has developed new programs, such as the National Low Emission Vehicle (NLEV) program and Tier 2 standards, which have significant effects on projections of toxic emissions and exposure**.** Finally, EPA has released an updated cancer risk assessment for benzene, a draft reassessment for 1,3-butadiene, and an assessment for diesel exhaust emissions.[5, 6, 7]

In light of this new information, and in response to peer review comments, EPA updated the estimates of emissions and exposure contained in the 1993 study.[8] A slightly different list of compounds was studied, and a different set of scenarios. Specifically, the updated study

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[4]Peer review comments on the 1993 study can be accessed at http://www.epa.gov/otaq/toxics.htm

[5]EPA 1998. Environmental Protection Agency, Carcinogenic Effects of Benzene: An Update, National Center for Environmental Assessment, Washington, DC. 1998. This report can be accessed at http://www.epa.gov/ncea/benzene.htm.

[6]EPA 1998. Environmental Protection Agency, Health Risk Assessment of 1,3-Butadiene. EPA/600/P-98/001A, February 1998. This report can be accessed at http://www.epa.gov/ncea/butadiene.htm

[7]EPA. 1999. Health Assessment Document for Diesel Emissions: SAB Review Draft. EPA/600/8-90/057D Office of Research and Development, Washington, D.C. The document is available electronically at www.epa.gov/ncea/diesel.htm.

[8]Analysis of the Impacts of Control Programs on Motor Vehicles Toxics Emissions and Exposure in Urban Areas and Nationwide (Volumes 1 and 2), November 1999. EPA420-R-99-029/030

considers exposure and risk for six air toxics (acetaldehyde, benzene, 1,3-butadiene, formaldehyde, MTBE, and diesel PM) for four years (1990, 1996, 2007, and 2020). Different control scenarios were modeled to reflect the regulatory controls under consideration at that time. Instead of the baseline and expanded use of reformulated gasoline, and expanded adoption of the California LEV standards, scenarios were chosen to reflect some of the options under consideration for both EPA's Tier 2 motor vehicle emission standards and gasoline sulfur controls rule, and the mobile source air toxics rule and the 2004 heavy-duty engine controls. Like the 1993 study, however, the 1999 update considered only on-highway vehicle emissions and not nonroad emissions. Finally, while the 1993 study evaluated only nationwide exposure and risk, the 1999 update examined exposure at the national level as well as for ten urban areas and 12 geographic regions. Providing even more detail, results were also reported by demographic group. As of the date of this Technical Support Document, EPA is still working on an update of the 1993 risk study. This update will be submitted for peer review and finalized in the next twelve to eighteen months and will also reflect comment received during the peer review process of the original 1993 study.

In the above air toxics studies, there are limitations in how ranges of exposures are modeled or characterized. For instance, the screening models the Agency has used do not consider 'hotspots' for elevated air toxics concentrations. For this reason, EPA has not been able to conduct a complete exposure assessment. Additional discussion of the limitations of these studies are included in Chapter 5 of this Technical Support Document. The Agency also needs to do more work on considering the costs and performance levels of pollution controls on air toxics. These activities will be included in the proposed Technical Analysis Plan discussed later in this TSD. Finally, the peer review comments of the 1999 emissions and exposure study contained many suggestions on how to improve the methodology and modeling techniques. These comments will also be taken into account as part of the Technical Analysis Plan.

2. Integrated Urban Air Toxics Strategy

EPA's Integrated Urban Air Toxics Strategy (IUATS), published July 19, 1999 (64 FR 137, 38706), focuses on reducing the human health threats of air toxics in urban areas. 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 HAPs from many sources. In the IUATS, EPA outlined future actions that we plan to take to reduce emissions of air toxics and improve our understanding of the health threats posed by air toxics in urban areas. The over-arching goal for the IUATS is to reduce cancer and noncancer risks associated with air toxics in urban areas. Also, because air toxics in urban areas may threaten the health of some people more than others, depending on factors such as where they live in relation to toxic sources, we intend to characterize exposure and risk distributions both geographically and demographically. This will include particular emphasis on highly exposed individuals (such as those in geographic hot spots) and specific population subgroups (e.g., children, the elderly, and low-income communities).

The goals of the Strategy reflect both the statutory requirements stated in section 112(k)

of the Act and the goals of 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 indoor air. EPA intends to characterize exposure and risk distributions both geographically and demographically. This will include particular emphasis on highly exposed individuals (such as those in geographic "hot spots") and specific population subgroups (e.g., children, the elderly, and low-income communities)

As a first step in the IUATS, EPA identified 33 of the 188 Section 112(b) toxic air pollutants that EPA concluded pose the greatest threat to public health in the largest number of urban areas (see Table I-1, above). It should be noted that while diesel exhaust emissions are not included as a specific pollutant in the list of 33 urban HAPs, many of the hazardous constituents of diesel exhaust emissions are included among the list of 33, and it is a pollutant that we identified in the IUATS as a concern in urban areas.

The IUATS outlines several steps that EPA will take to reduce urban air toxics and address risks, and as part of the IUATS, EPA has prepared an Action Plan. The key components of the Action Plan are as follows.

- Achieve reductions through regulatory actions and related projects. The strategy presents a framework for reducing air toxics emissions from all types of sources found in urban areas, including mobile sources, major industrial sources, and smaller stationary sources. Today's action contains mobile source-specific toxics regulations. We are also developing programs to reduce emissions from several area source categories (i.e., smaller commercial and industrial operations), and plan to complete regulations to address the new 13 area sources identified in the IUATS by 2004. Regulations are already under development or exist for the 16 other area source categories listed in the IUATS.

- Collaborate with interested parties. We are working with state, local, and tribal agencies, environmental groups, environmental justice communities, and affected industries, including small businesses, to assure that any actions under the IUATS are responsive to health concerns while promoting fairness, encouraging urban redevelopment, and minimizing regulatory burdens.

- Education and outreach efforts. We will make an effort to inform stakeholders about the IUATS and obtain their input on designing programs to implement it.

3. National Air Toxics Assessment Activities

National Air Toxics Assessment (NATA) activities are an important component of the IUATS and EPA's overall goal of reducing exposure to air toxics. These assessment activities include air toxics monitoring, emissions inventory development, exposure modeling, research activities, and risk assessment. Over time, these activities will help us set program priorities, characterize risks, and track progress toward reducing exposure to air toxics. Specifically, our current NATA activities include expanding air toxics monitoring, improving and periodically updating emissions inventories, periodically conducting national- and local-scale air quality, multimedia and exposure modeling, characterizing risks associated with air toxics exposures, and continued research on health and environmental effects and exposures to both ambient and indoor sources of air toxics.

As part of these NATA activities, EPA is now conducting an initial national-scale assessment to demonstrate our approach to characterizing air toxics risks nationwide. This initial screening-level assessment will help to characterize the potential health risks associated with inhalation exposures to the 33 urban HAPs and diesel particulate matter.[9] While such a broad-scale assessment is necessarily limited in the scope of the risks that it can assess quantitatively, and by the uncertainties inherent in the various types of data and methods currently available, it represents an important step in characterizing air toxics risks nationwide. Our initial national-scale assessment includes four major steps:

- Compile a national emissions inventory of 1996 air toxics emissions from outdoor sources of air toxics emissions.

- Estimate 1996 air toxics ambient concentrations across the continental United States (and Puerto Rico and the Virgin Islands) for the 33 urban HAPs and diesel PM; compare modeled ambient concentrations with monitor values.

- Estimate 1996 population exposures across the continental United States (and Puerto Rico and the Virgin Islands) to the 33 urban HAPs and diesel PM.

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[9]It should be noted that NATA will estimate only exposure to diesel particulate matter, and not risk.

- Characterize potential public health risks due to inhalation of the 33 urban HAPs.

In describing what national-scale assessment will include, it is also important to note the potentially important sources and pathways of risks to public health that are beyond the scope of this quantitative assessment. For example, while we recognize that indoor sources of air toxics emissions likely contribute substantially to the total exposures that people experience for a number of these HAPs, assessing these indoor sources of exposure cannot be done on a national scale at this time. Further, for a subset of these HAPs (i.e., those that persist and bioaccumulate in the environment), dietary exposures (e.g., eating contaminated fish) likely contribute much more to the total risk associated with exposure to these pollutants than do the inhalation exposures that will be addressed in this assessment. These and other important aspects of total population exposures to air toxics will be addressed more fully over time as part of our NATA activities as more comprehensive data and assessment tools become available.

Additionally, NATA activities include other key activities that will support further risk characterizations on the local and national level in the future. These include:

- Developing and implementing a plan to characterize the concentrations of ambient air toxics through an expanded monitoring network. Data from existing state and local air monitoring programs will be compiled to summarize our current knowledge about ambient concentrations of air toxics. Existing ambient air toxics monitoring data will be compiled and summarized and then used as a "reality check" on model output.

- Improving existing monitoring networks, guided by data analysis and model predictions, to improve the collection of ambient concentration data for future model evaluations. As the monitoring program matures, trend sites will be established to assess the effectiveness of all of our air toxics control programs.

- Evaluating air toxics on a more local scale (e.g., an urban area) using more refined air quality modeling tools that factor in specific local information such as terrain (e.g., mountainous or flat) and local weather patterns. The results of national and local-scale modeling can be compared to provide a more complete context for the evaluation of air toxics.

- Comparing air toxics inventories from 1990, 1996, and 1999 on a toxicity-weighted basis to help inform assessments of progress toward meeting the IUATS risk reduction goals.

- Recommending tools to state, local and tribal regulatory agencies for evaluating air toxics concentrations, exposures and risk. This will include a comparison of the results from national-scale models to those from more local-scale models.

While there continue to be significant uncertainties and gaps in methods, models, and

data that limit our ability to assess risks to public health and the environment associated with exposures to air toxics, continued research will enable future assessment activities, both at the national screening-level and at more local refined levels, to yield improved assessments of cumulative air toxics risks.

4. Urban Air Toxics Reports to Congress

Section 112(k) of the Clean Air Act requires the EPA Administrator to submit two Reports to Congress on actions taken under the Act that reduce the risk to public health posed by the release of hazardous air pollutants from area sources. The first of these two reports was issued on September 15, 2000 (EPA-453/R-99-007, July 2000). That report expands on much of the information provided in IUATS, such as the methodology for developing the emissions inventory, identifying the 33 urban hazardous air pollutants (urban HAPs), and identifying the area source categories that will be subject to regulation. The report also summarizes existing information on risk assessments that have been conducted in various urban areas. These studies were performed by EPA and various States over the last several years. Taking into consideration the uncertainties and limitations of each study, these assessments provide useful information on the potential nature and magnitude of exposures and health risks in urban areas. Finally, the Report also provides a very detailed discussion of 13 research needs to address in achieving the goals of the IUATS. These needs were identified in the following areas: exposure assessment, health effects, dose-response assessment, risk assessment, risk characterization and risk management. The research needs identified in the Report provide a valuable platform for development of the Air Toxics Research Strategy described below. The Report also provides a summary of ongoing EPA activities to address those needs.

5. Air Toxics Research Strategy

EPA's Office of Research and Development is developing an Air Toxics Research Strategy (ATRS) to provide information to guide the development of the Agency's air toxics research program over the next five to ten years. In particular, it provides an air toxics risk framework, a list of key overarching research questions within that framework, a set of summary research needs associated with each research question, the rationale for the strategy and guiding principles and four groups of air toxics that would be the focus of research for the next 10 years. The Mobile Source Air Toxics identified in today's action are included among the four groups of air toxics identified in the ATRS (e.g., aldehydes, halides, metals, and hydrocarbons/POM). The hydrocarbon/POM category includes benzene and diesel exhaust as well as several other gaseous air toxics. The ATRS will undergo SAB review in 2001.

D. Description of Motor Vehicle Air Pollution Control Programs

In this section, we present a short history of some of EPA's key programs addressing car, truck, and bus tail pipe emission controls, evaporative emission controls, and fuel controls.

1. Mobile Source Control Programs and the Clean Air Act

Our national mobile source emission control program began in the early 1970s, when we issued the first sets of motor vehicle standards to reduce air pollution. These early standards focused on emissions of hydrocarbons (HC), nitrogen oxides (NOx), and carbon monoxide (CO). While they were not designed to address toxics emissions specifically, these standards nevertheless helped to reduce the emission of toxics. Catalytic converters designed to reduce HC and CO emissions also reduce gaseous air toxics, and the removal of lead from gasoline to permit the use of catalytic converters (used to meet the HC and NOx standards) led to a significant reduction in the inventory of toxic lead emissions.

More recently, the 1990 Clean Air Act Amendments added new elements to our mobile source emission control programs. Like the earlier versions of the Act, the primary focus of the mobile source provisions, contained in Title II of the Act, continues to be directed at attainment of the National Ambient Air Quality Standards (NAAQS), primarily for ozone, CO, nitrogen dioxide, and particulate matter (PM). The CAA Amendments led to a set of EPA programs that focus on highway motor vehicles (passenger cars, light-duty trucks, and heavy duty trucks and truck engines), nonroad engines, and their fuels. Section 202(l), by contrast, calls on us to focus specifically on controls for hazardous air pollutants emitted from motor vehicles and their fuels.

Our mobile source emission control programs can take several forms. In some cases, the instructions in the Clean Air Act are very specific and prescribe specific levels of control. In these cases, our role is to promulgate these controls within fairly precise boundaries. For example, §202(g) of the Act sets out specific emission limits for Tier 1 controls for motor vehicles; these requirements went into effect beginning in 1994. Similarly, §211(k) of the Act also sets out specific requirements for gasoline fuel reformulation for certain areas of the country.

In other cases, the requirements of the Act are broader and direct us to consider controls in certain areas but give us discretion in determining the appropriate level. For example, §202(a)(1) of the Act directs us to "prescribe (and from time to time revise) ... standards applicable to the emission of any air pollutant from any class or classes of new motor vehicles or new motor vehicle engines, which ... cause, or contribute to, air pollution which may reasonably be anticipated to endanger public health or welfare." Section 202(a)(3)(A)(i) specifies that those regulations "shall contain standards which reflect the greatest degree of emission reduction achievable through the application of technology which ... will be available for the model year to which such standards apply, giving appropriate consideration to cost, energy, and safety factors associated with the application of such technology." This type of broad language is echoed in other sections of Title II, notably those concerning fuels (§211(c)(1)), nonroad engines (§213) and toxics (§202(l)).

In addition to developing traditional regulatory programs, we have also engaged in collaborative efforts with industry, states, and other outside parties using our authorities under the Clean Air Act. An example is the national low-emission vehicle (NLEV) program, in which we worked with the auto industry and the Northeast states to develop an innovative, voluntary program to put cleaner cars on the road before they could be mandated by programs developed

under the Clean Air Act. Another example is our reformulated gasoline program, where an early part of the regulatory process consisted of a broad "regulatory negotiation" during which many stakeholders participated in crafting the basic elements of the ultimate program.

The remainder of this chapter briefly describes our key mobile source emission control programs and their relationship to air toxics emission control.

2. Passenger Car Tailpipe Emission Controls

Since 1970, emission limits for hydrocarbons (HC), oxides of nitrogen (NOx), and carbon monoxide (CO) from cars have been steadily declining. The history of HC control is particularly important to today's rule because many of the gaseous toxics from motor vehicles and their fuels are hydrocarbons and are thus controlled through controls of HC. (HC emissions are a large component of volatile organic compounds (VOC) emissions; the two terms are generally used interchangeably in the context of motor vehicle emissions.)

Before our regulations, cars emitted 9 grams per mile (gpm) HC, or more. Our HC emission standards in the 1970s and 1980s cut these levels by more than an order of magnitude, to a level of 0.41 gpm in 1980. In 1991, we finalized Tier 1 controls for light-duty vehicles and light-duty trucks to be phased in from 1994 to 1996 (56 FR 25724). In 1998, we developed an innovative, voluntary nationwide program to make new cars, called National Low Emission Vehicles (NLEV), significantly cleaner than Tier 1 cars (63 FR 926). The NLEV program went into effect in the Northeast states in 1999 and will go into effect in the rest of the country in 2001. We recently finalized the Tier 2/Gasoline Sulfur control program with stringent NOx and non-methane organic gas (NMOG) standards for all passenger vehicles (see Chapter 6 of this TSD for a more detailed discussion of the Tier 2/Sulfur control program). Table I.B-1 illustrates the declining standards through the NLEV program that have resulted in VOC and air toxic reductions from car exhaust in the 1970s through the 1990s.[10]

Table I.B-1
Hydrocarbon (HC) Exhaust Emission Standards for Light-Duty Vehicles (gpm)

Year	1972	1975	1980	1994	1999
HC	3.4	1.5	0.41	0.31[1]	0.09[2]

1 The 1994 standard is a NMHC standard.
2 The 1999 standard is a NMOG standard.

We also control HC emissions from cars, and thus emissions of gaseous toxics, through a number of other standards and programs. For example, our requirements controlling carbon

[10] Our programs achieve VOC reductions through standards that limit HC, NMHC, or NMOG. For gasoline vehicles, the slight technical distinctions among these ways of expressing emissions can generally be ignored.

monoxide emissions at cold temperatures also have an important effect in reducing HC emissions at cold temperatures, since strategies that reduce cold CO also reduce cold HC (through such strategies as better control of the air/fuel ratio and quicker catalyst "light-off"). Another example is the Supplemental Federal Test Procedure and standards, finalized in 1996, which better capture actual driving conditions in the test procedures for our control programs (61 FR 54852). We also have programs that track how cars are performing in use. One program is the "on-board diagnostics" (OBD) program, which is required for all cars and light-duty trucks beginning in 1994 (58 FR 9468). Another such program is our state-run inspection and maintenance (I/M) program, in which the individual state programs check whether the emission control system on a vehicle is working correctly. I/M programs are currently in place in over 150 areas (57 FR 52950).

3. Heavy-Duty Truck Tailpipe Emission Controls

We have controlled emissions from heavy-duty engines and vehicles since 1984. These standards are expressed in terms of grams of pollutant emitted for horsepower-hour of work done by the engine (g/bhp-hr). As of 1998, new heavy-duty truck engines must meet standards of 4 g/bhp-hr NOx, 1.3 g/bhp-hr HC, and 0.10 g/bhp-hr PM. In a 1997 rulemaking, we finalized more stringent standards for diesel trucks only. These standards will become effective in 2004 (62 FR 54695). We recently issued a final rule to reaffirm these standards for diesel trucks for 2004, and to adopt separate standards for gasoline trucks (65 FR 59896; see below, Table I.B-3). Table I.B-2 illustrates the declining standards for NOx, PM and HC for heavy-duty trucks since 1984.

Table I.B-2
Heavy-Duty Standards for Diesel and Gasoline[1] Engines (g/bhp-hr)

Year	1984	1988	1990	1991	1993	1994	1996	1998	2004	2005
NOX	10.7 –>		6.0	5.0 –>				4.0	2.4[2]	1.0[3]
PM	N/A	0.60 –>		0.25 –>		0.10 –>				
HC	1.3 –>								2.4[2]	1.0[3]

1. Standards for Gasoline Engines are the same as for diesel, until 2004. See Table I.B-3 for gasoline vehicle standards that will be applicable to complete HD gasoline vehicles beginning in 2005
2. This is a combined NMHC + NOx standard for heavy-duty diesel engines only. Typically, HC emissions for HD diesel engines are in the 0.3 g/bhp-hr range or lower.
2. This is a combined NMHC + NOx standard for heavy-duty gasoline engines only. HC emissions for HD gasoline engines are expected to be in the 0.3-0.4 g/bhp-hr range.

Table I.B-3
Heavy-Duty GasolineVehicle Standards for 2005

Gross Vehicle Weight	NOX	HC[1]
8,000-10,000 lbs	0.9 gpm	0.28 gpm
10,001-14,000 lbs	1.0 gpm	0.33 gpm
14,001 lbs and above	See Table I.B.2	

1. The standards for HC is in the form of NMOG standards. Manufacturers have the option to submit test data in the form of NMHC emissions.

In our recent final rule for 2004 and later heavy-duty engines and vehicles, we also made improvements to the test procedure for heavy-duty diesel engines, including a "not-to-exceed" (NTE) test and a steady-state certification test to ensure that emissions are met under a wide range of operating conditions. This program also extended the on-board refueling vapor recovery (ORVR) program (see discussion, Section 1.B.5, below) to heavy-duty gasoline engines weighing between 8,500 and 10,000 lbs (gross vehicle weight). Also, we extended requirements for on-board diagnostic (OBD) systems to more diesel and gasoline fueled vehicles to help identify any possible failure of components of the emission control system.

Our proposed Heavy-Duty Engine/Diesel Sulfur (2007) program, published June 2, 2000 (65 FR 35430), would set stringent exhaust emission standards for heavy-duty engines and vehicles beyond the 2004 levels starting in 2007, and require reductions in sulfur levels from on-road diesel fuel starting in 2006. We describe this proposed program in greater detail in Chapter 6 of this TSD. In general, we project that the proposed standards would cut HC emission by about one-third, and diesel PM emissions by 90 percent.

4. Emission Control Programs for Buses

In 1993, we finalized the Urban Bus Retrofit/Rebuild Program which is intended to reduce the ambient levels of PM in urban areas (58 FR 21359). The program is limited to buses operating in metropolitan areas with 1980 populations of 750,000 or more, and applies only to 1993 and earlier model year buses whose engines are rebuilt or replaced after January 1, 1995. It requires that these urban buses be retrofitted with improved PM emission controls. Approximately 40 urban areas are affected. In addition, in 1993, we finalized more stringent PM standards that apply to new urban buses (58 FR 15781) as indicated in Table I.B-4.

Table I.B-4
New Urban Bus Standards (g/bhp-hr)

Year	1993	1994	1996
PM	0.10	0.07	0.05

5. Evaporative Emission Controls

Evaporative and refueling emissions are a significant portion of the HC emissions inventory for gasoline-fueled vehicles and trucks (in 1990 more than half of the VOC emissions from light-duty vehicles were evaporative emissions). In 1971, we began testing motor vehicles for evaporative emissions by subjecting test vehicles to typical drive and park conditions. The test procedure measures emissions from fuel evaporation during a simulated parking experience (diurnal emissions) and immediately following a drive (hot soak emissions). Currently, we measure diurnal emissions over a three-day test and also a supplemental two-day test. In 1993, we finalized revised evaporative emission test procedures which apply to light-duty and heavy-duty gasoline vehicles. These procedures were fully phased-in as of 1999 (58 FR 16002). We expect the test procedure to ensure that properly functioning vehicles will effectively control evaporative emissions for most in-use events. The 1993 rule also addressed fuel spitback during refueling with a vehicle test to ensure that no spillage occurs when a vehicle is refueled at a rate of up to 10 gallons (37.9 liters) per minute.

Our current evaporative emission and refueling spitback standards are shown in Table I.B-5 (the Tier 2/Gasoline Sulfur rule further reduced evaporative emission standards, and our new requirements for heavy-duty engines will also reduce evaporative emissions standards for heavy-duty gasoline trucks, as described in Chapter 6 of the TSD).

Table I.B-5
Current Light Duty Vehicles, Light Duty Truck, and Heavy-Duty Gasoline Vehicle Evaporative Hydrocarbon and Refueling Spitback Standards

	Evaporative Hydrocarbons			Refueling Spitback (grams/test)
Category	3 Diurnal + Hot Soak (grams/test)	2 Diurnal + Hot Soak (grams/test)	Running Loss (grams/mile)	
Light-Duty[1]	2	2.5	0.05	1.0
Heavy-Duty to 14,000 lbs GVW	3.0	3.5	0.05	1.0
Heavy-Duty above 14,000 lbs GVW	4.0	4.5	0.05	---

1. Note that we have different standards for light-duty trucks with fuel tanks over 30 gallons.

We have also finalized on-board refueling vapor recovery (ORVR) requirements for light-duty gasoline vehicles (59 FR 16262), and recently extended them to heavy-duty gasoline vehicles between 8,500 and 10,000 lbs (gross vehicle weight) (65 FR 59896). ORVR is a nationwide program for capturing refueling emissions by collecting vapors from the vehicle gas tank during refueling and storing them in the vehicle. The gas tank and fill pipe are designed so that when refueling the vehicle, fuel vapors in the gas tank travel to an activated carbon packed

canister, which adsorbs the vapor. When the engine is in operation, it draws the gasoline vapors into the engine intake manifold to be used as fuel. Table I.B-6 indicates the phase-in periods for ORVR for different size gasoline vehicles.

Table I.B-6
On-Board Vapor Recovery for Gasoline Vehicles Phase-In Periods and Standard

Category	Phase-In	Standard
Light-Duty Vehicles	1998-2000	0.2 g/gallon
Light Light-Duty Trucks (to 6,000 lbs gross vehicle weight)	2001-2003	
Heavy Light-Duty Trucks (6,000 to 8,500 lbs gross vehicle weight)	2004-2006	
Heavy-Duty Gasoline Vehicles (to 10,000 lbs gross vehicle weight)	2005-2006	

6. Fuel Control Programs

The emissions that come out of a vehicle depend greatly on the fuel that goes into it. Fuel composition and type are critical factors in the clean vehicle equation. Since 1990, the Clean Air Act explicitly recognizes that changes in fuels as well as in vehicle technology must play a role in reducing air pollution from motor vehicles.

One of the first and most successful programs to control harmful motor vehicle emissions by changing fuel composition was the removal of lead from gasoline. The lead phase-out began in the mid-1970s in order to enable the use of catalytic converters on cars to meet early HC standards. This resulted in dramatic reductions in ambient lead levels and alleviated many serious environmental and human health concerns associated with lead pollution. The Clean Air Act prohibited the introduction of gasoline containing lead or lead additives into commerce for use as a motor vehicle fuel after December 31, 1995. In February 1996, we finalized an action to implement the ban on leaded gasoline (61 FR 3832). The removal of lead from gasoline has essentially eliminated motor vehicle emissions of this highly toxic substance. The reduction and virtual elimination of lead from gasoline has resulted in significant risk reduction to the public and environment.

In 1990 and 1991, we promulgated regulations to reduce the volatility of gasoline (the basic regulations were promulgated at 55 FR 23658). Like the vehicle-based evaporative and refueling emission control programs discussed above, our gasoline volatility program has reduced VOC emissions by reducing evaporation of gasoline.

The reformulated gasoline (RFG) program (59 FR 7716) resulted from the mandate in the 1990 Clean Air Act Amendments requiring areas of the country with the worst ozone problems to use gasoline that is reformulated to help improve air quality. The RFG program, which began January 1, 1995, contains two phases. On an average basis under the Complex Model, Phase I required emissions reductions from 1990 conventional gasoline baseline gasoline of 16.5 percent for air toxics, 36.6 or 17.1 percent for VOC (depending on the region of the country), and 1.5 percent for NOx (40 CFR 80.41). RFG must also contain a minimum oxygen content of 2 percent by weight, a maximum benzene content of 1 percent by volume, and no lead, manganese, or other heavy metals. At its inception, RFG was required in the nine worst ozone areas, with the provision that other ozone nonattainment areas could voluntarily opt in to the program. Currently 17 states and the District of Columbia participate in the program, with RFG representing about 30 percent of the gasoline sold in the United States. Phase II of the program began January 1, 2000, and contains more stringent emissions reduction requirements. On an average basis, air toxics must be reduced by 21.5 percent, VOC by 27.4 or 29.0 percent (depending on the region of the country), and NO_x by 6.8 percent (summertime) and 1.5 percent (wintertime) from the 1990 conventional gasoline baseline (40 CFR 80.41).

Chapter 2: Identification of Mobile Source Air Toxics (MSATs)

Introduction

There are hundreds of compounds and elements known to be emitted from passenger cars, on-highway trucks, and various types of nonroad equipment, several of which may have serious effects on human health and welfare. In recognition of this fact, Congress instructed EPA, in Section 202(l)(2) of the Act, to set standards for hazardous air pollutants from motor vehicles and their fuels. Except for benzene and formaldehyde which are specifically mentioned in 202(l)(2), the Act does not specify the compounds that should be considered for control. Therefore, the first step in developing a mobile source air toxics control program is to identify the compounds that should be evaluated for possible control measures.

This chapter describes the methodology we used to identify our list of 21 mobile source air toxics (MSATs). A more detailed description of the health effects information for these compounds is provided in Chapter 3.

A. The Methodology Used to Identify Our List of Mobile Source Air Toxics

EPA developed the list of MSATs by first searching for lists of compounds in all available databases and recent (i.e., ten years old or less) studies[11] that speciated the emissions

[11]The list of databases and recent studies we used is provided at the end of this chapter in Appendix I.

from motor vehicles and their fuels. Data for vehicles and engines more than ten years old are considered to be outdated and thus are judged not to be representative of current emissions. We then compared the lists of compounds to the list of toxic compounds in EPA's Integrated Risk Information System (IRIS) database. IRIS is a database of compounds that identifies EPA's consensus scientific judgment on the characterization of the potential serious adverse health effects that may result from a lifetime exposure to the listed compounds. Where feasible, IRIS classifies the carcinogenic potential of the compounds and provides the noncancer reference concentrations or doses, RfC, or RfD, respectively. IRIS also lists compounds for which the Agency has reviewed currently available information and concluded that 1) there are insufficient data to calculate an RfC or RfD for the noncancer hazard potentially posed by the compound(s), and/or 2) there is an absence of sufficient information to identify a cancer hazard.

In our original analysis, we compared the lists of compounds identified in the motor vehicle emission databases and studies with the toxic compounds listed in IRIS and we identified 21 compounds, each of which have the potential to cause serious adverse health effects as reflected in IRIS (e.g., these compounds are known, probable, or possible human carcinogens and/or pollutants for which the Agency has calculated an RfC or RfD).[12] We therefore consider each of these compounds to be MSATs. EPA data suggests that nonroad engines and on-road vehicles emit the same pollutants as motor vehicles and their fuels, so we characterize this list as a list of toxic compounds from mobile sources.

We also compared the lists of compounds emitted from motor vehicles and their sources with other lists or sources of information on toxic substances, and identified two additional substances (propionaldehyde and 2,2,4-trimethylpentane) for which we requested comment. A summary of the public comments received on our MSAT list and our responses is provided below.

B. How we Applied the Methodology to Identify our List of Mobile Sources Air Toxics

In the sections that follow, we describe in more detail our methodology for identifying the list of MSATs and how we applied this methodology.

1. Identifying Pollutants Emitted from Mobile Source

In identifying a list of MSATs, EPA compiled all available databases and recent studies (i.e., ten years old or less) that contain information on the various species of compounds emitted from motor vehicles and their fuels. There are several limitations to these speciation studies that should be noted. To identify the species of compounds emitted from motor vehicles, vehicles and engines are tested on a dynamometer, which is basically a mechanical treadmill for the vehicle/engine. The test vehicle/engine is run through a set pattern of starts, stops, idle, and

[12]A further discussion of the potential cancer and noncancer risks, and other dose-response information for each MSAT can be found in Chapter 3 of the TSD.

acceleration, over a standard quantity of miles. The studies generally follow the U.S. Federal Test Procedure (FTP), though other test cycles may be used. The tailpipe and evaporative emissions are collected under a strict set of guidelines and then taken to a laboratory for analysis. Metal and particulate emissions are collected on particulate filters and analyzed. Analysis of metals usually doesn't specify the actual individual chemical form of the metal, but instead reports the total amount of the identified metal emitted. Particulate samples have routinely been collected for PM10 (all particles less than ten micrometers in diameter) which captures all PM emitted by mobile sources.[13] Once the chemical, metal, and/or particle analysis of the collected material is complete, the results are routinely presented as mass (grams or milligrams) of chemical "x" per mile driven (g/mi) or mass per unit of work accomplished (e.g., g/brake-horse power hour).

Many toxic air pollutants are hydrocarbons (HCs). To identify specific compounds in the complex exhaust mixture, the hydrocarbon component of exhaust is chemically separated (speciated). In addition, the compounds that comprise the particulate phase of mobile source emissions are also chemically speciated. Most test programs that characterize vehicle emissions identify only total hydrocarbons and particulate matter without separating the individual species of hydrocarbons and other elements. As a result, motor vehicle emissions may contain other toxic compounds that are not currently included on our MSAT list because studies have not yet specifically identified these compounds.

In addition, there are limited speciation data for certain vehicle classes. Databases and recent studies reporting emissions from light-duty gasoline vehicles (LDGV), heavy-duty diesel vehicles (HDDV), heavy-duty gasoline vehicles (HDGV), and gasoline nonroad engines are listed Appendix I at the end of this chapter. Data for other vehicle and engine types (e.g., light-duty diesel engines and nonroad diesel engines) either do not exist or are outdated (more than ten years old) and thus are judged not to be representative of current emissions. However, it is unlikely that the lack of recent data for these particular vehicle and engine types would lead us to overlook compounds that should be included on our list of MSATs, because the combustion processes for these missing vehicle and engine types are similar to those for the vehicle and engine types for which we do have data.

This compilation did not include speciation of emissions from alternative-fueled vehicles, which are currently few in number. It should be noted that, depending on the fuel used, these vehicles may also emit unburned ethanol[14] and methanol. Low level ethanol mixtures (10% ethanol and 90% gasoline) are widely used in the United States. Higher level ethanol mixtures (e.g., 85% ethanol) are used as alternative fuel sources in a small number of flexible fuel vehicles.

The speciation studies listed in Appendix I at the end of this chapter provide speciation

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[13] The majority of PM emitted by mobile sources is less than 1 micrometer in diameter.

[14]Ethanol is not currently listed in EPA's Integrated Risk Information System database.

profiles for the gaseous and/or particulate phase of mobile source emissions or fuels. Each study identifies the types of vehicles or engines from which data was collected (e.g., light-duty gas vehicles, heavy-duty diesel engines, nonroad engines, or a mix of various types). The vehicles tested represent a mix of older and newer models, with varying mileage. The type of fuel used was also specified.

In the next section we describe the health effects information that is used to determine which compounds emitted from mobile sources and their fuels may be considered MSATs.

2. Using IRIS to Identify Pollutants with Potential Serious Adverse Health Effects

IRIS is an EPA database of scientific information that contains the Agency consensus scientific positions on potential serious adverse health effects that may result from a lifetime exposure to substances found in the environment. IRIS was initially developed for EPA staff in response to a growing demand for consistent information on chemical substances for use in risk assessments, decision-making and regulatory activities. IRIS currently provides health effects information on over 500 specific chemical compounds.[15]

IRIS contains chemical-specific summaries of qualitative and quantitative health information. IRIS information includes the reference concentration for noncancer health effects resulting from inhalation exposure (RfC), the reference dose for noncancer health effects resulting from oral exposure (RfD) and the carcinogen assessment for both inhalation and oral exposure.

The RfC or RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime. RfCs and RfDs are based on an assumption of lifetime exposure and may not be appropriately applied to less-than-lifetime exposure situations. RfCs and RfDs may be derived for the noncarcinogenic effects of

How Chemicals Are Added to the IRIS Database

Our Office of Research and Development (ORD) maintains IRIS through a scientific consensus and review process that precedes entry of a pollutant into the IRIS database. This process consists of:

- An annual Federal Register announcement of the IRIS agenda and a call for scientific information from the public on the selected chemical substances;
- A search of the current literature;
- Development of health assessment and draft IRIS summaries;
- Internal EPA peer review;
- External peer review;
- Agency consensus review and management approval within EPA;
- Preparation of final IRIS summaries and supporting documents; and
- Entry of summaries and supporting documents into the IRIS database.

[15] EPA IRIS Database, http://www.epa.gov/iris/intro.htm

chemicals whether or not they are also carcinogenic.

The carcinogenicity assessments in IRIS begin with a qualitative weight-of-evidence judgment as to the likelihood that a chemical may be a carcinogen for humans (hazard identification). Using the EPA 1986 Risk Assessment Guidelines, a carcinogen can be classified as a known, probable, or possible human carcinogen (Group A, B or C, respectively). A quantitative assessment is performed depending on the weight-of-evidence and the suitability of the available information regarding a relationship between the dose of a compound and the effect it causes (dose-response data). If suitable dose-response data are available, a quantitative assessment is calculated which can be presented in one or m ore of three ways. If the agent is active upon ingestion, then the slope of a dose-response curve generated from orally exposed animals or humans is calculated. This is referred to as the oral slope factor and is presented as risk per milligram of agent ingested per kilogram of body weight per day. Alternatively, the carcinogenic activity of an agent might be assessed in terms of the concentration in drinking water or air, yielding unit risk estimates of risk per microgram agent per liter of drinking water or risk per microgram agent per cubic meter of air, respectively. The oral slope factor and the unit risk factors are generally characterized as upper-bound estimates of the human cancer risk; the true risk could be higher, but is likely to be lower.

IRIS also lists compounds for which the Agency has reviewed currently available information and concluded that 1) there are insufficient data to calculate an RfC or RfD for the noncancer hazard potentially posed by the compound(s), and/or 2) there is an absence of sufficient information to identify a cancer hazard.

Each reference dose/concentration and carcinogenicity assessment is reviewed by a group of EPA health scientists using consistent chemical hazard identification and dose-response assessment methods. These methods are discussed or referenced in the IRIS Background Documents which are specific to, and referenced in, each individual chemical profile on IRIS. It is important to note that the information in IRIS may be revised by EPA, as appropriate, when additional health effects data become available and new developments in assessment methods are adopted.

It is also important to note that IRIS does not provide situational information on individual instances of exposure. In order to evaluate potential public health risks, the summary health hazard and dose-response information in IRIS must be combined with data on specific exposure situations.

3. List of Mobile Source Air Toxics

In our notice of proposed rulemaking we compared the lists of compounds identified in the motor vehicle emission databases and studies with the toxic compounds listed in IRIS. We identified 21 compounds, each of which have the potential to cause serious adverse health effects as reflected in IRIS (e.g., these compounds are known, probable, or possible human carcinogens and/or pollutants for which the Agency has calculated an RfC or RfD) and in the ongoing agency

scientific assessments.[16] We therefore consider each of these compounds to be MSATs. We received comments on ten proposed MSATs (arsenic compounds, chromium compounds, dioxin/furans, diesel exhaust, n-hexane, lead compounds, manganese compounds, mercury compounds, nickel compounds, and styrene) as well as other compounds (methanol, methyl nitrite, ethanol, propionaldehyde, 2,2,4-trimethylpentane). We have changed the listing for diesel exhaust to diesel particulate matter and diesel exhaust organic gases. A discussion of the comments received on the proposed MSAT list is provided below and the MSAT list is provided in Table II.B-1.

It is important to note that inclusion on the list is not itself a determination by EPA that emissions of the compound in fact present a risk to public health or welfare, or that it is appropriate to adopt controls to limit the emissions of such a compound from motor vehicles or their fuels.[17] The purpose of the list is to provide a screening tool that identifies those compounds emitted from motor vehicles or their fuels for which further evaluation of emissions controls is appropriate. In conducting any such further evaluation, pursuant to sections 202(a) or 211(c) of the Act, EPA would consider whether emissions of the compound cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare. Such an evaluation would also consider the appropriate level of any controls, based on the criteria established in section 202(l)(2). Inclusion of a compound on the MSAT list does not decide these issues, but instead identifies those compounds for which such an evaluation would appear to be warranted.

[16]A further discussion of the potential cancer and noncancer risks, and other dose-response information for each MSAT can be found in Chapter 3 of the TSD.

[17]Note, however, that Congress has expressly directed EPA to adopt controls in accordance with 202(l)(2) for benzene and formaldehyde.

Table II.B-1
List of Mobile Source Air Toxics (MSATs)

Acetaldehyde	Ethylbenzene	Naphthalene
Acrolein	Formaldehyde	Nickel Compounds[a]
Arsenic Compounds[a]	n–Hexane	POM[c]
Benzene	Lead Compounds[a]	Styrene
1,3-Butadiene	Manganese Compounds[a]	Toluene
Chromium Compounds[a]	Mercury Compounds[a]	Xylene
Dioxin/Furans[b]		
Diesel Particulate Matter + Diesel Exhaust Organic Gases (DPM + DEOG)	MTBE	

[a] Although the different metal compounds generally differ in their toxicity, the on-road mobile source inventory contains emissions estimates for total metal compounds (i.e., the sum of all forms).

[b] This entry refers to two large groups of chlorinated compounds. In assessing their cancer risks, their quantitative potencies are usually derived from that of the most toxic, 2,3,7,8-tetrachlorodibenzodioxin.

[c] Polycyclic Organic Matter includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 100 degrees centigrade. A group of seven polynuclear aromatic hydrocarbons, which have been identified by EPA as probable human carcinogens, (benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, 7,12-dimethylbenz(a)anthracene, and indeno(1,2,3-cd)pyrene) are used here as surrogates for the larger group of POM compounds.

In response to public comments we are changing the way we list toxics in diesel exhaust as an MSAT. We believe a better approach is to list diesel particulate matter and diesel exhaust organic gases (DPM + DEOG) as the MSAT. This listing approach is more precise about the components of diesel exhaust expected to contribute to the observed cancer and noncancer health effects and provides a framework for developing regulatory control strategies. Currently available science, while suggesting an important role for the particulate phase component of diesel exhaust, does not attribute the likely cancer and noncancer health effects independently to diesel particulate matter as distinct from the gas phase components. Because the studies cannot separate the health effects of the particulate and gaseous components of diesel exhaust we are listing them together as a single MSAT.

While this listing departs slightly from the approach described above, we believe this is reasonable because 1) there are several nontoxic components of diesel exhaust (e.g., water vapor, nitrogen, oxygen) that we are excluding from the listing, 2) this listing includes the components of diesel exhaust that are likely to contribute to the cancer and noncancer hazard (with the

exception of the gaseous phase criteria pollutants, such as NOx, SO_2 and CO which are subject to National Ambient Air Quality Standards), 3) the more precise listing provides Federal and State government, industry, and public interest groups an ability to focus on the components of diesel exhaust that pose a potential concern for public health, and 4) this focus provides specific targets for emissions reductions should future analysis indicate that additional controls are necessary.

Throughout the TSD we are using diesel PM as the surrogate for the particulate matter and exhaust gas components of diesel exhaust. Data regarding exposure assessment and inventory analysis for specific constituents in the diesel exhaust organic gas phase are available for some compounds (e.g., benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and acrolein) and are presented in Chapters 4 and 5 of the TSD.

We received comments regarding the proposed listing of diesel exhaust that were either strongly supportive of the proposed listing or requested that diesel exhaust be removed from the list of MSATs. Among the supporting comments, one commenter specifically requested that the proposed diesel exhaust listing not be changed to diesel particulate matter, stating that future developments, while controlling particulate matter, could lead to increased risks from the gaseous diesel emissions. Among the comments requesting that diesel exhaust be removed from the MSAT list, recommendations for alternative listing approaches included diesel particulate matter, ambient particulate matter, or the specific components of diesel exhaust for which health effects have been determined.

The IRIS listing and the summary of cancer and noncancer health effects described in the draft Health Assessment Document for Diesel Exhaust are based on studies linking serious adverse health effects to whole diesel exhaust exposure, using diesel particulate matter as the measure of dose. Available science, while suggesting an important role in toxicity for the particle phase component of diesel exhaust, cannot rule out a role for the gas phase components such as semi-volatile organics that are partly in both the gas and particle phases. Accordingly, we are listing the MSAT diesel exhaust as diesel particulate matter and diesel exhaust organic gases (DPM + DEOG). This listing addresses commenters' concerns that diesel exhaust is an imprecise and overly-inclusive designation that does not focus attention properly on the toxic fractions of concern. In addition, the listing of DPM + DEOG provides industry with more specific targets for making improvements by reducing the toxic portions of the exhaust stream.

We are not listing ambient PM as an MSAT because it does not conform to the approach described above for listing an MSAT. The Agency has specific information regarding the cancer and noncancer hazard posed by diesel exhaust and we are acting on this basis. Listing the only specific compounds in of diesel exhaust for which health effects have been determined would be under-inclusive, because the cancer and noncancer health effects observed with exposure to diesel exhaust cannot currently be attributed to individual compounds.

We received comments questioning the listing of several other MSATs. One commenter questioned the listing of n-hexane. This commenter suggested that since the levels of n-hexane required to cause adverse health effects are significantly higher than those found in motor vehicle

exhaust, this compound should not be on the MSAT list. As explained above, our approach for listing a compound as an MSAT is based on the presence of the compound in the emissions from motor vehicles or their fuels and an Agency consensus view, as expressed on IRIS, that the compound has the potential to cause serious adverse health effects. In keeping with this approach, we will maintain n-hexane on our list of MSATs. It should be noted that inclusion of these compounds on the MSAT list is not itself a determination by EPA that environmental exposures to emissions of the compound in fact present a risk to public health or welfare, or that it is appropriate to adopt controls to limit the emissions of such a compound from motor vehicles or their fuels. The purpose of the list is to provide a screening tool that identifies those compounds emitted from motor vehicles or their fuels for which further evaluation of emissions controls is appropriate.

This same commenter suggested that EPA provide information regarding the source of chlorine in the formation of dioxin during combustion. Another commenter indicated that dioxin is not being emitted in significant quantities. Sources of chlorine include fuel, motor oil and intake air and while the emission rates measured for dioxin are low, the levels are above detection limits in several studies. As explained above, inclusion on the MSAT list does not imply that emissions are sufficiently high to pose actual risk levels needing immediate control. Rather, listing only identifies those toxics for which controls should be evaluated.

One commenter suggested that the decision to list styrene among the MSATs be deferred to a future rulemaking as the Agency is currently re-assessing the cancer and noncancer health effects of this compound and because the compound, while admittedly found in vehicle exhaust, is not routinely found in gasoline.[18] The Agency re-assessment of styrene is being conducted to incorporate new information regarding potential noncancer and cancer health effects resulting from exposure to this compound and will not result in the removal of styrene from the IRIS database.

One commenter provided evidence that in one speciation study,[19] arsenic was not found in motor vehicle exhaust at levels above the method detection limit and therefore, arsenic should not be included on the list as an MSAT being emitted from motor vehicles. We include arsenic on the motor vehicle list of MSATs due to the presence of this element in the SPECIATE database for motor vehicle emissions.[20] Updates to the SPECIATE database are planned for the

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[18]This commenter is correct that in the NPRM we mistakenly listed styrene as a component in gasoline. Styrene is a product of combustion of gasoline.

[19]Ball, James C. (1997) Emission Rates and Elemental Composition of Particles Collected from 1995 Ford Vehicles Using the Urban Dynamometer Driving Schedule, the Highway Fuel Economy Test, an the US06 Driving Cycle. Society of Automotive Engineers, SAW paper No. 97Fl-376.

[20]EPA (1993) Volatile Organic Compound (VOC) / Particulate Matter (PM) Speciation Data System (SPECIATE), Version 1.5. http://www.epa.gov/ttn/chief/software.html#speciate.

future which may change the listing of some compounds, including arsenic. Any changes to the SPECIATE database will be used to revise the MSAT list as required.

Regarding the listing of metals, we have chosen to list the entire group of metal compounds if any compound of the metal has been detected in mobile source exhaust and any compound of the metal is listed in IRIS as potentially causing adverse human health effects. Literature values report only the total amount of the metal compound identified and not the specific form of the metal being emitted in mobile source exhaust. For example, chromium (Cr) can be emitted from combustion sources in different forms, the most toxic of which is Cr+6. In the literature, the form of Cr emissions from mobile sources is unidentified. In our list of MSAT, we therefore list chromium compounds generally, and do not attempt to list specific forms of these metals because we lack metal speciation information. When we assess the range of potential health impacts associated with exposure to chromium compounds, we consider the health effects associated with all forms of the compound for which we have health effects information. For chromium, the most toxic form in IRIS is Cr+6; hence the health impacts described for chromium compounds refer to these most serious effects even though it is highly unlikely that all motor vehicle emissions of Cr are Cr+6. EPA believes this listing approach is a reasonable, health-protective way to handle the uncertainty surrounding motor vehicle emissions of metals. Moreover, it is consistent with Congress' list of HAPs for stationary sources in Section 112(b) of the Act. At the same time we recognize that to accurately assess the actual health risks associated with exposure to metal emissions from mobile sources, identification of the specific forms of the metals emitted would be important.

In the NPRM, we requested comment on whether methanol and ethanol, by virtue of their use in alternative fuel vehicles, should be included on the MSAT list. We did not include methanol on our proposed list of MSATs because it was not identified in our analysis of speciated emissions from motor vehicles.

During the comment period, one commenter directed EPA to studies that identify methanol as an emissions product of motor vehicles burning reformulated gasoline. This commenter suggested that further research needed to be conducted to determine whether methanol should be added to the list of MSAT. Recently submitted comments echoed the need to conduct further research and requested more time to consider the addition of methanol to the MSAT list.

In order to provide a full opportunity for public comment and to respond to these comments in more detail, we will address the addition of methanol to the MSAT list in a separate rulemaking. We believe it is reasonable to defer making a decision on listing methanol until after today's rulemaking, because listing in today's rulemaking would not result in additional controls. The existing motor vehicle VOC controls will reduce emissions of methanol along with

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Arsenic is found in profiles 32102 and 32103, for light duty diesel vehicles. These data were originally from the PM10 Source Composition Library for the South Coast Air Basin, Prepared by NEA, Inc., July 15, 1987.

other gaseous toxics and fuel controls will need to be considered in subsequent rulemakings. As part of the future notice addressing addition of methanol to our list of MSATs, we will also evaluate possible controls in accordance with section 202(l)(2) as appropriate.

One commenter responded to our request for commenton the addition of ethanol to the list of MSATs based on the presence of ethanol in alternative fuels and stated that ethanol should not be listed as an MSAT. At this time, EPA is not including ethanol in the list of MSATs because we do not have an Agency consensus view as expressed on IRIS regarding the potential adverse health effects associated with exposure to ethanol. The Agency is continuing toxicity testing and risk assessment of potential adverse health effects resulting from exposure to this compound. We will reassess available information regarding potential health effects of exposure to ethanol when we evaluate whether additional controls are appropriate in 2003.

As a final step, we compared our lists of emitted compounds to four lists of toxic air pollutants to confirm that our MSAT list was reasonable. The four lists of toxic air pollutants we used were: the Clean Air Act (CAA) Section 112(b) list of hazardous air pollutants; California EPA (CalEPA) list of toxic air contaminants (TAC); U.S. Department of Health and Human Service Agency for Toxic Substances and Disease Registry (ATSDR) list of Minimal Risk Levels (MRLs); and International Agency for Research on Cancer (IARC) monographs on cancer. Comparing these four lists against the emissions speciation studies and databases, we identified two additional compounds not included on our list of MSATs – propionaldehyde and 2,2,4-trimethylpentane. We received comments on both of these compounds. Some commenters suggested that further study was needed to determine the potential for adverse health effects, while others asserted the position that both compounds should be added to the list of MSATs based on their presence in the CAA section 112(b) HAP list or due to the presence of these compounds on the emissions lists.

After consideration of these comments, we are not including propionaldehyde or 2,2,4-trimethylpentane in the list of MSATs at this time. We believe that because we do not have an Agency consensus view as expressed on IRIS regarding the potential serious adverse health effects associated with exposure to these pollutants it is inappropriate to include them. EPA assessments of these compounds have been proposed and we will reassess the possible inclusion of these compounds in the list of MSATs when we evaluate whether additional controls are appropriate in 2003.

Appendix I
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Chapter 3: Health Effects of Mobile Source Air Toxics

Chapter 2 of this Technical Support Document describes how we identified the list of mobile source air toxics (MSATs). This chapter begins with a description of some of the key components of risk assessment and then provides specific information on the 21 MSATs, including their physical properties, uses, sources of potential exposure, and health hazards.

Since most cancer and noncancer health impacts of air toxics cannot be directly isolated and measured, risk assessment methods and tools have been developed to assist in evaluating them. The EPA framework for assessing and managing risks reflects the risk assessment and risk management paradigm set forth by the National Academy of Sciences in 1983[21] and incorporated into the 1986 EPA risk guidance.[22] The paradigm divides the risk assessment and management process into four general phases. The first three phases (exposure assessment, dose-response assessment, and risk characterization) comprise risk assessment. The fourth phase, risk management, involves evaluation of information provided by the risk assessment to the environmental manager who makes a risk management decision.

An exposure assessment is the quantitative or qualitative evaluation of contact to a specific pollutant and includes such characteristics as intensity, frequency, and duration of contact. The numerical output of an exposure assessment may be either exposure or dose, depending on the purpose of the evaluation and available data.

The dose-response assessment produces two sequential analyses. The first analysis is the hazard identification, which identifies contaminants that are suspected to pose health hazards, describes the specific forms of toxicity (e.g., neurotoxicity, carcinogenicity, etc.) that they may cause, and evaluates the conditions under which these forms of toxicity might be expressed in exposed humans. The types of effects that are relevant to a particular chemical (e.g., cancer, noncancer) are determined as part of the hazard identification.

The second analysis is the human health dose-response assessment, which generally describes the characterization of the relationship between the concentration, exposure, or dose of a pollutant and the resultant health effects. The nature of quantitative dose-response assessment varies among pollutants. Sufficient data often exist for criteria air pollutants, such as ozone or carbon monoxide, so that relatively complete dose-response relationships can be characterized. In such cases, there is no need for extrapolation to lower doses because adequate health effects data are available, often in humans, at environmental levels. However, such is not the case for most

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[21] National Academy of Science. 1983. Risk Assessment in the Federal Government: Managing the Process. Committee on the Institutional Means for Assessment of Risks to Public Health, National Research Council.

[22] EPA. 1986. . Guidelines for carcinogen risk assessment. Federal Register 51:33992-34003. September 24.

air toxics. Most, if not all, epidemiologic and toxicologic data on air toxics typically result from exposure levels that are high when compared to environmental levels. Dose-response assessment methods for air toxics generally consist of two parts. First is the evaluation of data in the observable range, and second is the extrapolation from the observable range to low doses/risks.

Chronic noncancer dose response assessment

Chronic noncancer effects include a wide range of effects in all organ systems, e.g., cardiovascular, immune, kidney. Hazard identification procedures for chronic noncancer effects are less formally described in EPA guidance than are procedures for the identification of carcinogens. The EPA has published guidelines for assessing several specific types of noncancer effects, including mutagenicity,[23] developmental toxicity,[24] neurotoxicity[25]; and reproductive toxicity.[26] For identification of long-term (chronic) hazards other than cancer, we review the health effects literature and characterize its strengths and weaknesses, using a narrative approach rather than, as with cancer, a formal classification scheme. Available data on different endpoints are ordered and discussed, and the effects (and their attendant dose/exposure levels) are described. Particular attention is given to effects that occur at relatively low doses or that may have particular relevance to human populations. The inhalation reference concentration (RfC) and oral reference dose (RfD) are the primary Agency consensus quantitative toxicity values for use in chronic noncancer risk assessment. The RfC or RfD is defined as an estimate, with uncertainty spanning perhaps an order of magnitude, of an inhalation exposure/oral dose to the human population (including sensitive subgroups) that is likely to be without appreciable risks of deleterious effects during a lifetime. The RfC or RfD is derived after a thorough review of the health effects database for an individual chemical and identification of the most sensitive and relevant endpoint and the principal study(ies) demonstrating that endpoint. Inhalation RfCs are derived according to the Agency's 1994 guidance.[27]

Acute noncancer dose-response assessment

Acute effects can result from short-term exposures to chemicals. At present, no peer reviewed metrics exist for evaluating short-term exposure to hazardous air pollutants. EPA's Office of Research and Development has proposed an Acute Reference Exposure (ARE)

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[23] EPA. 1986. Guidelines for mutagenicity risk assessment. Federal Register 51:34006-34012. Sept. 24.

[24] EPA. 1991. Guidelines for developmental toxicity risk assessment. Federal Register 56:63798-63826.

[25] EPA. 1998. Guidelines for neurotoxicity risk assessment. Federal Register 63:26926. May 14. http://www.epa.gov/ncea/nurotox.htm

[26] EPA. 1996. Guidelines for reproductive toxicity risk assessment. EPA/630/R-96/009. Federal Register 56274-56322, 31 October 1996. http://www.epa.gov/ORD/WebPubs/repro/

[27] EPA. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Washington D.C. EPA/600/8-90/066F.

approach[28] which is adaptable to any duration of exposure up to 24 hours.

Cancer dose response assessment

"Cancer" describes a group of related diseases that affect a variety of organs and tissues. Cancer results from a combination of genetic damage and nongenetic factors that favor the growth of damaged cells. The EPA's 1986 *Guidelines for Carcinogen Risk Assessment*[29] provide 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 1986 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:

> **Group A - Carcinogenic to Humans:** Applies when there are adequate human data to demonstrate the causal association of the agent with human cancer (typically epidemiologic data).
> **Group B - Probably Carcinogenic to Humans:** Agents with sufficient evidence (i.e., indicative of a causal relationship) from animal bioassay data, but either limited (i.e., indicative of a possible causal relationship, but not exclusive of alternative explanations) human evidence (Group B1), or with little or no human data (Group B2).
> **Group C - Possibly Carcinogenic to Humans:** Agents with limited animal evidence and little or no human data.
> **Group D - Not Classifiable as to Human Carcinogenicity:** Agents without adequate data either to suggest or refute the suggestion of the human carcinogenicity.
> **Group E - Evidence of Noncarcinogenicity for Humans:** Agents that show no evidence for carcinogenicity in at least two adequate animal tests in different species or in both adequate epidemiologic and animal studies.

Dose-response models such as the linear multistage model, which assumes a default assumption that chemical carcinogens would exhibit risks at any dose, have historically been used to calculate upper-bound unit risk estimates (URE). Typically, EPA has relied on the unit risk estimate as a quantitative measure of potential cancer hazard. A unit risk estimate represents an estimate of the increased cancer risk from a lifetime (assumed 70 year) exposure to a

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[28] EPA. 1998. Methods for exposure-response analysis for acute inhalation exposure to chemicals: development of the acute reference exposure. Review draft. Office of Research and Development, Washington, D.C. EPA/600/R-98/051.

[29] EPA. 1986. . Guidelines for carcinogen risk assessment. Federal Register 51:33992-34003. September 24.

concentration of one unit of exposure. The unit risk estimate for inhalation exposures is typically expressed as risk per microgram per cubic meter for air contaminants. The unit risk estimate is a plausible upper-bound estimate of the risk (i.e., the risk is not likely to be higher but may be lower and may be zero).

In 1996, EPA/ORD proposed major revisions of the carcinogen hazard identification scheme. The proposed revision to the cancer risk assessment guidelines,[30] currently under public review prior to finalization represents a considerable departure from the original guidelines. Considerable new knowledge has been developed regarding the processes of chemical carcinogenesis and the evaluation of human cancer risk. Please consult the guidelines for a complete explanation.

A . Acetaldehyde

Acetaldehyde is ubiquitous in the ambient environment. It is an intermediate product of higher plant respiration and is formed as a product of incomplete wood combustion in fireplaces and wood stoves, coffee roasting, burning of tobacco, vehicle exhaust fumes, and coal refining and waste processing. Hence, many individuals are exposed to acetaldehyde by breathing ambient air. It should be noted that residential fireplaces and wood stoves are the two highest sources of emissions, followed by various industrial emissions. Exposure may also occur in individuals occupationally exposed to acetaldehyde during its manufacture and use. In addition, acetaldehyde is formed in the body from the breakdown of ethanol; this would be a source of acetaldehyde among those who consume alcoholic beverages.[31]

Acetaldehyde is a saturated aldehyde that is found in vehicle exhaust and is formed as a result of incomplete combustion of both gasoline and diesel fuel. It is not a component of evaporative emissions. Acetaldehyde comprises 0.4 to 1.0 percent of exhaust total organic gases (TOG), depending on control technology and fuel composition.[32] Primary acetaldehyde emissions from mobile sources account for approximately 66 percent of the emissions in the 1996 National Toxics Inventory.

The atmospheric chemistry of acetaldehyde is similar in many respects to that of

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[30] EPA. 1996. Proposed Guidelines for Carcinogen Risk Assessment. Office of Research and Development, Washington, DC. EPA/600/P-92/003C. http://www.epa.gov/ORD/WebPubs/carcinogen/

[31] EPA. 1987. Health Assessment Document for Acetaldehyde. EPA/600/8-86-015A. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Research Triangle Park, NC. 1987.

[32] EPA 1993. Motor Vehicle-Related Air Toxics Study, U.S. Environmental Protection Agency, Office of Mobile Sources, Ann Arbor, MI, EPA Report No. EPA 420-R-93-005, April 1993.

formaldehyde.[33] Like formaldehyde, it can be both produced and destroyed by atmospheric chemical transformation. Mobile sources contribute to ambient acetaldehyde levels both through direct emissions of acetaldehyde and as a result of secondary formation of acetaldehyde from VOC emissions.

Although human data on the carcinogenic potential of acetaldehyde are extremely limited, acetaldehyde is classified as a probable human carcinogen.[34] This classification is based on a formal risk characterization using sufficient database of animal carcinogenicity studies.[35] Specifically, increased incidences of nasal tumors in male and female rats and laryngeal tumors in male and female hamsters have been documented after inhalation exposure. Updated noncancer and cancer potencies for acetaldehyde, addressing the issue of animal to human extrapolation, will be developed in a similar fashion to that for formaldehyde, as discussed later.

The primary acute effects associated with exposure to acetaldehyde include irritation of the eyes, skin, and respiratory tract. Effects on the respiratory system have been reported from studies of animals exposed to long-term lower concentrations. The data from these studies was found to be sufficient for EPA to develop a RfC[36] (inhalation reference concentration) for acetaldehyde exposure. Although no information is available on the reproductive or developmental effects of acetaldehyde in humans, data from animal studies suggest that acetaldehyde may be a potential developmental toxicant.[37,38]

B. Acrolein

Acrolein is an aldehyde primarily used as an intermediate in the manufacture of acrylic

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[33] Ligocki, M.P., and G.Z. Whitten, Atmospheric transformation of air toxics: acetaldehyde and polycyclic organic matter, Systems Applications International, San Rafael, CA, (SYSAPP-91/113), 1991.

[34] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[35] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[36] A RfC is defined as is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime. RfCs are based on an assumption of lifetime inhalation exposure and may not be appropriately applied to less-than-lifetime exposure situations. RfCs are derived for the noncarcinogenic effects of chemicals that are carcinogenic.

[37] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[38] EPA. 1987. Health Assessment Document for Acetaldehyde. EPA/600/8-86-015A. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Research Triangle Park, NC. 1987.

acid. It can be formed from the breakdown of certain pollutants in outdoor air or from burning tobacco or gasoline.[39] Acrolein is found in vehicle exhaust and is formed as a result of incomplete combustion of both gasoline and diesel fuel. It is not a component of evaporative emissions. Acrolein comprises 0.05 to 0.4 percent of exhaust total organic gases (TOG), depending on control technology and fuel composition. Primary acrolein emissions from mobile sources account for approximately 38 percent of the emissions in the 1996 National Toxics Inventory. The atmospheric chemistry of acrolein is expected to be similar in many respects to that of acetaldehyde and formaldehyde.

Although no information on the carcinogenic effects of acrolein in humans is available, limited laboratory data for animals exposed by drinking water ingestion indicated an increased incidence of adrenal cortical adenomas (non-malignant tumors of the adrenal glands adjacent to the kidney). EPA has classified acrolein as a Group C, possible human carcinogen, based on limited animal and mutagenticity data. A formal risk characterization has not conducted due to this limited data set.

The respiratory system is the primary target in humans and animals for acrolein toxicity resulting from inhalation exposure. Acute exposure results in upper respiratory tract irritation and congestion, whereas chronic exposures in animals indicated an increase in cell proliferation and in the numbers of white blood cells in the tissues lining the nasal passages. The data from these studies was found to be sufficient for EPA to develop a RfC (inhalation reference concentration) for acrolein exposure. No information is available on the reproductive or developmental effects of acrolein in humans.[40]

C. Arsenic Compounds

Arsenic, a naturally occurring element, is found throughout the environment. It is released into the air by volcanoes, the weathering of arsenic-containing minerals and ores, and by commercial or industrial processes. For most people, food is the largest source of arsenic exposure (about 25 to 50 µg/d), with lower amounts coming from drinking water and air. Among foods, some of the highest levels are in fish and shellfish; however, this arsenic exists primarily as organic compounds, which are essentially nontoxic. Elevated levels of inorganic arsenic may be present in soil, either from natural mineral deposits or contamination from human activities, which may lead to dermal or ingestion exposure. Workers in metal smelters and nearby residents may be exposed to above-average inorganic arsenic levels from arsenic released into the air. Other sources of inorganic arsenic exposure include burning plywood treated with an arsenic wood preservative or dermal contact with wood treated with arsenic.[41]

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[39] ATSDR. 1990. Toxicological Profile for Acrolein. USDHHS, PHS, ATSDR. TP-90-01.

[40] EPA 1991. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

[41] ATSDR. 1992. Toxicological Profile for Arsenic. USDHHS, PHS, ATSDR. TP-92/02.

Arsenic emissions from mobile sources are minimal, accounting for less than 1 percent of the emissions in the 1996 National Toxics Inventory. It is thought that the arsenic found in the emissions is due to impurities in either fuel additives or the fuel itself.

Inhalation exposure to inorganic arsenic has been shown to be strongly associated with lung cancer in humans, while ingestion of inorganic arsenic in humans has been linked to a form of skin cancer and also to bladder, liver, and lung cancer. There is sufficient data in humans for EPA to classify inorganic arsenic as a Group A, human carcinogen[42] and a full risk characterization has been conducted by EPA.

Acute (short-term) high-level inhalation exposure to arsenic dust or fumes has resulted in gastrointestinal effects (nausea, diarrhea, abdominal pain); central and peripheral nervous system disorders have occurred in workers acutely exposed to inorganic arsenic**.** Chronic (long-term) inhalation exposure to inorganic arsenic in humans is associated with irritation of the skin and mucous membranes. Human data suggest a relationship between inhalation exposure of women working at or living near metal smelters and an increased risk of reproductive effects, such as spontaneous abortions. However, as these studies evaluated smelter pollutants in general, arsenic's role is not clear. Chronic oral exposure has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyperpigmentation, and liver or kidney damage.[43] The data from these studies was found to be sufficient for EPA to develop a RfD[44] (oral reference dose) for oral arsenic exposure.

D. Benzene

Benzene is an aromatic hydrocarbon which is present as a gas in both exhaust and evaporative emissions from motor vehicles as well as from the burning of coal and oil. Benzene comprises 3.0 to 5.0 percent of mobile source exhaust total organic gases (TOG), which varies depending on control technology (e.g., type of catalyst) and the levels of benzene and aromatics in the fuel. The benzene fraction of evaporative TOG emissions is generally about one percent, but depends on control technology and fuel composition (e.g., benzene level and Reid Vapor

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[42] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[43] EPA 1999. Environmental Protection Agency, National Air Toxics Program: The Integrated Urban Strategy Report to Congress, Office of Air Quality Planning and Standards, RTP, NC. EPA 453/R-99-007.

[44] A RfD is defined as is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime. RfDs are based on an assumption of lifetime oral exposure and may not be appropriately applied to less-than-lifetime exposure situations. RfDs are derived for the noncarcinogenic effects of chemicals that are carcinogenic.

Pressure (RVP)).[45] Benzene emissions from mobile sources account for approximately 76 percent of the outdoor emissions in the 1996 National Toxics Inventory. Tobacco smoke contains benzene and accounts for nearly half the national exposure to benzene.[46]

Benzene is also used as a solvent for fats, waxes, resins, oils, inks, paints, plastics, and rubber; in the extraction of oils from seeds and nuts; and in photogravure printing. It is also used as a chemical intermediate. Benzene is also used in the manufacture of detergents, explosives, pharmaceuticals, and dyestuffs.[47]

The EPA has recently reconfirmed that benzene is a known (Group A) human carcinogen by all routes of exposure and a full risk characterization has been conducted by EPA.[48] Respiration is the major source of human exposure and at least half of the respiratory exposure is by way of gasoline vapors and automotive emissions. Long-term exposure to high levels of benzene in air has been shown to cause cancer of the tissues that form white blood cells. Specifically, benzene has been linked to acute (rapid and fatal) nonlymphocytic[49] leukemia, chronic (lingering, lasting) lymphocytic leukemia and possibly multiple myeloma (primary malignant tumors in the bone marrow), although the evidence for the latter has decreased with more recent studies. Leukemias, lymphomas, and other tumor types have been observed in experimental animals that have been exposed to benzene by inhalation or oral administration. Exposure to benzene and/or its metabolites has also been linked with genetic changes in humans and animals and increased proliferation of mouse bone marrow cells. The occurrence of certain chromosomal changes in individuals with known exposure to benzene may serve as a marker for those at risk for contracting leukemia.

A number of adverse noncancer health effects, blood disorders such as preleukemia and aplastic anemia, have also been associated with low-dose, long-term exposure to benzene.[50]

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[45] EPA 1993. Motor Vehicle-Related Air Toxics Study, U.S. Environmental Protection Agency, Office of Mobile Sources, Ann Arbor, MI, EPA Report No. EPA 420-R-93-005, April 1993.

[46] Wallace, L.A. 1995. Human exposure to environmental pollutants: a decade of experience. Clinical and Experimental Allergy 25:4-9.

[47] ATSDR. 1997. Toxicological Profile for Benzene (update). USDHHS, PHS, ATSDR. Atlanta , GA.

[48] EPA 1998. Environmental Protection Agency, Carcinogenic Effects of Benzene: An Update, National Center for Environmental Assessment, Washington, DC. 1998.

[49] Leukemia is a blood disease in which the white blood cells are abnormal in type or number. Leukemia may be divided into nonlymphocytic (granulocytic) leukemias and lymphocytic leukemias. Nonlymphocytic leukemia generally involves the types of white blood cells (leukocytes) that are involved in engulfing, killing, and digesting bacteria and other parasites (phagocytosis) as well as releasing chemicals involved in allergic and immune responses.

[50] EPA 1998. Environmental Protection Agency, Carcinogenic Effects of Benzene: An Update, National Center for Environmental Assessment, Washington, DC. 1998

People with long-term exposure to benzene may experience harmful effects on the blood-forming tissues, especially the bone marrow. These effects can disrupt normal blood production and cause a decrease in important blood components. Chronic inhalation exposure to benzene in humans and animals can result in pancytopenia,[51] a condition characterized by decreased numbers of circulating erythrocytes (red blood cells), leukocytes (white blood cells), and thrombocytes (blood platelets).[52,53] Some individuals that develop pancytopenia and continue to be exposed to benzene may develop aplastic anemia, a more severe blood disease that occurs when the bone marrow ceases to function. The aplastic anemia can progress to AML (acute mylogenous leukemia). The most sensitive noncancer effect observed in humans is the depression of absolute lymphocyte counts in the circulating blood.[54] EPA is currently reassessing the noncancer health impacts of benzene.

E. 1,3-Butadiene

1,3-Butadiene is formed in vehicle exhaust by the incomplete combustion of gasoline and diesel fuel. It is not present in vehicle evaporative and refueling emissions, because it is not present in any appreciable amount in gasoline. 1,3-Butadiene accounts for 0.4 to 1.0 percent of total exhaust TOG, depending on control technology and fuel composition.[55] 1,3-Butadiene emissions from mobile sources account for approximately 60 percent of the emissions in the 1996 National Toxics Inventory.

Sources of 1,3-butadiene released into the air also include manufacturing and processing facilities, especially oil refineries, chemical manufacturing plants, and plastic and rubber factories. Other sources are forest fires or other combustion, and cigarette smoke.[56]

1,3-Butadiene was classified by EPA as a Group B2 (probable human) carcinogen in

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[51] Pancytopenia is the reduction in the number of all three major types of blood cells (erythrocytes, or red blood cells, thrombocytes, or platelets, and leukocytes, or white blood cells).

[52] Aksoy, M. 1991. Hematotoxicity, leukemogenicity and carcinogenicity of chronic exposure to benzene. In: Arinc, E.; Schenkman, J.B.; Hodgson, E., Eds. Molecular Aspects of Monooxygenases and Bioactivation of Toxic Compounds. New York: Plenum Press, pp. 415-434.

[53] Goldstein, B.D. 1988. Benzene toxicity. Occupational medicine. State of the Art Reviews. 3: 541-554.

[54] Rothman, N., G.L. Li, M. Dosemeci, W.E. Bechtold, G.E. Marti, Y.Z. Wang, M. Linet, L.Q. Xi, W. Lu, M.T. Smith, N. Titenko-Holland, L.P. Zhang, W. Blot, S.N. Yin, and R.B. Hayes. 1996. Hematotoxicity among Chinese workers heavily exposed to benzene. Am. J. Ind. Med. 29: 236-246.

[55] EPA 1993. Motor Vehicle-Related Air Toxics Study, U.S. Environmental Protection Agency, Office of Mobile Sources, Ann Arbor, MI, EPA Report No. EPA 420-R-93-005, April 1993.

[56] ATSDR. 1992. Toxicological Profile for 1,3-Butadiene. USDHHS, PHS, ATSDR. TP-91/07.

1985[57]. This classification was based on evidence from two species of rodents and epidemiologic data. EPA recently prepared a draft risk assessment that proposes that sufficient evidence exists to characterize 1,3-butadiene be a known human carcinogen.[58] This designation is based on a combination of epidemiologic evidence as well as experimental evidence demonstrating causality. The Environmental Health Committee of EPA's Scientific Advisory Board (SAB), reviewed the draft document in August 1998 and recommended that designation of 1,3-butadiene as a known human carcinogen be based on observational studies in humans, without regard to mechanistic or other information.[59] In applying the 1996 proposed Guidelines for Carcinogen Risk Assessment, the Agency relies on both observational studies in humans as well as experimental evidence demonstrating causality and therefore the designation of 1,3-butadiene as a known human carcinogen remains applicable.[60] The SAB panel also recommended that EPA calculate the lifetime cancer risk estimates based on the human data from Delzell et al. 1995[61] and account for the highest exposure of "360 ppm-year" for 70 years. Further input recommended that EPA take into account additional data on health effects observed in female laboratory animals, hence indicating that females may be a sensitive subpopulation.

1,3-Butadiene also causes a variety of reproductive and developmental effects in mice and rats exposed to long-term, low doses of butadiene (EPA 1998c). The most sensitive effect was reduced litter size at birth and at weaning. These effects were observed in studies in which male mice exposed to 1,3-butadiene were mated with unexposed females. In humans, such an effect might manifest itself as an increased risk of spontaneous abortions, miscarriages, still births, or very early deaths. The data from these studies was found to be sufficient for EPA to develop a draft chronic, subchronic, and acute RfC for 1,3-butadiene exposure as part of the draft risk characterization mentioned above. The RfC values will be reported on IRIS.

F. Chromium Compounds

Chromium is a naturally occurring element in rocks, animals, plants, soil, and volcanic dust and gases. Chromium occurs in the environment predominantly in one of two valence states: trivalent chromium (Cr III), which occurs naturally and is an essential nutrient, and

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[57] EPA, 1985. Mutagenicity and carcinogenicity assessment of 1,3-butadiene. EPA/600/8-85/004F. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. Washington, DC.

[58] EPA 1998c. Environmental Protection Agency, Health Risk Assessment of 1,3-Butadiene. EPA/600/P-98/001A, February 1998.

[59] Scientific Advisory Board. 1998. An SAB Report: Review of the Health Risk Assessment of 1,3-Butadiene. EPA-SAB-EHC-98, August, 1998.

[60] EPA 1996. Proposed guidelines for carcinogen risk assessment. Federal Register 61(79):17960-18011.

[61] Delzell, E., N. Sathiakumar, M. Macaluso, M. Hovinga, R. Larson, F. Barbone, C. Beall, and P. Cole, 1995. A follow-up study of synthetic rubber workers. Final report prepared under contract to International Institute of Synthetic Rubber Producers, October 2, 1995.

hexavalent chromium (Cr VI), which, along with the less common metallic chromium (Cr 0), is most commonly produced by industrial processes. Chromium (III) is essential to normal glucose, protein, and fat metabolism and is thus an essential dietary element with a daily intake of 50 to 200 µg/d recommended for an adult. The body has several systems for reducing chromium (VI) to chromium (III). This chromium (VI) detoxification leads to increased levels of chromium (III).

Air emissions of chromium are predominantly of trivalent chromium, and in the form of small particles or aerosols. The most important industrial sources of chromium in the atmosphere are those related to ferrochrome production. Ore refining, chemical and refractory processing, cement-producing plants, automobile brake lining and catalytic converters for automobiles, leather tanneries, and chrome pigments also contribute to the atmospheric burden of chromium.[62] Total chromium emissions from mobile sources account for approximately 4 percent of the emissions in the 1996 National Toxics Inventory.

Human studies have clearly established that inhaled chromium (VI) is a human carcinogen, resulting in an increased risk of lung cancer. Animal studies have shown chromium (VI) to cause lung tumors via inhalation exposure. There is sufficient data in humans for EPA to classify chromium (VI) as a Group A, human carcinogen and a full risk characterization has been conducted by EPA.[63]

The respiratory tract is the major target organ for acute (short-term) and chronic (long-term) inhalation exposures to chromium (VI). Shortness of breath, coughing, and wheezing were reported from a case of acute exposure to chromium (VI), while perforations and ulcerations of the septum, bronchitis, decreased pulmonary function, pneumonia, and other respiratory effects have been noted from chronic exposure. Limited human studies suggest that chromium (VI) inhalation exposure may be associated with complications during pregnancy and childbirth, while animal studies have not reported reproductive effects from inhalation exposure to chromium (VI).[64] The data from these studies was found to be sufficient for EPA to develop a RfC for chromium (VI) exposure. Additional data from oral exposure studies was found to be sufficient for EPA to develop a RfD for chromium (VI) oral exposure.

Chromium (III) is much less toxic than chromium (VI). The respiratory tract is also the major target organ for chromium (III) toxicity, similar to chromium (VI) but data from animal studies do not demonstrate that the effects observed following inhalation of chromium (VI)

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[62] EPA. 1998. Toxicological Review of Trivalent Chromium (CAS No. 16065-83-1). In Support of Summary Information on the Integrated Risk Information System (IRIS). U.S. EPA, Washington D.C..

[63] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[64] EPA 1999. Environmental Protection Agency, National Air Toxics Program: The Integrated Urban Strategy Report to Congress, Office of Air Quality Planning and Standards, RTP, NC.

particulates. Chromium (III) is most appropriately designated a Group D carcinogen (not classified as to its human carcinogenicity) because there are inadequate data to conduct a full risk characterization and determine its potential carcinogenicity.[65]

G. Dioxin/Furans

Dioxin comes from both natural and industrial sources, such as medical and municipal waste incineration and paper-pulp production. Recent studies have confirmed that dioxins are formed by and emitted from heavy-duty diesel trucks and are estimated to account for one percent of total dioxin emissions in the dioxin inventory for the year 1995.[66] The actual process of dioxin and furan formation in the combustion of organic materials is discussed in detail in the EPA report, Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (TCDD) and Related Compounds.[67]

In general, dioxin exposures of concern have primarily been noninhalation exposures associated with human ingestion of certain foods (e.g., beef, pork, poultry, fish, eggs and dairy products) contaminated by dioxin. The two primary pathways for dioxin to enter the human diet are: air-to-plant-to-animal and water/sediment-to-fish. Vegetation receives these compound via atmospheric transport and deposition. The compounds are retained on plant surfaces and bioaccumulate in fatty tissues of animals that feed on the vegetation. In the aquatic food chain, dioxins enter water systems via direct discharge or deposition and runoff from watersheds. Fish accumulate dioxin through their direct contact with water, suspended particles, bottom sediments and through the consumption of aquatic organisms. Exposure to dioxin occurs over a lifetime, and the exposure is cumulative over a lifetime.

Based on recent human epidemiological studies from Europe and the United States, dioxin has been linked to several cancers, including lymphomas and lung cancer, by the International Agency For Research on Cancer (IARC). The IARC classifies the most potent form of dioxin, 2,3,7,8-tetrachlorodibenzo- p-dioxin (TCDD), as a "Group 1" carcinogen, meaning it is

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[65] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[66] U.S. EPA (2000). Sources of Dioxin-Like Compounds in the United States; In: Exposure and Human Health Reassessment of 2,3,7,8-TCDD and Related compounds. Part 1: Estimating Exposure to Dioxin-like Compounds; Volume 2. National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency, Washington, DC. March 2000 draft final. EPA/600/P-00/001Ab.

[67] Draft Final Report Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (TCDD) and Related Compounds, Part I: Estimating Exposure to Dioxin-Like Compounds, Volume 2: Sources of Dioxin-Like Compounds in the United States. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment - Washington Office, Exposure Assessment and Risk Characterization Group. EPA/600/P-00/001Bb, September 2000.

carcinogenic to humans.[68]

Low-grade exposure to dioxin/furans have also been linked to a wide array of other health problems, including changes in hormone levels and developmental defects in babies and children.[69] EPA classified dioxins as probable human carcinogens in 1985. Recently EPA has proposed, and the Scientific Advisory Board has concurred, to classify TCDD as a human carcinogen and the complex mixtures of dioxin-like compounds as likely to be carcinogenic to humans using the draft 1996 carcinogen risk assessment guidelines.[1] Using the 1986 cancer risk assessment guidelines, the hazard characterization for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin is 'known' human carcinogen and the hazard characterization for complex mixtures of dioxin-like compounds is 'probable' human carcinogens.

H. Diesel Exhaust: Diesel Particulate Matter and Diesel Exhaust Organic Gases

Diesel exhaust includes components in the gas and particle phases. Gaseous components of diesel exhaust include at least one organic compound known to cause cancer in humans (e.g., benzene) while possible or probable human carcinogens and compounds causing noncancer effects are also present in the gas-phase (e.g., formaldehyde, acetaldehyde, 1,3-butadiene, acrolein). The health effects of these and other gaseous compounds in diesel exhaust are discussed elsewhere in this chapter. Three classes of compounds associated with particle-phase diesel exhaust (e.g., polycyclic organic matter, metals, and dioxins) are also discussed here in relation to diesel exhaust particulate matter and are also discussed under separate sections in this chapter. Diesel exhaust is a complex mixture of carbon particles and associated organics and inorganics, and it is not known what fraction or combination of fractions cause the health effects (discussed below) that have been observed with exposure to diesel exhaust. While we are listing diesel exhaust as diesel particulate matter and diesel exhaust organic gases (DPM + DEOG) to provide specific targets for emission reduction and therefore a framework for developing regulatory control strategies, the available science cannot separate the health effects of the particulate and gaseous components of diesel exhaust. This listing includes the components of diesel exhaust that are likely to contribute to the cancer and noncancer hazard (with the exception of the gaseous phase criteria pollutants, such as NOx, SO_2 and CO which are subject to National Ambient Air Quality Standards); however, currently available science, as summarized in the draft Health Assessment for Diesel Exhaust, while suggesting an important role for the particulate phase component of diesel exhaust, does not attribute the serious likely cancer and noncancer health effects independently to diesel particulate matter separate as distinct from the gas phase components. Accordingly, we discuss the health effects attributed to whole diesel exhaust in this section.

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[68] IARC (1997). Polychlorinated dibenzo-para-dioxins and Polychlorinated Dibenzofurans. Volume 69, IARC Monogram on the Evaluation of Carcinogenic Risks to Humans, International Agency for Research on Cancer, Work Health Organization, Lyon France.

[69] U.S. EPA (1994) Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds: Volume III Summary Draft Document. EPA/600/BP-92/001c.

1. Cancer Effects of Diesel Exhaust

The EPA has concluded that diesel exhaust is likely to be carcinogenic to humans by inhalation at occupational and environmental levels of exposure.[70] The draft *Health Assessment Document for Diesel Exhaust* (draft Assessment), was reviewed in public session by the Clean Air Scientific Advisory Committee (CASAC) on October 12-13, 2000. The CASAC found that the Agency's conclusion that diesel exhaust is likely to be carcinogenic to humans is scientifically sound. CASAC concurred with the draft Assessment's findings with the proviso that EPA provide modifications and clarifications on certain topics. The Agency expects to produce the finalized Assessment in early 2001. Information presented here is consistent with that to be provided in the final Assessment.

In its review of the published literature, EPA found that about 30 individual epidemiologic studies show increased lung cancer risk associated with diesel emissions. In the draft Assessment EPA evaluated 22 studies that were relevant for risk assessment, 16 of which reported significant increased lung cancer risks, ranging from 20 to 167 percent, associated with diesel exhaust exposure. Published analytical results of pooling many of the 30 studies showed that on average, the risks were increased by 33 to 47 percent.[71] [72] Questions remain about the influence of other factors (e.g., effect of smoking, other particulate sources), the quality of the individual epidemiologic studies, exposure levels, and consequently the precise magnitude of the increased risk of lung cancer. From a weight of evidence perspective, EPA concludes that the epidemiologic evidence, as well as supporting data from certain animal and mode of action studies, support the Agency's conclusion that exposure to diesel exhaust is likely to pose a human lung cancer hazard to occupationally exposed individuals as well as to the general public exposed to typically lower environmental levels of diesel exhaust.

While available evidence supports EPA's conclusion that diesel exhaust is likely to be a human lung carcinogen, and thus is likely to pose a cancer hazard to humans, EPA has concluded that the available data is not sufficient to develop a confident estimate of cancer unit risk. The absence of a cancer unit risk for diesel exhaust limits our ability to quantify, with confidence, the potential impact of the hazard (magnitude of risk) on exposed populations. In the draft Assessment, EPA acknowledged this limitation and provided a discussion of the possible environmental cancer risk consistent with the majority of the occupational epidemiological findings of increased lung cancer risk and the exposure differences between the occupational and

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[70] EPA. 2000. Health Assessment Document for Diesel Exhaust: SAB Review Draft. EPA/600/8-90/057E Office of Research and Development, Washington, D.C. The document is available electronically at www.epa.gov/ncea/dieslexh.htm.

[71] Bhatia, R., Lopipero, P., Smith, A. (1998) Diesel Exhaust Exposure and Lung Cancer. *Epidemiol.* 9:84-91.

[72] Lipsett, M., Campleman, S. (1999) Occupational Exposure to Diesel Exhaust and Lung Cancer: A Meta-analysis. *Am J Public Health* 80:1009-1017.

environmental settings.[73]

2. Noncancer Effects of Diesel Exhaust

The acute and chronic exposure-related noncancer effects of diesel exhaust emissions are also of concern to the Agency. Acute exposure to diesel exhaust can result in physiologic symptoms consistent with irritation and inflammation, and evidence of immunological effects including increased reaction to allergens and some symptoms associated with asthma. The acute effects data, however, lack sufficient detail to permit the calculation of protective levels for human exposure.

For chronic diesel exhaust exposure, EPA is completing the development of an inhalation reference concentration (RfC) for diesel exhaust exposure. The RfC is an estimate of the continuous human inhalation exposure (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious non-cancer effects during a lifetime. While the limited amount of human data is suggestive of respiratory distress, animal test data is quite definitive in providing a basis to anticipate a hazard to the human lung based on the irritant and inflammatory reactions in test animals. Thus, EPA believes that chronic diesel exhaust exposure, at sufficient exposure levels, increases the hazard and risk of an adverse health effect.

In addition, it is also instructive to recognize that diesel exhaust particulate matter is part of ambient fine PM. A qualitative comparison of adverse effects of exposure to ambient fine PM and diesel exhaust particulate matter shows that the respiratory system is adversely affected in both cases, though a wider spectrum of adverse effects has been identified for ambient fine PM. Relative to the diesel PM database, there is a wealth of human data for fine PM noncancer effects. Since diesel exhaust PM is a component of ambient fine PM, the fine PM health effects data base can be informative. The final Assessment will discuss the fine PM health effects data and its relation to evaluating health effects associated with diesel exhaust.

I. Ethylbenzene

Ethylbenzene is a colorless, aromatic hydrocarbon, that smells like gasoline. It is used primarily in the production of styrene. It is also used as a solvent, as a constituent of asphalt and naphtha. It is present as a gas in both gasoline and diesel exhaust and evaporative emissions from gasoline powered vehicles.[74] Ethylbenzene emissions from mobile sources account for approximately 84 percent of the emissions in the 1996 National Toxics Inventory.

Ethylbenzene exposure also occurs from the use of consumer products, pesticides,

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[73] See Chapter 8.4 and 9.5.2 of the U.S. EPA (2000) Health Assessment Document for Diesel Emissions: SAB Review Draft. EPA/600/8-90/057E Office of Research and Development, Washington, D.C. The document is available electronically at www.epa.gov/ncea/dieselexh.htm.

[74] ATSDR. 1999. Toxicological Profile for Ethylbenzene (update). USDHHS, PHS, ATSDR.

solvents, carpet glues, varnishes, paints, and tobacco smoke. Indoor air usually has a higher average concentration of ethylbenzene (about 1 ppb) than ambient air, due to the use of household cleaning products or paints.[75]

Limited information is available on the carcinogenic effects of ethylbenzene in humans and animals. Based on inadequate data from animal bioassays and human studies, EPA has classified ethylbenzene as a Group D carcinogen, meaning it is not classifiable as to human carcinogenicity and a full risk characterization has not been conducted by EPA due to this limited data.[76]

Acute (short-term) exposure to ethylbenzene in humans results in noncancer respiratory effects, such as throat irritation and chest constriction, irritation of the eyes, and neurological effects such as dizziness. Chronic (long-term) exposure to ethylbenzene by inhalation in humans may result in effects on the blood. Animal studies have reported effects on the blood, liver, and kidneys from chronic inhalation exposure to ethylbenzene. The data from these studies was found to be sufficient for EPA to develop a RfC for ethylbenzene exposure. No information is available on the developmental or reproductive effects of ethylbenzene in humans, although animal studies have reported developmental effects, including birth defects in animals exposed via inhalation.

J. Formaldehyde

Formaldehyde is used mainly to produce resins used in particle board products and as an intermediate in the synthesis of other chemicals. It also has minor uses in agriculture, as an analytical reagent, in concrete and plaster additives, cosmetics, disinfectants, fumigants, photography, and wood preservation. The highest levels of airborne formaldehyde have been detected in indoor air, where it is released from various consumer products such as building materials and home furnishings.

Formaldehyde is the most prevalent aldehyde in vehicle exhaust. It is formed from incomplete combustion of both gasoline and diesel fuel and accounts for one to four percent of total exhaust TOG emissions, depending on control technology and fuel composition. It is not found in evaporative emissions. Primary formaldehyde emissions from mobile sources account for approximately 41 percent of the emissions in the 1996 National Toxics Inventory.

Formaldehyde exhibits extremely complex atmospheric behavior.[77] It is present in

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[75] ATSDR. 1999. Toxicological Profile for Ethylbenzene (update). USDHHS, PHS, ATSDR.

[76] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[77] Ligocki, M.P., G.Z. Whitten, R.R. Schulhof, M.C. Causley, and G.M. Smylie, Atmospheric transformation of air toxics: benzene, 1,3-butadiene, and formaldehyde, Systems Applications International, San

emissions and is also formed by the atmospheric oxidation of virtually all organic species, including biogenic (produced by a living organism) hydrocarbons. Mobile sources contribute both primary formaldehyde (emitted directly from motor vehicles) and secondary formaldehyde (formed from photooxidation of other VOCs emitted from vehicles).

By conducting a formal risk characterization, EPA has classified formaldehyde as a Group B1, probable human carcinogen, based on limited evidence for carcinogenicity in humans and sufficient evidence of carcinogenicity in animal studies[78]. Epidemiological studies in occupationally exposed workers suggest that long-term inhalation of formaldehyde may be associated with tumors of the nasopharyngeal cavity (generally the area at the back of the mouth near the nose), nasal cavity, and sinus. Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to formaldehyde causes an increase in the incidence of squamous (epithelial) cell carcinomas (tumors) of the nasal cavity. The distribution of nasal tumors in rats suggests that not only regional exposure but also local tissue susceptibility may be important for the distribution of formaldehyde-induced tumors. Research has demonstrated that formaldehyde produces mutagenic activity in cell cultures.

Since completion of the current IRIS cancer assessment several years ago, research has been published regarding formaldehyde and the occurrence of respiratory tract tumors in rats whose breathing patterns are different than those of humans. Due to new work done in partnership with the Chemical Industry Institute of Toxicology (CIIT), a biologically-based dose-response (BBDR) model[79] has been developed to address these issues. The CIIT model is composed of a computational fluid dynamics (CFD) model and a clonal growth model (CGM). The CFD model of airflow in rats and humans more accurately accounts for differences in geometry of respiratory system and delivery of formaldehyde to the tissue. The CGM accounts for mutations and tumor development. The models developed by CIIT will be used to describe nose, throat, and lung tumor development and also revise the current cancer potency estimate. The draft reassessment is expected to be ready for SAB review in spring 2001.

Formaldehyde exposure also causes a range of noncancer health effects. At low concentrations (0.05-2.0 ppm), irritation of the eyes (tearing of the eyes and increased blinking) and mucous membranes are the principal effects observed in humans. At exposures of 1 to 11 ppm, other human upper respiratory effects associated with acute formaldehyde exposure include a dry or sore throat, and a tingling sensation of the nose. Sensitive individuals may experience these effects at lower concentrations. Forty percent of workers at formaldehyde-producing factories reported nasal symptoms such as rhinitis (inflammation of the nasal membrane), nasal

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Rafael, CA (SYSAPP-91/106), 1991.

[78] EPA 1987. Environmental Protection Agency, Assessment of health risks to garment workers and certain home residents from exposure to formaldehyde, Office of Pesticides and Toxic Substances, April 1987.

[79]Chemical Industry Institute of Toxicology, Hazard Characterization and Dose-Response Assessment for Carcinogencity by the Route of Inhalation, September 28, 1999.

obstruction, and nasal discharge following chronic exposure.[80] In persons with bronchial asthma, the upper respiratory irritation caused by formaldehyde can precipitate an acute asthmatic attack, sometimes at concentrations below 5 ppm.[81] Formaldehyde exposure may also cause bronchial asthma-like symptoms in nonasthmatics.[82,83] It is unclear whether asthmatics are more sensitive than nonasthmatics to formaldehyde's effects.[84]

An increased incidence of menstrual disorders and pregnancy problems were observed in women workers using urea-formaldehyde resins. However, possible confounding factors were not evaluated in this study. In another study of hospital equipment sterilizing workers there was no reported association between formaldehyde exposure and increased spontaneous abortions. Developmental effects, such as birth defects, have not been observed in animal studies with formaldehyde.[85,86]

Though EPA has not completed the calculation of an RfC for formaldehyde inhalation exposure, additional data from oral exposure studies was found to be sufficient for EPA to develop a RfD for formaldehyde oral exposure.[87]

K. n-Hexane

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[80] Wilhelmsson, B. and M. Holmstrom. 1987. Positive formaldehyde PAST after prolonged formaldehyde exposure by inhalation. The Lancet:164.

[81] Burge, P.S., M.G. Harries, W.K. Lam, I.M. O'Brien, and P.A. Patchett. 1985. Occupational asthma due to formaldehyde. Thorax 40:225-260.

[82] Hendrick, D.J., R.J. Rando, D.J. Lane, and M.J. Morris. 1982. Formaldehyde asthma: Challenge exposure levels and fate after five years. J. Occup. Med. 893-897.

[83] Nordman, H., H. Keskinen, and M. Tuppurainen. 1985. Formaldehyde asthma - rare or overlooked? J. Allergy Clin. Immunol. 75:91-99.

[84] EPA 1991. Environmental Protection Agency. Formaldehyde risk assessment update. June 11, 1991. Office of Toxic Substances, U.S. Environmental Protection Agency, Washington, DC. External review draft, June 11, 1991.

[85] U.S. Environmental Protection Agency. Health and Environmental Effects Profile for Formaldehyde. EPA/600/x-85/362. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH. 1988.

[86] World Health Organization. Environmental Health Criteria for Formaldehyde. Volume 89. World Health Organization, Geneva, Switzerland. 1989.

[87] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999. http://www.epa.gov/ngispgm3/iris/index.html

n-Hexane is a colorless volatile liquid that is insoluble in water and is highly flammable. Commercial grades of n-hexane are used as solvents for glues, varnishes, cements, and inks. It is also used as a solvent in the extraction of edible fats and oils. n-Hexane is also a component of gasoline and it is also found in the exhaust and evaporative emissions from motor vehicles.[88] n-Hexane emissions from mobile sources account for approximately 43 percent of the emissions in the 1996 National Toxics Inventory.

The most probable route of human exposure to n-hexane is by inhalation. Individuals are most likely to be exposed to n-hexane in the workplace. Monitoring data indicate that n-hexane is a widely occurring atmospheric pollutant as well.[89]

No information is available on the carcinogenic effects of n-hexane in humans or animals. EPA has made no determination as to the human carcinogenicity of n-hexane.[90]

Acute (short-term) inhalation exposure of humans to high levels of n-hexane causes mild central nervous system (CNS) depression and irritation of the skin and mucous membranes. Nervous system effects include dizziness, giddiness, slight nausea, and headache in humans. Chronic (long-term) exposure to n-hexane in air is associated with polyneuropathy in humans, with numbness in the extremities, muscular weakness, blurred vision, headache, and fatigue observed. Neurotoxic effects have also been exhibited in rats. Mild inflammatory and degenerative lesions in the nasal cavity have been observed in rodents chronically exposed by inhalation. Limited information is available on the reproductive or developmental effects of n-hexane; one study reported testicular damage in rats exposed to n-hexane through inhalation. Birth defects have not been observed in the offspring of rats chronically exposed via inhalation in several studies. The data from these studies was found to be sufficient for EPA to develop a RfC for n-hexane exposure.[91]

L. Lead Compounds

The largest source of lead in the atmosphere has been from leaded gasoline combustion. With the phase down of lead in gasoline, however, air lead levels have decreased considerably, though lead is still a component of racing and aviation fuels. In the 1996 National Toxics Inventory, mobile sources account for approximately 24 percent of the total inventory. This

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[88] ATSDR. 1999. Toxicological Profile for Hezane. USDHHS, PHS, ATSDR.

[89] ATSDR. 1999. Toxicological Profile for Hexane. USDHHS, PHS, ATSDR.

[90] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[91] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999. http://www.epa.gov/ngispgm3/iris/index.html

declining trend should continue since there was a total lead phase-out from highway gasoline and its additives that went into effect in January 1996. Other airborne sources include combustion of solid waste, coal, and oils, emissions from iron and steel production, lead smelters, and tobacco smoke.[92]

Exposure to lead can also occur from food and soil. Children are at particular risk to lead exposure since they commonly put hands, toys, and other items, that may come in contact with lead-containing dust and dirt in their mouths. Lead-based paints were commonly used for many years and flaking paint, paint chips, and weathered paint powder may be a major source of lead exposure, particularly for children. Lead in drinking water is due primarily to the presence of lead in certain pipes, solder, and fixtures. Exposure to lead may also occur in the workplace, such as lead smelting and refining industries, steel and iron factories, gasoline stations, and battery manufacturing plants.[93] Lead has been listed as a pollutant of concern in EPA's Great Waters Program due to its persistence in the environment, potential to bioaccumulate, and toxicity to humans and the environment.[94]

Human studies are inconclusive regarding lead exposure and cancer, while animal studies have reported an increase in kidney cancer from lead exposure by the oral route. The EPA considers lead to be a Group B2, probable human carcinogen based on sufficient data in animals.[95,96]

Lead is a very toxic element, causing a variety of effects at low dose levels. Brain damage, kidney damage, and gastrointestinal distress are seen in humans receiving acute (short-term) exposure to high levels of lead. Chronic (long-term) exposure to lead affects the blood, central nervous system (CNS), blood pressure, kidneys, and Vitamin D metabolism in humans. Children are particularly sensitive to the chronic effects of lead, with slowed cognitive development, reduced growth and other effects reported. Reproductive effects, such as decreased sperm count in men and spontaneous abortions in women, have been associated with lead exposure. The developing fetus is at particular risk from maternal lead exposure, with low birth

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[92] ATSDR. 1999. Toxicological Profile for Lead (Update). USDHHS, PHS, ATSDR.

[93] ATSDR. 1999. Toxicological Profile for Lead (Update). USDHHS, PHS, ATSDR.

[94] EPA. 1997. Deposition of Air Pollutants to the Great Waters-Second Report to Congress, Office of Air Quality Planning and Standards, June 1997, EPA-453/R-97-011.

[95] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[96] EPA. 1989. Evaluation of the potential carcinogenicity of lead and lead compounds: In support of reportable quantity adjustments pursuant to CERCLA Section 102. Prepared by the Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-89/045A. (External Review Draft).

weight and slowed postnatal neurobehavioral development noted.[97] EPA has found the noncancer health effects sufficient to warrant the establishment of a National Ambient Air Quality Standard (NAAQS) for Lead.[98]

M. Manganese Compounds

Manganese is a naturally occurring substance found in many types of rock and soil; it is ubiquitous in the environment and found in low levels in water, air, soil and food. Manganese can also be released into the air by iron and steel production plants, power plants, and coke ovens. The average manganese levels in various media are as follows: levels in drinking water are approximately 0.004 ppm; average air levels are approximately 0.02 $\mu g/m^3$; levels in soil range from 40 to 900 ppm; the average daily intake from food ranges from 1 to 5 mg/d. People who work in factories where manganese metal is produced from manganese ore or where manganese compounds are used to make steel or other products are most likely to be exposed through inhalation to higher than normal levels of manganese.[99] Manganese compounds from mobile sources comprise less than 2 percent of the 1996 National Toxics Inventory.

No studies are available regarding the carcinogenic effects of manganese in humans, and animal studies are inadequate. Based on this information, EPA has classified manganese as a Group D carcinogen (not classifiable as to carcinogenicity in humans).[100]

Key health effects concerns in humans have been associated with neurotoxic and perhaps developmental effects. Chronic exposure to high levels of manganese by inhalation in humans results primarily in central nervous system (CNS) effects. Visual reaction time, hand steadiness, and eye-hand coordination were affected in chronically-exposed workers. A syndrome named manganism may result from chronic exposure to higher levels; manganism is characterized by feelings of weakness and lethargy, tremors, a mask-like face, and psychological disturbances. Respiratory effects have also been noted in workers chronically exposed by inhalation. Based on these occupational studies, EPA has found the data to be sufficient to develop a RfC for manganese oxides and salts exposure. Impotence and loss of libido have been noted in male workers afflicted with manganism attributed to high-level inhalation exposures to manganese.

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[97] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[98] EPA. 1986. Air Quality Criteria Document for Lead. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC, for the Office of Air Quality Planning and Standards. EPA-600/8-83/028dF.

[99] EPA 1994. EPA Health Effects Notebook for Hazardous Air Pollutants-Draft, EPA-452/D-95-00, December 1994, Office of Air Quality Planning and Standards, RTP, NC. http://www.epa.gov/ttn/uatw/hapindex.html

[100] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

Animal studies have reported reproductive effects such as sterility and developmental effects such as decreased activity levels in the offspring of animals exposed to manganese.[101,102]

In December, 1995, the fuel additive methylcyclopentadienyl manganese tricarbonyl (MMT), an octane enhancer commercially labeled as HiTEC 3000, became legal to blend into unleaded gasoline in the U.S. The approved fuel waiver for MMT allows up to 0.03125 (1/32) gram per gallon manganese (60 FR 36414, July 17, 1995). Ethyl Corporation is still required to perform health research but is able to do the research while the product is marketed. On May 19 2000, the Agency notified Ethyl Corporation of the final test program requiring emission and health effects testing for the gasoline additive MMT, in accordance with the Alternative Tier 2 provision of the fuels and fuel additives health effects testing regulations.[103] The Alternative Tier 2 health effects testing will give the general public a greater awareness of the comparative risks associated with inhalation exposures to gasoline fuels containing MMT. The Alternative Tier 2 test requirements are within two general categories, pharmacokinetic testing of manganese compounds and characterization of manganese emissions from vehicles utilizing fuels containing MMT. These Alternative Tier 2 testing requirements are intended to be the first stage in a two-stage Alternative Tier 2 test program. EPA intends to evaluate the results produced in the first stage of testing, as well as any other information which may be submitted to or obtained by EPA in the meantime, in determining the specific nature and scope of the second stage of Alternative Tier 2 testing. Any additional Alternative Tier 2 tests proposed for fuel and additives containing MMT in the future will be announced in a separate Federal Register notice. The docket number for the MMT Alternative Tier 2 testing requirements is A-98-35. Section 211 allows for the development of a Tier 3 set of tests, if necessary, to further answer questions related to these fuels in the interest of protecting public health.

N. Mercury Compounds

Mercury exists in three forms: elemental mercury, inorganic mercury compounds (primarily mercuric chloride), and organic mercury compounds (primarily methyl mercury). All forms of mercury are quite toxic, and each form exhibits different health effects. Elemental mercury is used in thermometers, barometers, and pressure-sensing devices. It is also used in batteries, lamps, industrial processes, refining, lubrication oils, and dental amalgams. Inorganic mercury was used in the past in laxatives, skin-lightening creams and soaps, and in latex paint. In 1990, EPA canceled registration for all interior paints that contained mercury. Mercury use in exterior paint was discontinued after 1991. Methyl mercury has no industrial uses; it is formed in

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[101] ATSDR. 1990. Toxicological Profile for Manganese (Draft). U.S. Public Health Service, U.S. Department of Health and Human Services, Altanta, GA.

[102] EPA. 1994. ORD's "Re-evaluation of Inhalation Health Risks Associated with MMT in Gasoline, July 1, 1994". Office of Research and Development, Washington, D.C.

[103] The fuels and fuel additives testing program regulations are codified at 40 CFR part 79, subpart F. The Alternative Tier 2 provisions appear at 40 C.F.R. § 79.58(c).

the environment from the methylation of the inorganic mercurial ion.[104, 105]

The most recent data, for various varieties of gasoline vehicles and heavy duty diesel vehicles, showed negligible emissions of elemental mercury and no indication that inorganic mercury is emitted using laboratory test cycles.[106] Analytical methods typically used to collect and measure mercury from mobile sources are not sensitive enough to measure trace level emissions. Thus, if data were developed using appropriate collection media and sensitive analytical methods, such as cold-vapor atomic fluorescence, mercury may be detected in mobile source emissions.[107] Current inventory estimates of mobile source mercury emissions rely on an older database that speciates PM emissions from mobile sources.[108] For one category of vehicle, light duty diesel vehicles, no recently developed emissions factors were available, so we used factors developed in the 1987 South Coast Air Quality Management District study. The mobile source emissions estimate, therefore has zero emissions from gasoline powered vehicles and heavy duty diesels, but there were emission estimates for light duty diesels. There is no reason to believe that light duty vehicles are uniquely emitters of mercury among all mobile sources; the more likely explanation is a measurement artifact. Mercury emissions from mobile sources are traditionally not speciated and are presented as total elemental mercury emissions. Mercury compounds from mobile sources comprise less than 4 percent of the 1996 National Toxics Inventory.

A major source of exposure for elemental mercury is through inhalation in occupational settings.[109] Mercury has been listed as a pollutant of concern in EPA's Great Waters Program due to its persistence in the environment, potential to bioaccumulate, and toxicity to humans and the environment.[110]

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[104] ATSDR. 1999. Toxicological Profile for Mercury (update). USDHHS, PHS, ATSDR.

[105] EPA. 1997. Mercury Study Report to Congress. Volume II: An Inventory of Anthropogenic Mercury Emissions in the United States. EPA-452/R-97-004. http://www.epa.gov/oar/mercury.html

[106] Ball, James C. Emission Rates and Elemental Composition of Particles Collected from 1995 Ford Vehicles Using the Urban Dynamometer Driving Schedule, the Highway Fuel Economy Test, and the US06 Driving Cycle. Society of Automotive Engineers, SAE paper No. 97FL-376. 1997.

[107] Schroeder, W.H., Hamilton, M.C. and Stobart, S.R. (1985) The use of noble metals as collection media for mercury and its compounds in the atmosphere. Revs. In Anal. Chem. 8:179-209.

[108] Cooper. J.A. et al., NEA, INC. PM10 for the South Coast Air Basin, Volumes I and II. Prepared for the South Coast Air Quality Management District, El Monte, CA. July 15, 1987

[109] EPA. 1997. Mercury Study Report to Congress. Volume IV: An Assessment of Exposure to Mercury in the United States. EPA-452/R-97-006. http://www.epa.gov/oar/mercury.html

[110] EPA. 1997. Deposition of Air Pollutants to the Great Waters-Second Report to Congress, Office of Air Quality Planning and Standards, June 1997, EPA-453/R-97-011.

Human and animal studies are inconclusive regarding the carcinogenicity of elemental mercury. Based on this data, the EPA has classified elemental mercury as a Group D, not classifiable as to human carcinogenicity.[111] Mercuric chloride is categorized as a Group C, possible human carcinogen.

Acute (short-term) exposure to high levels of elemental mercury in humans results in central nervous system (CNS) effects such as tremors, mood changes, and slowed sensory and motor nerve function. High inhalation exposures can also cause kidney damage. Acute inhalation exposure also has effects on the gastrointestinal tract and respiratory system in humans. Chronic (long-term) inhalation exposure to elemental mercury in humans also affects the CNS, with effects such as erethism (increased excitability), irritability, excessive shyness, and tremors.[112] The data from these studies was found to be sufficient for EPA to develop a RfC for elemental mercury exposure. Additional data from oral exposure studies was found to be sufficient for EPA to also develop a RfD for elemental mercury oral exposure.

O. MTBE

Methyl *tert*-butyl ether (MTBE) is a colorless liquid that has been used in the United States since the late-1970's as an octane-enhancing replacement for lead. Currently, MTBE's main use is as a fuel oxygenate as part of the Wintertime Oxygenated Fuel and Federal reformulated gasoline (RFG) programs. MTBE emissions from mobile sources account for approximately 86 percent of the total MTBE inventory in the 1996 National Toxics Inventory. Human exposure to MTBE may occur via inhalation, ingestion, or dermal contact.

The majority of the research on the health effects of MTBE to date has focused on the effects of inhalation exposure. By the inhalation route, MTBE has been found to cause increases in liver and kidney weights and increased severity of spontaneous kidney lesions, as well as swelling around the eyes and increased prostration in laboratory rats[113]. These effects were cited as the basis for EPA's inhalation reference concentration (RfC) for MTBE. In addition to non-cancer effects, long-term inhalation exposure to high concentrations of MTBE has been associated with tumors in the kidneys and testes of male rats and in the liver of female mice.[114]

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[111] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

[112] EPA. 1997. Mercury Study Report to Congress. Volume V: Health Effects of Mercury and Mercury compounds. EPA-452/R-97-007. http://www.epa.gov/oar/mercury.html

[113] EPA 1993. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

[114] Bird, M. G.; Burleigh-Flayer, H. D.; Chun, J. S.; Douglas, J. F.; Kneiss, J. J.; Andrews, L. S. (1997) Oncogenicity studies of inhaled methyl tertiary-butyl ether (MTBE) in CD-1 mice and F-344 rats. J. Appl. Toxicol.

However, there have been no human or animal health effects studies concerning the ingestion of MTBE in drinking water. In one study, animals were given MTBE in olive oil by gavage for up to 24 months.[115,116] Lymphomas and leukemia were observed in the female rats and testicular tumors in the male rats.

A report by EPA's Office of Research and Development concluded that, under the 1986 EPA cancer risk assessment guidelines, inhalation cancer test results would support placing MTBE in Group C as a "possible human carcinogen."[117] An Interagency Assessment of Oxygenated Fuels similarly concluded that "While there are no studies on the carcinogenicity of MTBE in humans, there is sufficient evidence to indicate that MTBE is an animal carcinogen and to regard MTBE as having a human hazard potential. However, estimates of human risk from MTBE contain large uncertainties in both human exposure and cancer potency."[118] Other public health bodies, such as the International Agency for Research on Cancer[119] and the U.S. Department of Health and Human Services[120] have indicated that there is not enough information to classify MTBE with regard to human carcinogenicity under their classification schemes. The Agency will continue to reevaluate the potential carcinogenicity of MTBE as new information becomes available.

P. Naphthalene

Naphthalene occurs as a white solid or powder that is insoluble in water. It has a strong, mothball odor. The primary use for naphthalene is in the production of phthalic anhydride. Other uses include carbamate insecticides, surface active agents and resins, dye intermediates,

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17(suppl. 1): S45-S55.

[115] Belpoggi F., Soffritti M and Maltoni C. 1995. Methyl-tertiary-butyl ether (MTBE)- a gasoline additive - causes testicular and haematopoietic cancers in rats. Toxicol Ind Health 11:119-149.

[116] Belpoggi, F.; Soffritti, M.; Maltoni, C. (1998) Pathological characterization of testicular tumours and lymphomas-leukaemias, and of their precursors observed in Sprague-Dawley rats exposed to methyl-tertiary-butyl-ether (MTBE). Eur. J. Oncol. 3: 201-206.

[117] EPA. 1994. Health risk perspectives on fuel oxygenates. Washington, DC: Office of Research and Development; report no. EPA 600/R-94/217.

[118] Interagency Oxygenated Fuels Assessment Steering Committee. 1997. Interagency assessment of oxygenated fuels. Washington, DC: National Science and Technology Council, Committee on Environment and Natural Resources and Office of Science and Technology Policy. http://www.epa.gov/otaq/fuels.html.

[119] International Agency for Research on Cancer. 1999. Methyl tert-butyl ether (group 3). http://193.51.164.11/htdocs/Monographs/Vol73/73-13.html.

[120] National Institute of Environmental Health Sciences, National Toxicology Program. 2000. Summary of RG1, RG2, and NTP Board Subcommittee Recommendations for the Report on Carcinogens. Ninth Edition. http://ehis.niehs.nih.gov/roc/toc9.html

synthetic tanning agents, and moth repellents. Naphthalene is found in small quantities in gasoline and diesel fuels. Naphthalene emissions have been measured in larger quantities in both gasoline and diesel exhaust and evaporative emissions from mobile sources. Individuals may be exposed to naphthalene through the use of mothballs. Workers may be occupationally exposed during its manufacture and use, especially in coal-tar production, wood preserving, tanning, or ink and dye production. Coal tar production, wood preserving, and other industries release small amounts. Naphthalene has also been detected in tobacco smoke.[121]

Workers occupationally exposed to vapors of naphthalene and coal tar developed laryngeal carcinomas or neoplasms of the pylorus and cecum. Di-, tri-, and tetramethyl naphthalene contaminants of coal tar were found to be carcinogenic when applied to the skin of mice, but naphthalene alone was not. An increased number of lung adenomas were reported in mice exposed by inhalation, but this was not dose-related. No carcinogenic responses were reported in rats exposed to naphthalene in their diet and by injection. The human carcinogenic potential of naphthalene via the oral or inhalation routes cannot be determined at this time based on human and animal data; however, there is suggestive evidence. EPA has classified naphthalene as a Group C, possible human carcinogen but no risk estimate can be derived due to the weak data set.[122]

Acute (short-term) exposure of humans to naphthalene by inhalation, ingestion, and dermal contact is associated with hemolytic anemia, damage to the kidneys, and, in infants, brain damage. Symptoms of acute exposure include headache, nausea, vomiting, diarrhea, malaise, confusion, anemia, jaundice, convulsions, and coma. Cataracts have also been reported in workers acutely exposed to naphthalene by inhalation and ingestion. Chronic (long-term) results from rodent studies, supported by other subchronic and acute studies, identify nasal and respiratory lesions as critical effects from chronic inhalation exposure to naphthalene. The data from these animal studies was found to be sufficient for EPA to develop a RfC for naphthalene exposure. Additional data from rat oral exposure studies was found to be sufficient for EPA to also develop a RfD for naphthalene oral exposure.[123]

Q. Nickel Compounds

Nickel is a natural element of the earth's crust; as a result, small amounts are found in food, water, soil, and air. Food is the major source of nickel exposure, with an average intake for

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[121] EPA. 1998. Toxicological Review of Naphthalene, In support of Summary Information on the Integrated Risk Information system. Washington, D.C. http://www.epa.gov/ngispgm3/iris/subst/0436.htm

[122] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

[123] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

adults estimated to be approximately 100 to 300 µg/d. Individuals also may be exposed to nickel in occupations involved in its production, processing, and use, or through contact with everyday items such as nickel-containing jewelry and stainless steel cooking and eating utensils, and by smoking tobacco. Nickel is found in ambient air at very low levels as a result of releases from oil and coal combustion, nickel metal refining, sewage sludge incineration, manufacturing facilities, and other sources. Nickel compounds have also been identified in trace quantities in exhaust emissions from gasoline and diesel engines.[124] Nickel compounds from mobile sources comprise less that nine percent of the 1996 National Toxics Inventory.

The EPA has not evaluated soluble salts of nickel as a class of compounds for potential human carcinogenicity. Human and animal studies have reported an increased risk of lung and nasal cancers from exposure to nickel refinery dusts and nickel subsulfide. EPA has classified nickel refinery dust and nickel subsulfide as Group A, human carcinogens and has completed a formal risk characterization for nickel and nickel compounds.[125] Sufficient data from animal studies of soluble nickel compounds (i.e., nickel carbonyl) have reported lung tumors though the human data is insufficient. EPA has classified nickel carbonyl as a Group B2, probable human carcinogen, and given its high instability, nickel carbonyl exposure is extremely rare.[126]

Nickel dermatitis, causing itching of the fingers, hands, and forearms, is the most common effect in humans from chronic (long-term) skin contact with nickel. Respiratory effects have also been reported in humans from inhalation exposure to nickel. No information is available regarding the reproductive or developmental effects of nickel in humans, but animal studies have reported reproductive and developmental effects. The data from these studies was found to be insufficient for EPA to develop a RfC for nickel exposure. Additional data from animal oral exposure studies was found to be sufficient for EPA to also develop a RfD for nickel soluble salts exposure.[127]

R. POM (Polycyclic Organic Matter)

Polycyclic organic matter, or POM, defines a broad class of compounds that includes the polycyclic aromatic hydrocarbon compounds (PAHs), of which benzo[a]pyrene is a member.

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[124] ATSDR. 1997. Toxicological Profile for Nickel (Update). USDHHS, PHS, ATSDR

[125] EPA. 1986. Health Assessment Document for Nickel and Nickel Compounds. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/600/8-83/012FF.

[126] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

[127] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

The primary source of POM is formation during combustion. A less significant formation mechanism is the volatilization of light weight POM compounds, which occurs in the production and use of naphthalene. Polycyclic organic compounds have been detected in ambient air from sources including cigarette smoke, gasoline and diesel engine exhausts, asphalt road paving, coal burning, application of coal tar, agricultural burning, residential wood burning, and hazardous waste sites. The compounds present in POM and their relative amounts differ among different sources (e.g, POM from diesel exhaust is chemically different than POM from wood burning).[128] POM from mobile source particulate, as the sum of the seven PAHs that are probable human carcinogens, accounts for approximately six percent of the 1996 National Toxics Inventory.

PAHs have been found in some drinking water supplies. Cooking meat or other foods at high temperatures increases the amount of PAHs in the food. Occupational exposure to PAHs may occur in coal tar production plants, coking plants, coal-gasification sites, smokehouses, municipal trash incinerators, and other facilities. POM has been listed as a pollutant of concern in EPA's Great Waters Program due to its persistence in the environment, potential to bioaccumulate, and toxicity to humans and the environment.[129]

Skin exposures to mixtures of carcinogenic PAHs cause skin disorders in humans and animals. No information is available on the reproductive or developmental effects of POM in humans, but animal studies have reported that oral exposures to benzo[a]pyrene causes reproductive and developmental effects. Cancer is the major concern from exposure to POM. Epidemiologic studies have reported an increase in lung cancer in humans exposed to coke oven emissions, roofing tar emissions, and cigarette smoke; all of these mixtures contain POM compounds. Animal studies have reported respiratory tract tumors from inhalation exposure to benzo[a]pyrene and forestomach tumors, leukemia, and lung tumors from oral exposure to benzo[a]pyrene.[130] The EPA has classified seven PAHs (benzo[*a*]pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene) as Group B2, probable human carcinogens based only on sufficient animal data.[131] No formal risk characterization has been conducted.

EPA has determined that the data set is insufficient for EPA to develop either a RfC or a RfD for POM exposure.

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[128] ATSDR. 1995. Toxicological Profile for Poylcyclic Aromatic Hydrocarbons (PAHs) (Update). USDHHS, PHS, ATSDR.

[129] EPA. 1997. Deposition of Air Pollutants to the Great Waters-Second Report to Congress, Office of Air Quality Planning and Standards, June 1997, EPA-453/R-97-011.

[130] EPA. 1991. Dose-Response Analysis of Ingested Benzo[a]pyrene (CAS No. 50-32-8). Human Health Assessment Group, Office of Health and Environmental Assessment, Washington, DC. EPA/600/R-92/045.

[131] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1999.

S. Styrene

Styrene is a colorless liquid that has a sweet smell. It is used predominately in the production of polystyrene and resins. Styrene is also used as an intermediate in the synthesis of materials used for ion exchange resins and to produce copolymers. Styrene is also emitted in significant quantities in the exhaust gases of both gasoline and diesel powered engines.[132] Styrene from mobile sources accounts for approximately 40 percent of the 1996 National Toxics Inventory.

Indoor air is the principal route of styrene exposure for the general population, due to building materials, consumer products, and tobacco smoke. Occupational exposure to styrene occurs in the reinforced plastics industry and polystyrene factories.[133]

Several epidemiologic studies suggest there may be an association between styrene exposure and an increased risk of leukemia and lymphoma. However, the evidence is inconclusive due to confounding factors. Animal studies have produced both negative and positive results. EPA is currently assessing the potential of styrene to cause cancer.

Acute (short-term) exposure to styrene results in mucous membrane and eye irritation, and gastrointestinal effects in humans. Chronic (long-term) exposure of humans to styrene results in effects on the central nervous system (CNS), such as headache, fatigue, weakness, depression, peripheral neuropathy, minor effects on some kidney enzyme functions and on the blood. Human studies are inconclusive on the reproductive and developmental effects of styrene; several studies did not report an increase in developmental effects in women who worked in the plastics industry, while an increased frequency of spontaneous abortions and decreased frequency of births were reported in another study. respiratory lesions as critical effects from chronic inhalation exposure to naphthalene. The data from human studies was found to be sufficient for EPA to develop a RfC for styrene exposure. Additional data from animal oral exposure studies was found to be sufficient for EPA to also develop a RfD for styrene oral exposure.[134]

T. Toluene

Toluene occurs as a colorless, flammable, refractive liquid that is slightly soluble in water. It has a sweet, pungent odor. The major use of toluene is as a mixture added to gasoline to improve octane ratings. Toluene is also used to produce benzene and as a solvent in paints, coatings, adhesives, inks, and cleaning agents. It is used in the production of polymers used to

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[132] ATSDR. 1992. Toxicological Profile for Styrene. USDHHS, PHS, ATSDR TP-91/25.

[133] ATSDR. 1992. Toxicological Profile for Styrene. USDHHS, PHS, ATSDR TP-91/25.

[134] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

make nylon, plastic soda bottles, and polyurethanes, and for pharmaceuticals, dyes, cosmetic nail products, and the synthesis of organic chemicals. The highest concentrations of toluene usually occur in indoor air from the use of common household products (paints, paint thinners, and adhesives) and cigarette smoke. The deliberate inhalation of paint or glue by solvent abusers may produce high levels of exposure to toluene, as well as to other chemicals. Toluene exposure may also occur in the workplace, especially in occupations such as printing or painting, where toluene is frequently used as a solvent.[135]

Mobile sources are the principal source of toluene to the ambient air. Toluene is found in both gasoline and diesel fuel as well as the exhaust emissions of both types of engines. Toluene from mobile sources accounts for approximately 74 percent of the 1996 National Toxics Inventory. Toluene can also be released to the ambient air during the production, use, and disposal of industrial and consumer products that contain toluene.

None of the data suggest that toluene is carcinogenic. Two epidemiological studies did not detect a statistically significant increased risk of cancer due to inhalation exposure to toluene. However, these studies had many confounding factors. Animal studies have been negative for carcinogenicity. Considering no human data and inadequate animal data, and the lack of positive results in the majority of genotoxic assays, EPA has classified toluene as a Group D compound (not classifiable as to human carcinogenicity).[136]

The central nervous system (CNS) is the primary target for toluene toxicity in both humans and animals for acute (short-term) and chronic (long-term) exposures. CNS dysfunction (which is often reversible) and narcosis have been frequently observed in humans acutely exposed to low or moderate levels of toluene by inhalation; symptoms include fatigue, sleepiness, headaches, and nausea. Cardiac arrhythmia has also been reported in humans acutely exposed to toluene. CNS depression has been reported to occur in chronic abusers exposed to high levels of toluene. Symptoms include ataxia, tremors, cerebral atrophy, nystagmus (involuntary eye movements), and impaired speech, hearing, and vision. Chronic inhalation exposure of humans to toluene also causes irritation of the upper respiratory tract, eye irritation, sore throats, nausea, skin conditions, dizziness, headaches, and difficulty with sleep.[137]

Human studies have also reported developmental effects, such as CNS dysfunction, attention deficits, and minor craniofacial and limb anomalies, in the children of pregnant women exposed to toluene or mixed solvents by inhalation. Reproductive effects, including an

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[135] ATSDR 1995. Toxicological Profile for Toluene (Update). USDHHS, PHS, ATSDR.

[136] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

[137] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

association between paternal exposure to toluene and an increased odds ratio for spontaneous abortions but not birth defects, have also been noted. However, these studies are not conclusive due to many confounding variables. Animal studies have shown toluene to have developmental, but not reproductive, effects from inhalation exposure. The data from human and animal studies was found to be sufficient for EPA to develop a RfC for toluene exposure. Additional data from animal oral exposure studies was found to be sufficient for EPA to also develop a RfD for toluene oral exposure.[138]

U. Xylene

Mixed xylenes are colorless liquids with a sweet odor. They are used in the production of ethylbenzene, in solvents, and for paints and coatings. They are also blended into gasoline and are also present in diesel fuel. Xylenes are emitted in the exhaust emissions of both gasoline and diesel powered engines accounting for 78 percent of the 1996 National Toxics Inventory. Xylenes are distributed throughout the environment; they have been detected in air, rainwater, soils, surface water, sediments, drinking water, and aquatic organisms. Xylenes have also been detected in indoor air; xylenes have been widely used in home use products such as paints. Occupational exposure to mixed xylenes may occur at workplaces where mixed xylenes are produced and used as industrial solvents.[139]

No information is available on the carcinogenic effects of mixed xylenes in humans, and animal studies have reported negative results from exposure via gavage (experimentally placing the chemical in the stomach). Considering there is no human data and the animal data is negative, EPA has classified mixed xylenes as a Group D compound (not classifiable as to human carcinogenicity).[140]

Acute (short-term) inhalation exposure to mixed xylenes in humans results in irritation of the nose and throat, gastrointestinal effects such as nausea, vomiting, and gastric irritation, mild transient eye irritation, and neurological effects. Chronic (long-term) inhalation exposure of humans to mixed xylenes results primarily in central nervous system (CNS) effects, such as headache, dizziness, fatigue, tremors and uncoordination. Other effects noted include labored breathing and impaired pulmonary function, increased heart palpitation, severe chest pain and an abnormal EKG, and possible effects on the blood and kidney. This data has been determined by EPA to be insufficient to calculate a RfC. Insufficient data are available on the developmental or reproductive effects of mixed xylenes in humans. Animal studies have reported developmental

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[138] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

[139] ATSDR. 1995. Toxicological Profile for Xylenes (Update). USDHHS, PHS, ATSDR.

[140] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

effects, such as an increased incidence of skeletal variations in fetuses, and fetal resorptions via inhalation. Additional data from animal oral exposure studies was found to be sufficient for EPA to develop a RfD for oral xylene exposure.[141]

[141] EPA 1999. Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. http://www.epa.gov/ngispgm3/iris/index.html

Chapter 4: Impacts of Motor Vehicle Emission Control Programs on MSAT Emissions

In Chapter 2 of the TSD we identified the 21 MSATs. We now turn to an evaluation of the impact of existing and proposed controls on inventories of those air toxics by examining the emissions inventories and estimated reductions expected to be achieved by our various mobile source control programs.

The data and information available on emissions of these 21 MSATs vary considerably. While we have 1996 inventory data for all of the MSATs except naphthalene, we do not have inventory projections for all of them. Therefore, we are examining the projected impacts of our current mobile source control program by groupings of air toxics. More specifically, we have projections of future emissions for five gaseous toxics (benzene, formaldehyde, 1,3-butadiene, acetaldehyde, MTBE) and for diesel PM (as the surrogate for diesel PM and diesel exhaust organic gases) and we present these in this section. We do not have emissions projections for the remaining gaseous toxics (acrolein, POM, styrene, toluene, xylene, ethylbenzene, naphthalene, and n-hexane) but, because these compounds are part of VOCs, we believe it is reasonable to utilize VOC emissions inventory projections to estimate the expected impact of our control programs on these other gaseous MSATs. Finally, we also do not have emissions inventory projections for the metals on the MSAT list (arsenic compounds, chromium compounds, mercury compounds, nickel compounds, manganese compounds, and lead compounds) or for dioxins/furans. While metal emissions and dioxin/furans emissions are associated with particles and it is possible that some of these compounds track PM emissions to some extent, we do not have good data on these relationships. Therefore, we are not presenting emission projections for these compounds in this analysis. We believe this is reasonable because the mobile source contribution to metals inventories is small and comes primarily from engine wear and impurities in engine oil, or from fuel additives.

As we describe in the following discussion, there have been and will continue to be significant reductions in MSATs as a result of on-highway emission control regulations. By 2020, we project on-highway emission control programs (up to and including our Tier 2 control program and our recently proposed 2007 heavy-duty engine rule) will reduce benzene emissions by 73 percent, formaldehyde emissions by 76 percent, 1,3-butadiene emissions by 72 percent, and acetaldehyde emissions by 67 percent from 1990 levels. Under these controls we project on-highway diesel PM emissions will be reduced by 94 percent by 2020, as compared with 1990 levels.

This chapter consists of two parts. First, we describe two previous EPA studies that have estimated inventories of MSAT emissions. Second, we describe the methodologies used for this rulemaking to develop our toxics emissions inventories, including our estimates of how our current and proposed on-highway emission control programs will reduce MSAT emissions in the future.

A. Previous EPA Studies of Toxics Emissions Inventories

The following section presents a discussion of two previous EPA studies that have estimated toxics emissions inventories from mobile sources.

1. The 1999 EPA Motor Vehicle Air Toxics Study

We developed inventory estimates for several gaseous MSATs (acetaldehyde, benzene, 1,3-butadiene, formaldehyde, MTBE) and also for diesel PM as part of the 1999 EPA Motor Vehicle Air Toxics Study, "Analysis of the Impacts of Control Programs on Motor Vehicle Toxic Emissions and Exposure in Urban Areas and Nationwide,"[142] (hereafter referred to as the 1999 EPA Motor Vehicle Air Toxics Study, or the 1999 Study). The six pollutants examined in the 1999 Study were chosen becuase we had adequate data to perform a rigorous modeling analysis for those pollutants (examining the impact of fuel properties, emission control technologies, and type of in-use operation on the emission inventories for these six pollutants). The modeling performed for the 1999 Study form the basis of the inventories presented in Section B. of this chapter.

The 1999 EPA Motor Vehicle Air Toxics Study provided estimates of toxics emission inventories for these compounds for 1990, 1996, 2007, and 2020. The 1990 inventories reflected toxics emissions before any of the programs added by the 1990 Clean Air Act Amendment were implemented. The 1996 inventories reflected the impact of some of the new Clean Air Act programs, such as Phase 1 of the RFG program. The 2007 and 2020 inventories were intended to reflect the impact of all of our mobile source regulations under development at the time the study was completed (including the Tier 2 standards for light-duty and the proposed 2007 standards for heavy-duty engines).

As noted above, the inventory estimates presented in Section B. of this chapter are based on the inventories presented in the 1999 Study. A number of updates, as described in Section B. of this chapter, have been made to account for updated information for heavy-duty engines.

2. The 1996 National Toxics Inventory

The 1996 National Toxics Inventory (NTI) prepared in connection with the Agency's National Air Toxic Assessment (NATA), contains 1996 emission estimates for all 21 MSATs, except naphthalene.[143] The 1996 NTI contains 1996 emissions estimates for both on-highway

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[142] EPA. 1999. Analysis of the Impacts of Control Programs on Motor Vehicle Toxics Emissions and Exposure in Urban Areas and Nationwide. Prepared for U. S. EPA, Office of Transportation and Air Quality, by Sierra Research, Inc., and Radian International Corporation/Eastern Research Group. Report No. EPA 420 –R-99-029/030.

[143] Naphthalene emissions are not reported in the 1996 NTI separately from 16-PAH.

and nonroad sources.[144] (Because diesel exhaust is not included on the list of 112(b) hazardous pollutants that are the focus of the 1996 NTI, diesel PM estimates were not compiled in the 1996 NTI.) Table IV.A-1 presents the MSAT inventories from the 1996 NTI and indicates the on-highway and nonroad percentages of the national inventories for each of the MSATs. The percentages are based on the total national inventories which include on-highway and nonroad mobile sources, major and area stationary sources, and other sources such as forest fires.

[144] The nonroad inventory in the 1996 NTI includes emissions data for a number of nonroad categories including aircraft. Under the Clean Air Act definition, nonroad vehicles do not include aircraft. For convenience, in this document the term "nonroad" will generally include aircraft. It should be noted that the nonroad emissions estimates contained in the 1996 NTI are based on the draft NONROAD model, and, therefore, are subject to change.

Table IV.A-1
1996 On-Highway and Nonroad Emission Inventories of Some MSATs from the 1996 National Toxics Inventory (short tons)

Compound	On-Highway		Nonroad		Mobile Sources	
	Tons	Percent of Total National Emissions	Tons	Percent of Total National Emissions	Tons	Percent of Total National Emissions
1,3-Butadiene*	23,500	42%	9,900	18%	33,400	60%
Acetaldehyde*	28,700	29%	40,800	41%	69,500	70%
Acrolein*	5,000	16%	7,400	23%	12,400	39%
Arsenic Compounds*	0.25	0.06%	2.01	0.51%	2.26	0.57%
Benzene*	168,200	48%	98,700	28%	266,900	76%
Chromium Compounds*	14	1.2%	35	3%	49	4.2%
Dioxins/Furans*[145]	0.0001	0.2%	N.A.	N.A.	0.0001	0.2%
Ethylbenzene	80,800	47%	62,200	37%	143,000	84%
Formaldehyde*	83,000	24%	86,400	25%	169,400	49%
Lead Compounds*	19	0.8%	546	21.8%	565	22.6%
Manganese Compounds*	5.8	0.2%	35.5	1.3%	41.3	1.5%
Mercury Compounds*	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*	10.7	0.9%	92.8	7.6%	103.5	8.5%
POM (as sum of 7 PAH)*	42.0	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%

* Also on the urban HAPs list for the Integrated Urban Air Toxics Strategy (64 FR 38706, July 19,1999).

[145]Mass given in tons of TEQ (toxic equivalency quotient). EPA's 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 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 NTI data reflect certain interesting characteristics of mobile source air toxics emissions. First, mobile sources account for the majority of the 1996 national inventory of three of the gaseous MSATs that are included on the urban HAP list.[146] These three are 1,3-butadiene (60 percent), acetaldehyde (70 percent), and benzene (76 percent). Mobile sources account for 39 percent of the national inventory of acrolein and 49 percent of the national inventory of formaldehyde, two other gaseous urban HAPs. All of these MSATs are formed as part of the combustion process. In addition, benzene is also released through evaporative emissions from gasoline.

Second, with regard to the other MSATs that are included on the urban HAP list, the mobile source contribution generally is small (arsenic compounds, chromium compounds, manganese compounds, nickel compounds, POM, and dioxins/furans). The sole exception is lead compounds. Mobile sources contribute 23 percent of the national inventories of lead compound emissions due primarily to nonroad sources and, more specifically, to the use of a lead-additive package which boosts the octane of aviation gasoline.[147] The mobile source contribution to the other metals on the urban HAP list comes primarily from engine wear, from some fuel additives, or from impurities in engine oil.

With regard to the gaseous MSATs that are not included on the urban HAP list, mobile source contributions are high due to the presence of some of these compounds in gasoline (e.g., ethylbenzene, MTBE, n–hexane, toluene, and xylene).

In addition, mobile sources account for almost all diesel PM emissions. (A limited number of stationary sources, such as large generators, do operate on diesel fuel. Because there are relatively few stationary sources that operate on diesel fuel, we believe that diesel PM from stationary sources is relatively small compared to diesel PM from mobile sources. However, for this analysis we have not generated an estimate of diesel PM from stationary sources.) As shown later in this chapter, we estimate that 1996 on-highway diesel PM emissions are approximately 180,000 tons. We estimate that 1996 nonroad diesel PM emissions are approximately 346,000 tons, as discussed in Chapter 8 of this Technical Support Document.

B. Impacts of Motor Vehicle Emission Controls on Emission Inventories

Many of the programs that we have put in place since the passage of the 1990 Clean Air Act Amendments to achieve attainment of the National Ambient Air Quality Standards (NAAQS) for ozone, PM and CO have also reduced MSAT emissions. For example, measures to control hydrocarbons from motor vehicles are also effective in controlling gaseous toxics. In

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[146] This list can be found in the National Air Toxics Programs: The Integrated Urban Strategy; Notice. July 19, 1999, 64 Federal Register 38706-38740.

[147] Aviation gasoline is used by a relatively small number of aircraft, those with piston engines, which are generally used for personal transportation, sightseeing, crop dusting, and similar activities.

addition, certain programs address air toxics directly, such as the RFG program and the phase-out of leaded gasoline. We describe some of our key mobile source control programs in Chapter 1 of this Technical Support Document.

This section presents the inventories of MSATs and describes how we derived these inventories. To provide a framework for understanding our results, we first present an overview of our various inventory methodologies. We then present the emissions estimates for the five gaseous toxics addressed in the 1999 EPA Motor Vehicle Air Toxics Study. Next, we discuss our VOC emission trends as a surrogate to reflect the emission trends of other gaseous toxics for which we do not have specific inventory projections, including acrolein, POM, styrene, xylene, toluene, ethylbenzene, naphthalene, and n-hexane. We conclude by discussing the trend for diesel PM emissions.

We are not reporting inventory trends for the metals on our list of MSATs (arsenic compounds, chromium compounds, mercury compounds, nickel compounds, manganese compounds, and lead compounds) or for dioxins/furans. Metals in mobile source exhaust can come from fuel, fuel additives, engine oil, engine oil additives, or engine wear. Formation of dioxin and furans requires a source of chlorine. Thus, while metal emissions and dioxin/furan emissions are associated with particles, there are a number of other factors that contribute to emission levels. While it is possible that these compounds track PM emissions to some extent, we do not have good data on these relationships.

1. Overview of Inventory Methodologies

We analyzed emissions trends for gaseous air toxics addressed in the 1999 EPA Motor Vehicle Air Toxics Study (benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and MTBE), for VOC as a surrogate for the emissions trends for other gaseous air toxics, and for diesel PM. We estimated emissions for each of these classes of air toxics for four separate years (1990, 1996, 2007, and 2020). The methods used to estimate these emissions are summarized below and described in more detail in the following pages.

In the 1999 Study, we produced inventory estimates for various years and control scenarios that account for the effects of fuel and vehicle technology changes for five gaseous toxics. The inventories were calculated for different vehicles, including light-duty vehicles and trucks, and heavy-duty gasoline and diesel vehicles. For this rulemaking analysis, we used the light-duty vehicle and truck toxics emissions inventories from the 1999 Study directly to estimate emissions for all four years of interest. (The light-duty inventories used in this analysis include the effect of the Tier 2 program recently adopted by EPA). To calculate toxics emissions inventories for heavy-duty gasoline and diesel vehicles, we relied on inventories contained in the 1999 Study that included the impacts of all heavy-duty engine programs up until the 2004 model year standards, and adjusted the inventories to account for the impact of the recently proposed 2007 heavy-duty engine standards, including updated modeling information for heavy-duty engines. A description of the methodology used to project the heavy-duty gasoline and diesel vehicle toxic emissions inventories (assuming the proposed 2007 heavy-duty engine standards

take effect) is contained in section B.2.a. of this chapter.

We did not evaluate other gaseous air toxics in our 1999 Study. However, since all of these compounds are VOCs, we expect their emissions trends to follow the VOC emissions trend. For 1996 and later years, we developed VOC inventories assuming all of the on-highway control program, including the recent 2007 heavy-duty engine proposal, have taken effect. These VOC inventory estimates are based on our most up-to-date information on both light-duty vehicle and heavy-duty engine emissions factors and VMT developed for MOBILE6, and also reflect county-by-county information on VMT distribution by vehicle class, roadway type, and speed.

For 1990, we modified the modeling methods applied in the 1999 Study to produce direct estimates of VOC emissions that account for the effects of fuel and vehicle technology changes as well as the information that will be used in EPA's MOBILE6 emissions model.[148] We could not use the inventories presented in the 2007 heavy-duty engine proposal, because they do not extend back as far as 1990. Our other alternative was to use the 1990 VOC estimates from EPA's Trends Report. However, such estimates would not be comparable to the 1996 and later estimates, since EPA's 1990 Trends estimates have not been updated to reflect the data and analyses that will be used in MOBILE6.

Diesel PM inventories for 1996 and later years were developed assuming all of the on-highway control programs, including the recent 2007 heavy-duty engine proposal, have taken effect and included a number of modeling updates. The light-duty diesel PM emission estimates reflect the effects of the Tier 2 program for light-duty vehicles and trucks. The heavy-duty diesel PM emissions estimates incorporate recent findings on heavy-duty diesel engine PM emissions that were not reflected in the 1999 Study including updated emission factors and VMT. The modeling also considered county-by-county information on VMT distribution by vehicle class, roadway type, and speed.

The modeling performed for the 2007 heavy-duty engine proposal did not produce heavy-duty diesel PM estimates for 1990, so we chose to use the heavy-duty diesel PM inventory estimates for 1990 from EPA's Trends Report.[149] We believe this approach is reasonable, since we have not substantially changed our estimates of emissions from 1990 and earlier engines since the Trends Report estimates were developed. We also used data from the EPA's Trends Report for light-duty diesel PM estimates for 1990. The 1990 diesel PM inventories from EPA's Trends Report (presented in this analysis) and the diesel PM inventory from the 1999 Study are roughly comparable; the Trends Report inventory is 235,000 tons while the 1999 Study inventory is

[148] The analysis methodology is described in a memorandum from Meredith Weatherby, Eastern Research Group, to Rich Cook, EPA, entitled "Estimating of 1990 VOC and TOG Emissions" in EPA Air Docket A-2000-12.

[149] EPA. 2000. National Air Pollution Emission Trends, 1900-1998 (March 2000). Office of Air Quality Planning and Standards, Research Triangle Park, NC. Report No. 454/R-00-002.

202,000 tons. The 1999 Study results do not explicitly account for county-specific inputs, unlike Trends and the heavy-duty engine inventory presented in this analysis; furthermore, we consider it likely that the 1999 Study underestimates nationwide diesel PM emissions due to the way it extrapolates urban emissions to broader regions.

2. 1999 EPA Motor Vehicle Air Toxics Study

Section 202(l)(1) of the Clean Air Act calls on EPA to study the need for and feasibility of controlling toxic air pollutants associated with motor vehicles and motor vehicle fuels. We completed the study required under Section 202(l)(1) in April 1993. The report, entitled "Motor Vehicle-Related Air Toxics Study," is available on our website (www.epa.gov/otaq/toxics.htm).[150] Specific pollutants or pollutant categories which are discussed in this report include benzene, formaldehyde, 1,3-butadiene, acetaldehyde, diesel particulate, gasoline particulate, gasoline vapors as well as selected metals. The study focuses on carcinogenic risk although discussions of non-cancer effects for these and other pollutants are also included. The study provided estimates of emissions, exposure, and risk, with projections to the year 2010. Peer review comments on this study were received in 1994.[151] Peer review comments suggested improvements to EPA's exposure modeling and risk assessment methodology.

In response to these comments, EPA updated its exposure model for motor vehicle-related air toxics. Also, since 1993, significant new information on vehicle emission rates has been developed, and much more is known about the impact of fuel properties on toxic emissions. Moreover, EPA has updated its cancer risk assessment for benzene, and has released draft risk assessments for 1,3-butadiene and diesel exhaust emissions. Furthermore, EPA has developed new programs, such as the NLEV and Tier 2 standards, which significantly impact projections of toxic emissions, exposure, and risk.

In light of new information that was developed after 1993, and in response to peer review comments, EPA has updated estimates of emissions and exposure. The updated final emissions and exposure assessment, "Analysis of the Impacts of Control Programs on Motor Vehicle Toxics Emissions and Exposure in Urban Areas and Nationwide," (the 1999 EPA Motor Vehicle Air Toxics Study) was released in November, 1999.[152]

The remainder of this subsection provides additional information on how we developed the inventories contained in the 1999 Study. We also present the updated toxic emissions

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[150] EPA. 1993. Motor Vehicle-Related Air Toxics Study. Report No. EPA 420-R-93-005.

[151] Peer review comments on the 1993 study can be accessed at http://www.epa.gov/otaq/toxics.htm

[152] EPA. 1999. Analysis of the Impacts of Control Programs on Motor Vehicle Toxics Emissions and Exposure in Urban Areas and Nationwide. Prepared for U. S. EPA, Office of Transportation and Air Quality, by Sierra Research, Inc., and Radian International Corporation/Eastern Research Group. Report No. EPA 420 –R-99-029/030.

inventory results developed for this analysis assuming all currently promulgated mobile source control programs are in effect (including the Tier 2 program) as well as the proposed 2007 heavy-duty engine standards. While we addressed diesel PM emissions in the 1999 Study, we discuss the diesel PM emissions inventory projections in a later section, to reflect recent updates to the heavy-duty engine inventory modeling.

a. Methodology for Estimating Gaseous Mobile Source Air Toxic Emission Inventories

In the 1999 Study we estimated emissions of benzene, formaldehyde, acetaldehyde, 1,3-butadiene, and MTBE using a toxic emission factor model, MOBTOX5b. This model is based on a modified version of MOBILE5b, which estimates emissions of regulated pollutants, and essentially applies toxic fractions to total organic gas (TOG) estimates. The model accounted for differences in toxic fractions between technology groups, driving cycles, and normal versus high emitting vehicles and engines ("high emitters"). Impacts of fuel formulations were also addressed in the modeling. The TOG basic emission rates used in this modeling incorporated available elements from MOBILE6 used to develop the VOC inventory for the Tier 2 final rule. (The modeling did not incorporate impacts of evaporative emission standards in the Tier 2 rule which are expected to result in further reductions in evaporative emissions from light-duty vehicles and trucks. Therefore, for those toxics emissions which have an evaporative emissions component (i.e., benzene and MTBE), the 2007 and 2020 inventories presented in this chapter are slightly overestimated, and the reductions are slightly underestimated.)

We modeled toxic emissions for 10 urban areas and 16 geographic regions. These urban areas and geographic regions are listed in Table IV.B-1. They were selected to encompass a broad range of I/M programs, fuel parameters, and temperature regimes. The intent of the selection process was to best characterize the different combinations needed to perform accurate nationwide toxic emissions estimates. Every U. S. county in the country was then "mapped" to one of these modeled areas or regions (i.e., the emission factor for the modeled area was also used for the area "mapped" to it). Mapping was done based on a combination of geographic proximity, I/M program, and fuel control programs. Details of this process are provided in the 1999 Study. We then multiplied the resulting county-level emission factors by the county-level VMT estimates from EPA's Emission Trends Database and summed the results across all counties to come up with nationwide emissions in tons. This approach was also used to develop the inventory estimates in the 1996 NTI.[153]

Table IV.B-1
Metropolitan Areas and Regions Included in Toxic Emissions Modeling

Chicago, IL	Atlanta, GA	Florida

[153] Note that 1996 NTI estimates for the Northeast States were developed using VMT data supplied by those states, rather than estimates in the Emission Trends Database.

Denver, CO	Western WA/OR	Northeast States – non-I/M and non-RFG
Houston, TX	Northern CA	Northeast States - I/M and non-RFG
Minneapolis, MN	Southern CA	Northeast States - non-I/M and RFG
New York, NY	ID/MT/WY	Ohio Valley – non-I/M and non-RFG
Philadelphia, PA	UT/NM/NV	Ohio Valley – I/M and non-RFG
Phoenix, AZ	West TX	Ohio Valley – I/M and RFG
Spokane, WA	ND/SD/NB/IA/KS/Western MO	Northern MI/WI
St. Louis, MO	AR/MS/AL/SC/Northern LA	

Modeling for these areas was done on a seasonal basis. Information on fuel properties for 1990 and 1996 was obtained from surveys conducted by the National Institute for Petroleum and Energy Research (NIPER) and the American Automobile Manufacturers Association (AAMA). Fuel parameters for 2007 and 2020 were projected from 1996 baseline values using information from a February 26, 1999 report from Mathpro to the American Petroleum Institute.[154] Data from the EPA Emission Trends Database and other agency sources were used to develop appropriate local modeling parameters for I/M programs, Stage II refueling controls, fuel RVP, average ambient temperature, and other inputs.

Exhaust Emissions

Analysis of speciation data from 1990 technology light-duty gasoline vehicles done for the EPA Complex Model for Reformulated Gasoline showed that the fraction of toxic emissions relative to TOG differs among the eight technology groups within the Complex Model as well as between normal emitters and high emitters.[155] This difference is especially significant for 1,3-butadiene; its toxic/TOG fraction is about three times larger for high emitters than for normal emitters. If this difference is not taken into account, the impact of I/M programs and fleet turnover to vehicles with lower deterioration rates will be underestimated. Thus, the input format for exhaust toxic adjustment factors in MOBTOX5b was structured to allow input of high and normal emitter toxic emission rates for a given "target" fuel. The target fuel is simply the fuel of concern in the modeling analysis. These toxic emission rates were then weighted to come up with a composite toxic emission factor based on a distribution of normal and high emitters. This

[154] Costs for Meeting 40 ppm Sulfur Content Standard for Gasoline in PADDs 1-3, via MOBILE and CD TECH Desulfurization Processes. A Study performed for the American Petroleum Institute by Mathpro, Inc., February 26, 1999. EPA Air Docket A-97-10, Document Number IV-G-122.

[155] EPA. 1994. Regulatory Impact Analysis for the Final Rule on Reformulated and Conventional Gasoline, February, 1994.

distribution is not supplied directly by the MOBILE model. Instead, this distribution was determined from the fleet average TOG emission rate on baseline fuel as determined by MOBILE and average normal and high TOG emission rates on baseline fuel derived from the Complex Model. Essentially, "toxic-TOG curves" were developed that plot the target fuel toxic emission rate against the base fuel TOG emission rate.

To construct these curves, the distribution of normal and high emitters was determined in the following manner for each model year. A TOG gram per mile emission rate for normal emitters (TOG-N) and a TOG emission rate for high emitters (TOG-H) on baseline fuel were input into MOBTOX5b. TOG-N from newer technology light-duty gasoline vehicles and trucks were obtained from an unconsolidated version of the Complex Model, which provides output for normal emitters in each of eight technology groups. The Complex Model provides estimates for mass of exhaust VOC, which is TOG minus the mass of methane and ethane. TOG was estimated by applying a conversion factor which accounts for the mass of these compounds. The conversion factor was derived by analysis of weight percent emissions of methane and ethane from available speciation data. Based on the distribution of technology groups in a given model year, the individual TOG estimates were weighted appropriately to obtain a composite estimate for all normal emitters. Since the unconsolidated model's TOG-N emission rates are applicable only to Tier 0 light duty vehicles, they had to be adjusted for Tier 1 and later vehicles. This adjustment was performed by multiplying the unconsolidated model results by the ratio of the emission standard for these later vehicles to the Tier 0 emission standard. TOG-H was also obtained from the unconsolidated version of the Complex Model. TOG-H was assumed to be the same for all Tier 0 and later high emitting vehicles.

For benzene, 1,3-butadiene, formaldehyde, and acetaldehyde, milligram per mile toxic emission rates for normal and high emitters running on a given fuel formulation were also entered into MOBTOX5b using output from the unconsolidated version of the Complex Model. An example of the data file format is provided in Table IV.B-2.

Table IV.B-2
Example of Data File Format for Toxic Adjustment Factors

IV	*MYA*	*MYB*	*TOG-N*	*TOG-H*	*BZ-N*	*BZ-H*	*AC-N*	*AC-H*	*FR-N*	*FR-H*	*BD-N*	*BD-H*
1	1965	1974	0.000	10.00	0.00	276.93	0.00	109.72	0.00	224.28	0.00	93.15
1	1975	1980	0.000	10.00	0.00	263.61	0.00	108.70	0.00	173.41	0.00	44.57
1	1981	1987	0.640	4.03	28.63	113.23	5.07	32.89	7.16	44.59	2.14	25.84
1	1988	1999	0.570	4.03	17.49	116.45	4.02	28.65	5.67	36.68	2.04	30.82

Notes: IV = vehicle class, MYA = initial model year, MYB = final model year, TOG-N = TOG for normal emitters running on baseline fuel in g/mi, TOG-H = TOG for high emitters on baseline fuel in g/mi, BZ = benzene in mg/mi for vehicles running on fuel A, AC = acetaldehyde in mg/mi on fuel A, FR = formaldehyde in mg/mi on fuel A, BD = 1,3-butadiene in mg/mi on fuel A.

Using the information in the data file, an overall FTP (Federal test procedure) toxic

emission rate for each vehicle class in a given model year is calculated. This overall rate takes into account the distribution of normal and high emitters by calculating the slope and intercept of a straight line (the "toxic-TOG" curve), where the FTP toxic emission rates for a vehicle class in a given model year are a linear function of the baseline fuel TOG emission rate:

$$TOX_{Flt,\ Fuel\ A,\ FTP} = A + B*TOG_{Baseline\ \ fuel,\ FTP} \quad (1)$$

A and B are determined as follows:

$$A = (TOG\text{-}H*TOX\text{-}N - TOG\text{-}N*TOX\text{-}H)/(TOG\text{-}H - TOG\text{-}N) \quad (2)$$

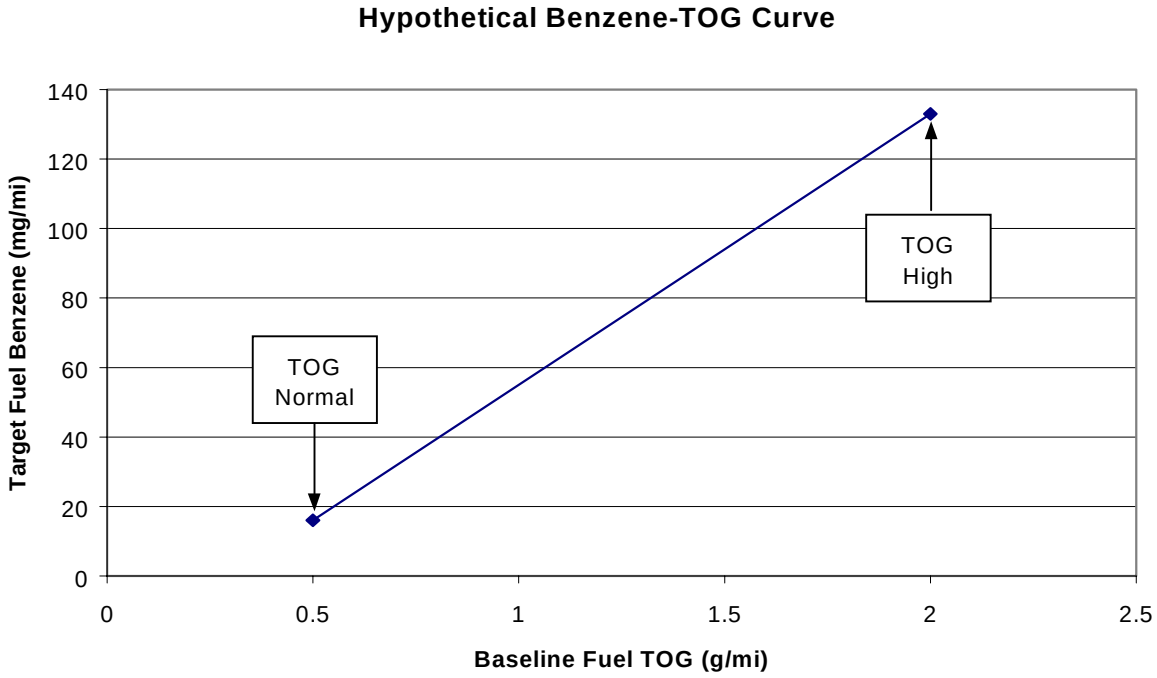
$$B = (TOX\text{-}H - TOX\text{-}N)/(TOG\text{-}H - TOG\text{-}N) \quad (3)$$

where:

TOX-N = toxic emission rate for normal emitters derived from the Complex Model
TOX-H = toxic emission rate for high emitters derived from the Complex Model
TOG-N = total organic gas emission rate for normal emitters derived from the Complex Model
TOG-H = total organic gas emission rate for high emitters derived from the Complex Model

These relationships can be thought of graphically, as illustrated in Figure IV.B-1, below.

Figure IV.B-1
Example Plot of Target Fuel Benzene Versus
Baseline Fuel TOG under FTP Conditions



An issue related to the above methodology is whether the linear assumption is valid for baseline TOG values above the high emitter point and below the normal emitter point. This is particularly relevant in cases where A and B values are determined from Tier 0 vehicles (e.g., the Complex model), but the results are applied to Tier 1 and LEV-category vehicles. For the simple example presented above, negative benzene emissions are estimated for the target fuel when the baseline fleet-average TOG emission rate falls below 0.295 g/mi. Thus, for fleet-average emission rates below (and above) the normal (and high) emitter values, a different methodology was needed. In those cases, it was assumed that the toxic emission rate was the same on a fractional basis (for VOC emission rates below the Tier 0 normal emitter rate, for example, the toxic fraction stays constant at the toxic fraction for Tier 0 normal emitters). In the example above, the benzene emission rate for a baseline TOG value of 0.1 g/mi would be calculated as follows:

$$BZ_{(TOG=0.1\ g/mi)} = 0.1\ g/mi * (16\ mg/mi\ BZ / 0.5\ g/mi\ TOG) = 3.2\ mg/mi$$

This has the effect of forcing the toxic-TOG curve from the normal-emitter point back through the origin and thus avoids negative toxic emission rate estimates for Tier 1 and LEV-category vehicles. The same approach is used in cases where the fleet-average baseline TOG emission rate is above the high emitter point.

For non-light-duty vehicle classes and older technology light-duty vehicles, such as non-

catalyst and oxidation catalyst vehicles, adequate toxic emissions data were not available to distinguish between emission rates of normal and high emitters. In such cases, the toxic fraction was assumed to be constant regardless of the VOC emission level.

Next, aggressive driving corrections were applied to the FTP toxic emission rates for light-duty vehicles. These corrections were provided in an external data file and were multiplicative in form. Several recent studies suggest that toxic fractions of TOG differ between FTP and aggressive driving conditions.[156] Thus, another adjustment to the toxic emission rates was applied to take into account this difference in toxic fractions. This adjustment took the form of the ratio of the toxic mass fraction over the unified cycle (FTP and off-cycle) to the toxic mass fraction over the FTP. The adjustment was obtained from an analysis of unpublished CARB data as described in EPA (1999d). The toxic emission rate under the unified cycle (FTP and off-cycle) was calculated in the model as follows:

$$TOX_{UC} = TOX_{FTP} * ADJ_{Aggressive\ Driving} * ADJ_{TOX\ UC/FTP} \quad (4)$$

where

TOX_{UC} = Unified Cycle toxic emission rate
TOX_{FTP} = FTP toxic emission rate
$ADJ_{Aggressive\ Driving}$ = Adjustment to TOG emissions for aggressive driving
$ADJ_{TOX\ UC/FTP}$ = Adjustment for difference in toxic mass fraction over the UC versus FTP

MOBTOX5b then applies temperature, speed, humidity and load corrections.

Evaporative, Refueling, Running Loss, and Resting Loss Emissions

MOBTOX5b estimated evaporative, refueling, running loss, and resting loss toxic emissions for benzene.[157] Benzene fractions of total hydrocarbons were entered in an external data file. Separate fractions were entered for hot soak, diurnal, refueling, running loss, and resting loss. Toxic fractions for evaporative, refueling and running loss emissions of benzene from gasoline vehicles were obtained from the Complex Model (EPA 1994). The Complex Model does not estimate resting loss emissions. EPA assumed that the benzene fractions of diurnal and resting loss emissions were the same.

Calculating Gaseous Toxic Emissions Under the Proposed 2007 Heavy-Duty Engine Standards

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[156] These studies include: Auto/Oil Air Quality Improvement Research Program. "Technical Bulletin No. 19: Dynomometer Study of Off-Cycle Exhaust Emissions"; April, 1996; Black, F.; Tejada, S.; Gurevich, M. "Alternative Fuel Motor Vehicle Tailpipe and Evaporative Emissions Composition and Ozone Potential", J. Air & Waste Manage. Assoc. 1998, 48, 578-591; and CARB, 1998, Unpublished data.

[157] 1,3-Butadiene, formaldehyde, and acetaldehyde are not found in fuel and hence are not found in nonexhaust emissions. Because their nonexhaust emissions are zero, they were not included in the portions of MOBTOX5b used to estimate nonexhaust emissions.

We expect the recently proposed 2007 heavy-duty engine standards to reduce gaseous toxics emissions. To estimate the effect of the proposed 2007 and later model year heavy-duty engine standards on toxics inventories, we started with the toxics inventories estimated in the 1999 Study assuming all heavy-duty engine programs up until the 2004 model year standards are in effect. Using these "baseline" inventory estimates and the nationwide vehicle miles traveled (VMT) estimates from the 1999 Study, we then estimated the "baseline" gram per mile emissions for the five toxics (on a nationwide, average basis). Next, we estimated the percent reduction in hydrocarbon emissions expected from the proposed 2007 heavy-duty engine standards for each of the projection years (as described in more detail in the discussion of VOC inventories below). To obtain the estimated "control" emission factors, we applied the same percent reduction to the "baseline" gram per mile toxic emission estimates. Finally, we then multiplied the gram per mile estimates by updated nationwide vehicle mile traveled (VMT) estimates to obtain the heavy-duty gasoline and diesel toxic inventories used in this analysis.[158] (Because benzene and MTBE have an exhaust and an evaporative component, we applied the percent reduction based on total (exhaust and evaporative) NMHC benefits for these compounds. For formaldehyde, acetaldehyde, and 1,3-butadiene, which do not have an evaporative component, we applied the percent reduction based on exhaust NMHC only.)

b. Projected Emissions Inventories of Selected Gaseous Toxics

Tables IV.B-3 and IV.B-4 present the on-highway emission inventories for the five gaseous MSATs addressed in 1999 EPA Motor Vehicle Air Toxics Study. The inventory estimates reflect all of the on-highway control programs currently promulgated including the Tier 2 program, plus the recently proposed 2007 heavy-duty engine standards.

[158] "VMT Estimates for the 2007 Heavy-Duty Final Rule Analyses," EPA memorandum from Penny Carey, ASD, May 9, 2000. EPA Air Docket A-2000-12, Document Number IV-B-03.

Table IV.B-3
Annual Emissions Inventory for Selected Toxics for the Total U.S.
On-Highway Vehicles Only*
(thousand short tons per year)

Compound	1990 Emissions	1996 Emissions	2007 Emissions	2020 Emissions
1,3-Butadiene	36	24	12	10
Acetaldehyde	41	31	17	13
Benzene	257	171	89	68
Formaldehyde	139	93	43	34
MTBE	55	67	26	18

* - Includes the impact of our current on-highway control programs and the proposed 2007 and later model year heavy-duty engine standards.

Table IV.B-4
Estimated Percent Reduction for Selected Toxics for the Total U.S.
On-Highway Vehicles Only*

Compound	Cumulative Percent Reduction from 1990		
	1996	2007	2020
1,3-Butadiene	35%	67%	72%
Acetaldehyde	23%	58%	67%
Benzene	33%	65%	73%
Formaldehyde	33%	69%	76%
MTBE	-22%	52%	67%

* - Includes the impact of our current on-highway control programs and the proposed 2007 and later model year heavy-duty engine standards.

3. VOC Emissions Inventory

With the exception of the five gaseous MSATs examined in the 1999 EPA Motor Vehicle Air Toxics Study, we do not have detailed emissions data for the other gaseous MSATs (acrolein, POM, styrene, xylene, toluene, ethylbenzene, naphthalene, and n-hexane). In this section, we present the VOC emissions trend as a surrogate to understand the trend in emissions of the other gaseous MSATs in order to estimate projected inventory impacts from our current and proposed

mobile source emission control programs. First, we describe how we developed our VOC inventory estimates, and then we present the results from these analyses.

a. VOC Inventory Methodology

As described in the methodology overview section, other gaseous air toxics were not evaluated explicitly in our 1999 Study. However, since all of these compounds are VOCs, we expect their emissions trends to follow the VOC emissions trend. For 1996 and later years, we based our VOC inventories on modeling that reflects adoption of all current mobile source program plus the proposed 2007 heavy-duty engine standards. We have also integrated our most recent information on emissions factors with county-specific modeling inputs. (One recent update with regard to emission factors is that we assume the technologies used by diesel engine manufacturers to meet the proposed 2007 standards will result in only a small compliance margin, instead of the larger margin we had assumed in the modeling used for the toxics proposal.) A detailed description of the modeling used as the basis for this rule can be found in the docket for this rulemaking.[159159,160,161,162162] We do not include crankcase VOC emissions in our estimates of VOC emissions since these emissions are not a portion of the exhaust from the engine, and toxics speciation data are based on tailpipe exhaust.

In the modeling that was used as the basis for this rulemaking, we did not recalculate the 1990 VOC inventory. We could not use the 1990 VOC estimates from EPA's Trends Report, since such estimates would not be comparable to the 1996 and later estimates contained in this analysis, because EPA's 1990 Trends estimates have not been updated to reflect the data and analyses that were used in this analysis (and will be used in MOBILE6). Therefore, for 1990 we modified the modeling methods applied in the 1999 Study to produce direct estimates of VOC emissions that account for the effects of fuel and vehicle technology changes as well as the information that will be used in EPA's MOBILE6 emissions model.[163]

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[159] EH Pechan & Associates, Inc, "Procedures for Developing Base Year and Future Year Mass and Modeling Inventories for the Heavy-Duty Diesel (HDD) Rulemaking," Prepared for U.S. Environmental Protection Agency, September 29, 2000. EPA Air Docket A-2000-12, included in Item IV-A-01.

[160] EH Pechan & Associates, Inc, "Data Summaries of Base Year and Future Year Mass and Modeling Inventories for the Heavy-Duty Diesel (HDD) Rulemaking–Detailed Report," Prepared for U.S. Environmental Protection Agency, September 29, 2000. EPA Air Docket A-2000-12, included in Item IV-A-01.

[161] Memorandum from Michael Samulski and John Koupal to Docket A-99-06, "Heavy-Duty Vehicle Emission Factors and Adjustment Factors for the Final 2007 Heavy-Duty Rule Inventory Analysis," May 26, 2000. EPA Air Docket A-2000-12, Item IV-B-01.

[162] Memorandum from Michael Samulski to Docket A-99-06, "Revisions to Heavy-Duty Vehicle Emission Factors and Adjustment Factors for the Final 2007 Heavy-Duty Rule Inventory Analysis," November 2, 2000. EPA Air Docket A-2000-12, Item IV-B-02.

[163] The analysis methodology is described in a memorandum from Meredith Weatherby, Eastern Research Group, to Rich Cook, EPA, entitled "Estimating of 1990 VOC and TOG Emissions" in EPA Air Docket A-2000-12, Document Number IV-D-04.

b. Projected VOC Emissions Inventory

The results of this analysis, presented in Table IV.B-5, show that on-highway VOC inventories are projected to decrease by 72 percent between 1990 and 2020 with the currently promulgated and proposed on-highway control programs. We assume that other gaseous toxics will decrease by approximately 72 percent as well. Most of the emission decrease is expected to occur before 2007.

Table IV.B-5
Annual VOC Emissions Summary for the Total U.S.
On-Highway Vehicles Only*

	1990	1996	2007	2020
Inventory (1,000 short tons/year)	7,585	4,933	3,028	2,153
Cumulative Annual Reductions from 1990 (1,000 short tons)	--.	2,652	4,557	5,433
Cumulative Percent Reductions from 1990	--	35%	60%	72%

* - Includes the impact of our current on-highway control programs and the proposed 2007 and later model year heavy-duty engine standards.

4. Diesel PM Inventory

This section describes how we derived diesel PM estimates for this rule, and then presents those estimates. We do not have inventory data on all of the organic gas components of diesel exhaust, and so we are using diesel PM as the surrogate for the particulate matter and organic gas components of diesel exhaust. Where we have data regarding specific constituents in the diesel exhaust organic gas phase we presented those above.

a. Diesel PM Inventory Methodology

As described in the methodology overview section, our diesel PM emissions estimates are based on several sources. For 1990, we used the diesel PM emissions estimates from EPA's Emissions Inventory Trends Report.[164] These estimates account for county-specific inputs in a more reliable way than our 1999 Study.[165]

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[164] EPA. 2000. National Air Pollution Emission Trends, 1900-1998 (March 2000). Office of Air Quality Planning and Standards, Research Triangle Park, NC. Report No. 454/R-00-002.

[165] The 1999 Study's approach is the more appropriate one when estimating emissions, ambient concentrations, and exposures for urban areas, which were the focus of the study.

For 1996 and later years, the diesel PM inventories have been updated for this final rule. (As referenced in the previous section on VOC inventories, a detailed description of the modeling used as the basis for this rule can be found in the docket for this rulemaking.) The light-duty diesel PM emissions are based on the PART5 model, which is similar in structure and function to the MOBILE series of models. It calculates exhaust and non-exhaust (e.g., road dust) particulate emissions for each vehicle class included in the MOBILE models. A particle size cut-off of 10 µm was specified in the model inputs since essentially all exhaust PM from diesel engines is smaller than 10 µm. The light-duty PM inventory was developed from county-specific modeling inputs. We believe this updated inventory is a more appropriate source for national light-duty diesel PM emission estimates than the 1999 Study because it better accounts for county-specific conditions.

The heavy-duty engine diesel PM emission inventories for 1996 and later years have been updated for this final rule. These estimates incorporate recent findings on heavy-duty diesel engine PM emissions that were not reflected in the 1999 Study and are developed from county-specific modeling inputs. (As referenced in the previous section on VOC inventories, a detailed description of the modeling used as the basis for this rule can be found in the docket for this rulemaking.) We did not include crankcase PM emissions in our estimates because these emissions are not a portion of the exhaust emissions, and toxics speciation data are based on tailpipe exhaust.

In the modeling used as the basis for this rulemaking, we did not recalculate the 1990 VOC inventory. Therefore, we chose to use the heavy-duty diesel PM inventory estimates for 1990 from EPA's Trends Report.[166] We believe this approach is reasonable, since we have not substantially changed our estimates of emissions from 1990 and earlier engines since the Trends Report estimates were developed. We also used data from the EPA's Trends Report for light-duty diesel estimates for 1990.

b. Projected Diesel PM Emissions Inventory

Our diesel PM inventory estimates are presented in Table IV.B-6. Diesel PM emissions are expected to decline by 63 percent in 2007, as compared with 1990 levels. These emissions are expected to decline even more by 2020, by 94 percent as compared with 1990 levels.

[166] EPA. 2000. National Air Pollution Emission Trends, 1900-1998 (March 2000). Office of Air Quality Planning and Standards, Research Triangle Park, NC. Report No. 454/R-00-002.

Table IV.B-6
Annual Diesel PM Emissions Summary for the Total U.S.
On-Highway Vehicles Only*

	1990	1996	2007	2020
Inventory (1,000 short tons/year)	235	182	85	15
Cumulative Annual Reductions from 1990 (1,000 short tons)	--	53	150	220
Cumulative Percent Reductions from 1990	--.	23%	64%	94%

* - Includes the impact of our current on-highway control programs and the proposed 2007 and later model year heavy-duty engine standards.

Chapter 5: Mobile Source Air Toxic Ambient Concentrations and Exposures

The purpose of this chapter is to review what we know about ambient concentrations and exposures associated with emissions of mobile source air toxics. First, we will review monitoring and modeled data on ambient concentrations of five of the 21 mobile source air toxics. These compounds are benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and diesel PM. We will then review results of an on-highway vehicle inhalation exposure assessment prepared by EPA. The exposure estimates are compared to estimates of the on-highway vehicle contribution to modeled ambient concentrations. As discussed in Chapter 4, we have the most reliable inventory data for the five compounds listed above, and hence the most accurate modeled mobile source estimates of ambient concentrations and exposure. As exposure estimates from the NATA national scale assessment are not yet available for comparison, the national scale assessment modeled ambient concentration estimates are used to evaluate the reasonableness of the exposure estimates. We will also discuss what we know about inhalation exposures in various micro-environments. Diesel PM is used as the dosimeter to assess exposure to whole diesel exhaust (which includes diesel PM and diesel exhaust organic gases), which the Agency has classified as a likely human carcinogen.

Because of uncertainties associated with assessing ambient concentrations and exposures, particularly for micro-environments and mobile source "hotspots," we have developed a technical analysis plan to further investigate these issues. The plan is described in the preamble.

A. Survey of Data Ambient Concentrations of Mobile Source Air Toxics.

In this section, analyses of monitor data for benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and diesel PM from EPA and State and local programs are reviewed. They will then be compared to modeled ambient concentrations. This comparison provides a check on modeled concentrations that will be used to assess exposures to MSATs.

1. Ambient Monitoring

As discussed below, monitor data for air toxics are somewhat limited. However, they are still very useful for evaluating the reasonableness of modeled ambient concentrations and bounding exposure estimates. Monitor data can also be used to identify the locations where concentrations are highest. It should be noted, however, that duration of exposure as well as concentration level influence the potential for chronic health risks. Thus, if individuals spend only a short period of time at a location where high monitored values of a pollutant have been found, there may not be a big impact on overall exposure or risk.

EPA is working with State and local air agencies to develop a monitoring network that will develop estimates of ambient concentrations that are representative of regional area

concentrations throughout the U.S. A National Air Toxics Monitoring Pilot Program has been established to provide funds for State and local governments to conduct monitoring in four urban areas and six small city and rural areas. The urban areas selected for this pilot program are Providence, Rhode Island; Seattle, Washington; Tampa Bay, Florida; and Detroit, Michigan. Awards have been made or are imminent for the six small city and rural areas. Monitoring sites are being selected to provide a better understanding of the sources and magnitudes of variability associated with ambient air toxics concentrations within and between neighborhood scale areas across the U.S. and to characterize annual average concentrations for geographic areas under various environmental conditions. Sampling will take place in late 2000 and 2001.

a. EPA Monitoring Data

This section summarizes monitored air toxics concentration data from the EPA Aerometric Information Retrieval System (AIRS), Air Quality System.[167] Using data where year-round measurements were available, we calculated 1996 mean ambient concentrations nationwide for benzene, acetaldehyde, formaldehyde, and 1,3-butadiene. These data are presented in Table V.A-1. Also presented are the number of monitor sites, standard deviations and the concentrations at monitor sites in the 95th percentile. The 95th percentile concentrations for these compounds are about twice the level of the mean concentrations. Data are especially limited for aldehydes, where only 26 sites have complete data in 1996.

Because the monitored ambient concentration results have been compiled using data from a number of different sources, often using different collection and chemical analysis methods, there is a significant amount of variability in the numbers. In addition, differences in criteria used to select the sites of monitors may make it difficult to use the data to draw conclusions about implications for population exposures. For example, many monitors are placed at sites where readings are expected to be high, often to monitor specific facilities. Conversely, other monitors might be sited away from areas of highest concentration (for instance to measure concentrations in residential areas).

Air toxics trends were recently analyzed by EPA using ambient monitor data, and results were summarized in the recently released 1998 Air Quality Trends Report.[2] Between 1993 and 1998, ambient concentrations of benzene decreased by 37% (Figure V.A-1). It is likely that this decrease is largely attributable to penetration of new highway vehicles compliant with tighter VOC standards into the existing fleet, and use of reformulated gasoline. Data for 1,3-Butadiene were also analyzed, but a consistent downward trend was not observed.

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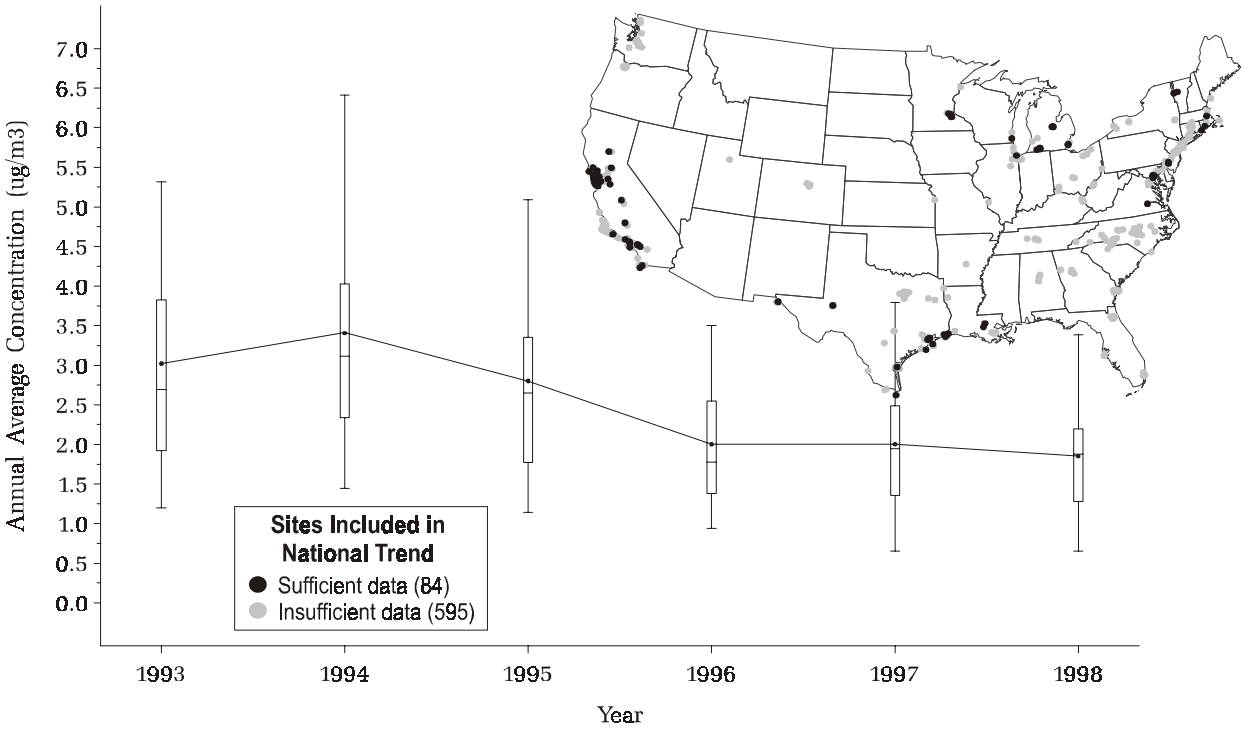
[167] The AIRS Air Quality System contains measurements of ambient concentrations of air pollutants and associated meteorological data. The data is collected by thousands of monitoring stations operated by EPA, national, state and local agencies. EPA uses this data to assess the overall status of the nation's air quality and to prepare reports to Congress as mandated by the Clean Air Act. EPA also uses the data to identify areas where improvements in air quality are needed.

Table V.A-1
Monitored 1996 ambient concentration estimates nationwide from AIRS

Compound	No. of sites	Mean Conc. ($\mu g/m^3$)	Standard deviation	95th percentile conc. ($\mu g/m^3$)
Benzene	119	1.90	0.91	3.7
1,3-Butadiene	51	0.79	2.5	1.6
Formaldehyde	26	2.90	1.2	5.4
Acetaldehyde	26	1.89	0.81	3.5

Figure V.A.-1

National trend in annual average benzene concentrations in metropolitan areas, 1993–1998 (Source:1998 Air Quality Trends Report).
(Lines on box plots show highest value, 75th percentile value, average, 25th percentile value, and lowest value)



b. State and Local Monitoring Data

Table V.A-2 presents ambient monitor concentration estimates from studies conducted in the South Coast Air Basin and the State of Minnesota, with highway and nonroad concentrations estimated using inventory apportionment.[3, 4] In other words, the relative contribution of each source sector to overall ambient concentration was assumed to be proportional to the contribution of each source sector to total direct emissions. It should be noted that there is considerable uncertainty in apportioning concentrations of aldehydes in this way, because much of the ambient concentration of aldehydes is formed secondarily from other precursors. The South Coast represents an area with a toxic emissions inventory dominated by mobile sources. According to American Association of Automobile Manufacturers' fuel survey data, Minnesota has high levels

of benzene in gasoline (average 1.73% in 1996, compared to a nationwide average of 1.1% for non-RFG areas). As a result, we might expect that ambient benzene concentrations are higher in Minnesota than in other States, but the average monitor values for benzene in Minnesota are not significantly higher than the nationwide average based on AIRS in Table V.A-1.

Table V.A-2
Monitored average ambient concentration estimates (µg/m^3), and estimated highway and nonroad contributions, in the South Coast Air Basin and in Minnesota

	South Coast Air District (1998-9)			Minnesota (1996)		
Compound	All Sources	Highway	Nonroad	All Sources	Highway	Nonroad
Benzene	3.53	2.46	0.73	1.69	0.48	0.78
1,3-Butadiene	0.79	0.53	0.21	N.A.	N.A.	N.A.
Formaldehyde	4.82	2.28	2.21	1.64	0.65	0.43
Acetaldehyde	3.17	1.52	1.57	N.A.	N.A.	N.A.

Sources: 1. South Coast Air Quality Management District. 1999. Multiple Air Toxics Exposure Study in the South Coast Air Basin – MATES-II. 2. Minnesota Pollution Control Agency. 1999. MPCA Staff Paper on Air Toxics.

c. Diesel PM Monitoring

We do not have a way of actually measuring diesel PM. However, there are two indirect methods of estimating diesel PM ambient concentrations based on monitor data. First, monitoring data on elemental carbon concentrations can be used as a surrogate to determine ambient diesel PM concentrations. Elemental carbon is a major component of diesel exhaust, contributing to approximately 60%-80% of diesel particulate mass, depending on engine technology, fuel type, duty cycle, lube oil consumption, and state of engine maintenance.[5 6 7 8] In most areas, diesel engine emissions are major contributors to elemental carbon, with other potential sources including gasoline exhaust, combustion of coal, oil, or wood, charbroiling, cigarette smoke, and road dust. Because of the large portion of elemental carbon in diesel particulate matter, and the fact that diesel exhaust is one of the major contributors to elemental carbon in most areas, ambient diesel PM concentrations can be bounded using elemental carbon measurements. The Agency's draft *Health Assessment Document for Diesel Exhaust*[9] presents one approach for calculating ambient diesel particulate matter concentrations using elemental carbon measurements. In the absence of a more sophisticated modeling analysis, using elemental carbon as a surrogate for diesel PM is a useful approach where elemental carbon concentrations are available.

The second approach for estimating ambient diesel PM concentrations based on monitor data uses the chemical mass balance (CMB) model in conjunction with ambient PM measurements to estimate ambient diesel PM concentrations. Inputs to the CMB model include particulate matter measurements made at the receptor site as well as measurements made of each of the source types suspected to affect the site. Because of the co-emission of diesel and gasoline particulate matter in time and space, chemical molecular species that provide markers for separation of these sources have been identified. Recent advances in chemical analytical techniques have facilitated the development of sophisticated molecular source profiles, including detailed speciation of organic compounds, which allow the apportionment of particulate matter to gasoline and diesel sources with increased certainty. Older studies that made use of only elemental carbon source profiles have been published and are summarized here, but are subject to more uncertainty. It should be noted that since receptor modeling is based on the application of source profiles to ambient measurements, this estimate of diesel particulate matter concentrations includes the contribution from on-highway and nonroad sources of diesel PM. In addition, this model accounts for primary emissions of diesel PM only; the contribution of secondary aerosols is not included.

Ambient diesel PM concentration estimates using these two approaches are summarized in Table V.A-3.

2. Modeled Ambient Concentrations

In this section, data on modeled ambient concentrations of air toxics are reviewed. Sources of data include the Agency's Cumulative Exposure Project and the National Air Toxics Assessment.

a. Cumulative Exposure Project

In 1998, EPA's Office of Policy, Planning, and Evaluation released results of a modeling study that estimated outdoor concentrations of hazardous air pollutants.[10] This analysis was done as part of the Cumulative Exposure Project (CEP). The study estimated 1990 annual average outdoor concentrations of 148 toxics, nationally, and by census tract. In this study, county level emissions were allocated to individual census tracts using spatial surrogates (for example, roadway miles were used a surrogate for gasoline highway motor vehicle emissions). Emissions were also allocated temporally into three-hour time blocks. The emission estimates were obtained from an inventory developed specifically for the CEP. Since the time this inventory was developed, higher quality and more recent emissions data have become available.

Table V.A-3
Ambient Diesel Particulate Matter Concentrations from Receptor Modeling and Elemental Carbon Measurements

Location	Year of Sampling	Diesel PM_{10} & $PM_{2.5}$ $\mu g/m^3$ (mean)	Diesel PM % of Total PM	Source of Data
West LA, CA	1982, annual	4.4	13%	Source-Receptor Modeling
Pasadena, CA	1982, annual	5.3	19%	
Rubidoux, CA	1982, annual	5.4	13%	
Downtown LA, CA[11]	1982, annual	11.6	36%	
Phoenix area, AZ[12]	Winter,1989-90	4-22*	†	
Phoenix, AZ[13]	1994-95, annual	0-5.3 (2.4)	0-27%	
California, 15 Air Basins[14]	1988-92, annual	0.2-3.6*	†	
Manhattan, NY[15]	3 days, Spring, 1993	13.2-46.7*	31-68%	
Welby, CO	60 days, Winter, 1996-97	0-7.3 (1.7)	0-26%	
Brighton, CO[16]	60 days, Winter, 1996-97	0-3.4 (1.2)	0-38%	
Boston, MA	1995, annual	0.7-1.7 (1.1)	3-15%	Elemental Carbon Measurement
Rochester, NY	1995, annual	0.4-0.8 (0.5)	2-9%	
Quabbin, MA	1995, annual	0.2-0.6 (0.4)	1-6%	
Reading, MA	1995, annual	0.4-1.3 (0.6)	2-7%	
Brockport, NY[17]	1995, annual	0.2-0.5 (0.3)	1-5%	
Washington, DC[18]	1992-1995, annual	1.3-1.8 (1.6)	6-10%	
South Coast Air Basin[1]	1995-1996, annual	2.4-4.5‡	†	

*PM10 † Not Available

‡ The Multiple Air Toxics Exposure Study in the South Coast Air Basin reported values for maximum monthly elemental carbon concentrations across a ten-site network.

Concentrations were estimated from these inventory data using a dispersion model known as ASPEN (Assessment System for Population Exposure Nationwide). The modeling estimated concentrations attributable to major, area, and mobile sources. In order to model a large number of pollutants nationwide, ASPEN makes a number of simplifying assumptions. For instance, where specific latitude and longitude coordinates were not available, facilities were randomly located within a county. Moreover, concentration estimates at the census tract level were estimated using modeling assumptions to allocate emissions from the county level, and the model is very sensitive to the assumptions used. In addition, dispersion of emissions from non-point sources (e.g. on-highway and nonroad vehicles) was treated simplistically. For resident tracts that have radii greater than 0.3 km, non-point source ambient concentrations are estimated on the

basis of five pseudo point sources. The average concentration for the census tract is determined by spatially averaging the ambient concentrations associated with the receptors defined for the five pseudo sources which fall within the bounds of the tract. For resident tracts with radii less than 0.3 km, ambient concentrations are set equal to zero. Other limitations include: terrain impacts on dispersion were not included; no long range transport was included; and reliance on long term climate summary data. Because of these limitations, the results are most meaningfully interpreted when viewed over large geographic areas (i.e., at the national or State level). Comparison of modeled concentrations to monitored concentrations indicate that the model is more likely to underestimate monitored values than to overestimate them. However, the model appears to be relatively accurate for benzene.

Table V.A-4 presents the CEP's estimated nationwide average concentrations of benzene, 1,3-butadiene, formaldehyde, and acetaldehyde from all sources, as well as the contribution attributable to mobile sources, separated into on-highway and nonroad. The mobile source concentration estimates were allocated to on-highway and nonroad sources based on the on-highway and nonroad shares of the nationwide CEP inventory. Allocation of aldehydes was based on direct emissions. There is considerable uncertainty in apportioning concentrations of aldehydes in this way, because much of the ambient concentration of aldehydes is formed secondarily from other precursors.

Table V.A-4
Average estimated nationwide concentrations of selected air toxics in 1990 from the Cumulative Exposure Project ($\mu g/m^3$)

Compound	Conc. ($\mu g/m^3$) All sources	Mobile Contribution to Ambient Conc.	On-Highway Contribution to Ambient Conc. (% of avg. conc.)	Nonroad Contribution to Ambient Conc. (% of avg. conc.)
Benzene	2.10	1.1	0.87 (41)	0.23 (11)
1,3-Butadiene	0.15	0.11	0.08 (53)	0.03 (20)
Formaldehyde	1.50	0.76	0.50 (33)	0.26 (17)
Acetaldehyde	0.72	0.44	0.29 (40)	0.15 (21)

Source: Systems Applications International. 1998. Modeling Cumulative Outdoor Concentrations of Hazardous Air Pollutants. Report No. SYSAPP 98-96/33, Prepared for U. S. EPA, Office of Policy, Planning and Evaluation, February, 1998.

b. National Air Toxics Assessment Results for Mobile Sources

As part of its National Air Toxics Assessment (NATA) activities, EPA has conducted a national-scale air toxics assessment using the ASPEN dispersion model, in conjunction with the 1996 National Toxics Inventory, to estimate ambient concentrations of 33 air toxics identified in the IUATS, plus diesel PM. The NATA national scale assessment reported distributions of concentrations across census tracts nationally and at the county level. Since this national scale assessment, like the CEP analysis, uses ASPEN, it has similar limitations. Again, the results are most meaningfully interpreted when viewed over large geographic areas. The NATA national scale analysis also apportioned the contribution to ambient concentrations between major, area, nonroad mobile, and on-highway sources. Results are available at http://www.epa.gov/ttn/uatw/nata. Table V.A-5 presents draft mean and median nationwide gaseous toxic ambient concentrations attributable to on-highway and nonroad mobile sources using this modeling method.[168] Estimates are provided for benzene, 1,3-butadiene, formaldehyde, and acetaldehyde. Both mean and median values are reported, because high outlying values may bias the means. The estimates take into account photochemical reactivity. Mean concentrations for other mobile source toxics estimated using ASPEN can be found in Appendix 1. For pollutants which are on the list of hazardous air pollutants included in the National Scale Analysis, the on-highway contribution, nonroad contribution, and the contribution from all sources is provided. There are a number of additional compounds on the mobile source air toxics list that are not included in the National Scale Analysis. For these compounds, mobile source only contributions were estimated using ASPEN.[19] Diesel PM estimates are being revised to account for inventory changes and will be included in the release for EPA Science Advisory Board review.

EPA has performed "model to monitor" comparisons for several compounds in the NATA assessment. Results are available on the NATA website given above and summarized in Table V.A-6 below. In general, the statistics for benzene suggest good agreement between the model and the monitors. The median of the ratios is close to one, with a small standard deviation; this suggests that on average, the model estimates and monitor averages are close to each other. For almost half the sites, the range of county model estimates covers the monitor average; the remaining sites are missed on the low side and some on the high side, suggesting no clear bias. The model's estimates are within a factor of two of the monitored concentrations for about 90% of the benzene monitors in this study. Comparisons for formaldehyde and acetaldehyde do not show such good agreement. However, they compare more favorably when the maximum estimated modeled concentration is examined within 30 km of the monitoring site. This indicates that emissions sources were not precisely located or the monitors were sited to find peak concentrations.

[168] The nationwide numbers are for 48 States plus the Virgin Islands and Puerto Rico.

Table V.A-5
Draft average estimates of mobile source contributions to nationwide concentrations of selected air toxics in 1996 from the NATA national scale assessment

Compound	On-Highway Contribution to Ambient Conc. ($\mu g/m^3$)		Nonroad Contribution to Ambient Conc. ($\mu g/m^3$)	
	Mean	Median	Mean	Median
Benzene	0.55	0.45	0.24	0.16
1,3-Butadiene	0.05	0.04	0.02	0.01
Formaldehyde	0.38	0.29	0.48	0.20
Acetaldehyde	0.40	0.32	0.27	0.12

Table V.A-6
Agreement of modeled ambient concentrations from the NATA national scale assessment and monitored ambient concentrations on a point by point basis

Compound	Number of Sites	Median of Ratios	Percentage of sites within a factor of two	Percentage of sites where monitored concentrations underpredicted
Benzene	87	0.92	89	59
Formaldehyde	32	0.65	53	88
Acetaldehyde	32	0.60	59	91

c. Diesel PM Estimates

Two dispersion model studies reporting diesel PM have been conducted in Southern California. Results are summarized in Table V.A-7. Secondary formation of particulate matter accounted for 27% to 67% of the total particulate matter associated with diesel engines.[20] [21] Dispersion modeling conducted in Southern California reported that the on-highway contribution to the reported diesel PM levels ranged from 63% to 89%.

Table V.A-7
Annual average diesel particulate matter concentrations predicted from dispersion modeling

Location	Year of Sampling	Diesel $PM_{2.5}$ $\mu g/m^3$ (mean)	Diesel $PM_{2.5}$ % of Total $PM_{2.5}$
Azusa, CA	1982, annual	1.4**	5%
Pasadena, CA	1982, annual	2.0**	7%
Anaheim, CA	1982, annual	2.7**	12%
Long Beach, CA	1982, annual	3.5**	13%
Downtown LA, CA	1982, annual	3.5**	11%
Lennox, CA	1982, annual	3.8**	13%
West LA, CA[22]	1982, annual	3.8**	16%
Claremont, CA[23]	18-19 Aug 1987	2.4**	8%
Long Beach, CA	24 Sept 96	1.9	8%
Fullerton, CA	24 Sept 96	2.4	9%
Riverside, CA[24]	25 Sept 96	4.4	12%

+Value in parenthesis includes secondary diesel PM (nitrate, ammonium, sulfate and hydrocarbons) due to atmospheric reactions of primary diesel emissions of NOx, SO_2 and hydrocarbons.
**On-highway diesel vehicles only

B. Modeled Inhalation Exposures

As part of the National Air Toxics Assessment (NATA) national scale assessment, 1996 inhalation exposure estimates are being developed to assess the exposure concentrations attributable to on-highway and nonroad mobile sources. As mentioned previously, this effort uses a dispersion model, ASPEN, to model ambient concentrations of air toxics at the county level. These data will then be used as input into version 4 of the Hazardous Air Pollutant Exposure Model (HAPEM4). A report with exposure and risk characterization results using HAPEM4 will be submitted to the Agency's Science Advisory Board for review later this year, and final results should be publicly available subsequently in 2001. HAPEM4 estimates inhalation exposures to air toxics from outdoor sources. It predicts inhalation exposure through a series of calculation routines. The model makes use of census data, human activity patterns, ambient air quality levels, climate data, and microenvironmental factors (indoor/outdoor concentration relationships) to estimate an expected range of inhalation exposure concentrations for groups of individuals. The microenvironmental factors are based on empirical field data, which is better for some pollutants than for others. Although this model is not designed to estimate individual exposures, it can provide exposure distribution estimates for the general population as well as for various subpopulations of interest (e.g., children aged 0-17 years).

Table V.B-1
California annual average diesel PM exposure estimates for all mobile sources from the California Population Indoor Exposure Model

Year	California Exposure Estimates (On-Highway & Nonroad) in $\mu g/m^3$	
1990	California Annual Average	1.5
2007	Projected California Annual Average	1.3
2020	Projected California Annual Average	1.2

Source: CARB. Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant Appendix III Part A: Exposure Assessment.

Exposure estimates for diesel PM from on-highway and nonroad sources were recently modeled by CARB using the California Population Indoor Exposure Model (CPIEM). Results from this model are presented in Table V.B-1 below and described in more detail in CARB's "Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant Appendix III Part A: Exposure Assessment".[25] Other than these two efforts, the only available estimates of inhalation exposure to motor vehicle related air toxics from ambient sources were developed using the Hazardous Air Pollutant Exposure Model for Mobile Sources, version 3 (HAPEM-MS3).[26, 27, 28] This model uses CO as a tracer for air toxics exposure, rather than using modeled ambient concentrations of toxics as inputs like HAPEM4. Also, this version of the model is designed to address exposure attributable to on-highway vehicle emissions, whereas HAPEM4 addresses exposures attributable to all source categories. Methods used to develop exposure estimates using this model are discussed below.

1. Methodology for Modeling Inhalation Exposures to Benzene, Formaldehyde, Acetaldehyde, 1,3-Butadiene and Diesel PM: HAPEM-MS3

Estimates of exposure for gaseous air toxics using a previous version of the HAPEM-MS model were published in the 1993 Motor Vehicle-Related Air Toxics Study.[29] Based on peer review comments, a number of improvements were made to the model, principally by EPA's Office of Research and Development, resulting in HAPEM-MS3. These improvements include:

- 1980 census and CO monitoring data were replaced with 1990 data for base-year modeling;
- 32 additional micro-environments were added to the 5 micro-environments in the original model (20 of these micro-environments were indoors);
- Activity data from three cities were used, rather than data from one city as in the original model; and
- More detail on seasonal and regional exposures was retained.

Exposure modeling projections considering currently planned controls and using HAPEM-MS3 are presented in the next section. This section focuses on methods used to develop these estimates, limitations and uncertainties, and an evaluation of the reasonableness of the baseline estimates for 1990 and 1996, based on a comparison to other data sources.

a. Modeling Approach

Exposure modeling was done for 1990. Data from 10 urban areas were used. These areas were Atlanta, GA, Chicago, IL, Denver, CO, Houston, TX, Minneapolis, MN, New York, NY, Philadelphia, PA, Phoenix, AZ, Spokane, WA, and St. Louis, MO. As mentioned previously, HAPEM-MS3 uses CO as a tracer for toxics. Thus, these areas were selected because a large percentage of the population lived within reasonable proximity to CO monitors, and also to represent good geographic coverage of the U.S. Since most ambient CO comes from cars and light trucks, we believe CO exposure is a reasonable surrogate for exposure to other motor vehicle emissions, including toxics emissions. The HAPEM model links human activity patterns with ambient CO concentration to arrive at average exposure estimates for 22 different demographic groups (e.g., outdoor workers, children 0 to 17, working men 18 to 44, women 65+) and for the total population. The model simulates the movement of individuals between home and work and through a number of different micro-environments. The CO concentration in each micro-environment is determined by multiplying ambient concentration by a micro-environmental factor derived from regression analysis of ambient and personal monitor data. Each micro-environmental factor has a multiplicative term, which represents ambient exposure, and an additive term, which represents exposure to emissions originating within micro-environments. These factors were derived by IT Corporation based on the results of regression analyses using paired ambient and personal exposure monitor measurements from CO studies in Denver and Washington.[30, 31] In our modeling, we set the additive term to zero, to eliminate non-ambient sources of CO, such as gas stoves.[169] The multiplicative term has a component that represents penetration from the ambient air into the micro-environment, and a factor that represents the proximity of the micro-environment to monitors. Thus, even though a compound may have a penetration rate of close to one, the microenvironmental factor could be significantly less than one if the microenvironment is typically found a significant distance from where CO monitors are located. HAPEM-MS3 micro-environmental factors are given in Appendix 2.

With the 1990 CO exposure estimates generated by the HAPEM-MS3 model for each urban area, EPA determined the fraction of exposure attributable to on-highway vehicle emissions. This calculation was accomplished by scaling the exposure estimates (which reflect exposure to total ambient CO) by the fraction of the 1990 CO emissions inventory from on-highway motor vehicles, determined from the EPA Emission Trends database.[32, 33] This scaling

[169]It should be noted that in some microenvironments, there are large additive terms attributable to motor vehicles, such as at service stations and in garages. HAPEM-MS3 does not account for these non-ambient sources of exposure to motor vehicle emissions.

takes into account the contribution of background CO to the inventory. Nationwide urban CO exposure attributable to on-highway vehicle emissions was estimated by first calculating a population-weighted average CO exposure for the ten modeled areas. This number was adjusted by applying a ratio of population-weighted annual average CO ambient concentrations for urban areas in the entire country versus average ambient CO concentration for the modeled areas. To estimate rural exposure, the urban estimate was scaled downward using rough estimates of urban versus rural exposure from the 1993 *Motor Vehicle-Related Air Toxics Study* (EPA 1993a). The scaling factor was 0.56, based on an average CO concentration in rural areas of 470 $\mu g/m^3$ and a concentration in urban areas of 842 $\mu g/m^3$.

Modeled CO exposure attributable to on-highway vehicle emissions for 1990 was divided by 1990 CO grams per mile emission estimates to create a conversion factor. The conversion factor was applied to modeled toxic emission estimates (in grams per mile) to determine exposure to on-highway vehicle toxic emissions, as shown in Equation 6:

$$TOX_{Exposure(\mu g/m3)} = [CO_{Exposure(\mu g/m3)}/CO_{EF(g/mi)}]_{1990} \times TOX_{EF(g/mi)} \qquad (6)$$

where:

$TOX_{Exposure(\mu g/m3)}$ = exposure to on-highway vehicle toxic emissions
$CO_{Exposure(\mu g/m3)}$ = exposure to on-highway vehicle CO emissions
$CO_{EF(g/mi)}$ = CO emission factor
$TOX_{EF(g/mi)}$ = toxic emission factor

The exposure estimates for calendar years 1996, 2007, and 2020 were adjusted for VMT growth relative to 1990. We also included in the model various assumptions regarding transformation of the toxics. For example, benzene was treated as inert, but 1,3-butadiene exposure was adjusted to account for its atmospheric transformation into other species. The multiplicative factors used to adjust for this transformation were 0.44 for summer, 0.70 for spring and fall, and 0.96 for winter.[34] These factors account for the difference in reactivity between relatively inert CO, which is being used as the tracer for toxics exposure, and the more reactive 1,3-butadiene. In contrast, estimated exposures to formaldehyde and acetaldehyde were based on direct emissions. For these pollutants, removal of direct emissions in the afternoon was assumed to be offset by secondary formation. Limitations and problems with this assumption are discussed in the following section. Annual average exposure estimates to the gaseous air toxics for the entire population in 1990 and 1996 are presented in Tables V.B-2 and V.B-3. Annual average inhalation exposure to diesel PM was estimated to be 0.82 $\mu g/m^3$ in 1990 and 0.73 $\mu g/m^3$ in 1996. Estimates were also developed for outdoor workers, and children 0 - 17 years of age. Exposure among outdoor workers was higher than for the entire population, and among children it was slightly lower.

As discussed in Chapter 4, gaseous toxic and diesel PM emission estimates for heavy duty gasoline and diesel vehicles in 1996, 2007, and 2020 were revised for the 2007 heavy-duty

engine proposed rule. Thus, exposure estimates for these years were adjusted upward to account for the discrepancy.

b. Limitations and Uncertainties

Use of the HAPEM-MS3 model to estimate exposure to toxic emissions from motor vehicles introduces several notable sources of uncertainty. First, the model may underestimate CO exposures of the maximally exposed population. Although a validation study of HAPEM-MS3 has not been done, such an analysis has been done for the pNEM/CO Model (NAAQS Exposure Model for CO), which uses an approach similar to that used in HAPEM-MS3 as well as much of the same data.[35] Generally speaking, pNEM/CO's estimates of CO exposures for the population in the 5th percentile were overestimated by about 33%, and those in the 98th percentile were underestimated by about 30%. This result suggests that pNEM/CO underestimates CO exposures of the maximally exposed population. These results would likely also hold for HAPEM-MS3 estimates of toxics exposure as well, and suggest that the model is probably best suited for estimating average exposures.

Second, the data used to derive micro-environmental factors are limited. As described earlier, the data are obtained from only two cities. Thus, the regression equations used to derive the micro-environmental factors are subject to substantial error. Moreover, activity data are very limited for some demographic groups. For instance, there was very little activity data for African Americans or Hispanic Americans in the database.

Third, because we set the additive terms of the micro-environmental factors to zero, the HAPEM-MS3 results do not account for exposures to emissions originating within micro-environments. For instance, the model does not account for indoor exposure to evaporative benzene emissions from vehicles parked in attached garages. The potential impact of these additional sources of emissions within micro-environments is discussed in Section V.C.

Fourth, the modeling done in this assessment assumes that the on-highway fleet emissions ratio of CO to diesel PM can be used as an adjustment factor to convert estimated CO personal exposures to diesel PM exposures. However, most CO emitted from on-highway vehicles is emitted by gasoline vehicles, while most on-highway diesel PM is emitted from heavy-duty diesel engines. Even though gasoline- and diesel-fueled on-highway vehicles travel the same roadways, temporal and spatial patterns for diesel vehicle operation are different than gasoline-fueled vehicles. This could result in underestimates of diesel PM exposure, for instance, in areas where the proportion of heavy-duty diesel vehicle traffic is significantly greater than average relative to light-duty gasoline vehicle traffic. Conversely, overestimates of commuting exposures could occur where there is very little heavy-duty diesel vehicle traffic.

Similarly, the model also does not take into account the fact that spatial and temporal allocation of benzene evaporative emissions are different than CO emissions. However, in modern technology vehicles, with evaporative emission controls, benzene emissions are

dominated by the exhaust component. The modeling approach also assumes that emissions of toxics vary linearly with CO as a function of ambient temperature. Also, although we know that emissions of CO as well as the toxic compounds modeled are all higher at lower temperatures, we do not know if the relationship is linear. Moreover, the assumption that exposure increases proportionally to VMT does not account for urban spreading or building of new roadways within an urban area.

Finally, as mentioned previously, we assumed that estimated exposure to formaldehyde and acetaldehyde was based on direct emissions. As will be discussed in Section V.C.4 below, we believe this assumption results in an underestimate of acetaldehyde exposure by about a factor of three. Thus, HAPEM-MS3 based estimates of acetaldehyde exposure must be adjusted to account for this underestimate.

c. Improvements to HAPEM

As mentioned previously, a new version of HAPEM (HAPEM4) has been developed for use in the National Air Toxics Assessment. This new version has a number of major modifications and improvements including the capability to model inhalation exposure for all outdoor sources. Thus a separate version of HAPEM for motor vehicles is no longer needed. The model has been revised to accept monitored or modeled toxics concentrations directly as input, rather than using CO as a surrogate for toxics. The model also now incorporates a new time-activity database derived from the CHAD (Consolidated Human Activity Database), developed by EPA's Office of Research and Development. It should be noted that HAPEM4 still has limitations. It estimates only inhalation exposures to air toxics from outdoor sources, and cannot estimate maximum exposures, only population exposures. Moreover, the microenvironmental factors in the model are better for some pollutants than for others.

2. Comparison of Exposure Modeling Results to Modeled Ambient Concentrations

In this section, we compare HAPEM-MS3 exposure modeling results to modeled ambient concentrations from the Cumulative Exposure Project and NATA national scale assessment.[170] It should be noted that average ambient concentrations do not represent average inhalation exposures because they do not take into account human activity patterns, intrusion of ambient air toxics into specific micro-environments, or emissions of air toxics from micro-environmental sources. Nonetheless, we expect them to be within the same order of magnitude, and in the absence of other exposure estimates, these data represent the best surrogate source of information with which to evaluate the reasonableness of HAPEM-MS3 results.

A number of other limitations and uncertainties make it difficult to directly compare

[170] Comparisons to monitor data were not made, due to the difficulty in estimating mobile source contributions with accuracy.

modeled ambient concentration estimates to exposure estimates. First, uncertainties result from the surrogates (e.g., roadway miles and population density) used to allocate emissions from the county to census tract level for use in dispersion modeling. Also, the ASPEN dispersion model does not include a terrain component, and relies on long-term climate summary data.

We first compared HAPEM-MS3 average 1990 nationwide exposure estimates for on-highway vehicles for the entire U.S. population to 1990 CEP ambient concentration estimates for on-highway vehicles. Results are presented in Table V.B-2. These results indicate close correspondence except for acetaldehyde, where CEP results are about 70% higher.

We also compared HAPEM-MS3's 1996 average nationwide estimates of gaseous toxic exposure from on-highway vehicle emissions to estimates of the on-highway vehicle contribution to ambient concentrations from the draft NATA national scale analysis. Results are presented in Table V.B-3. Overall, average modeled ambient concentrations from the national scale analysis are within the same order of magnitude of the average HAPEM-MS3 exposure results.

Agreement is fairly close for benzene,1,3-butadiene, and formaldehyde, while HAPEM-MS3 estimates for acetaldehyde are low compared to ambient concentration estimates from the NATA national scale analysis. HAPEM-MS3 exposure estimates for formaldehyde and acetaldehyde do not account for photochemistry, and removal of primary emissions are assumed to be offset by secondary formation. ASPEN, on the other hand, accounts for aldehyde photochemistry. It assumes that about 68% of formaldehyde is primary but only about 20% of acetaldehyde is assumed to be primary. Since most ambient acetaldehyde is secondary, the HAPEM-MS3 exposure estimate based on direct emissions will underestimate acetaldehyde exposure. Thus, we have adjusted HAPEM-MS3-based estimates of acetaldehyde exposure by a factor of 3 to be consistent with NATA modeled ambient concentrations. Subsequent estimates of acetaldehyde exposure will include this adjustment.

Given the limitations inherent in making comparisons, the results available at this time suggest that the HAPEM-MS3 approach provides reasonable estimates of inhalation exposure with the exception of acetaldehyde, which we are adjusting to account for the model’s inherent limitations

Table V.B-2
Comparison of 1990 average exposure attributable to on-highway vehicle emissions (HAPEM-MS3) to 1990 ambient concentration estimates attributable to on-highway vehicle emissions (CEP)

Compound	HAPEM-MS3 Based Exposure ($\mu g/m^3$)	CEP Ambient Conc. ($\mu g/m^3$)
Benzene	1.07	0.87
1,3-Butadiene	0.11	0.08
Formaldehyde	0.57	0.50
Acetaldehyde	0.17*	0.29

*Unadjusted estimate based on direct emissions – adjusted level which includes secondary formation is 0.51 $\mu g/m^3$.

Table V.B-3
Comparison of 1996 annual average exposures attributable to on-highway vehicles (HAPEM-MS3) and the on-highway vehicle portion of 1996 modeled ambient concentrations (National Scale Assessment)

Compound	**HAPEM-MS3 On-Highway Vehicle Exposure ($\mu g/m^3$)**	**NATA On-Highway Vehicle Mean Ambient Concentration ($\mu g/m^3$)**
Benzene	0.71	0.55
1,3-Butadiene	0.08	0.05
Formaldehyde	0.37	0.38
Acetaldehyde	0.13*	0.40

*Unadjusted estimate based on direct emissions – adjusted level which includes secondary formation is 0.38 $\mu g/m^3$.

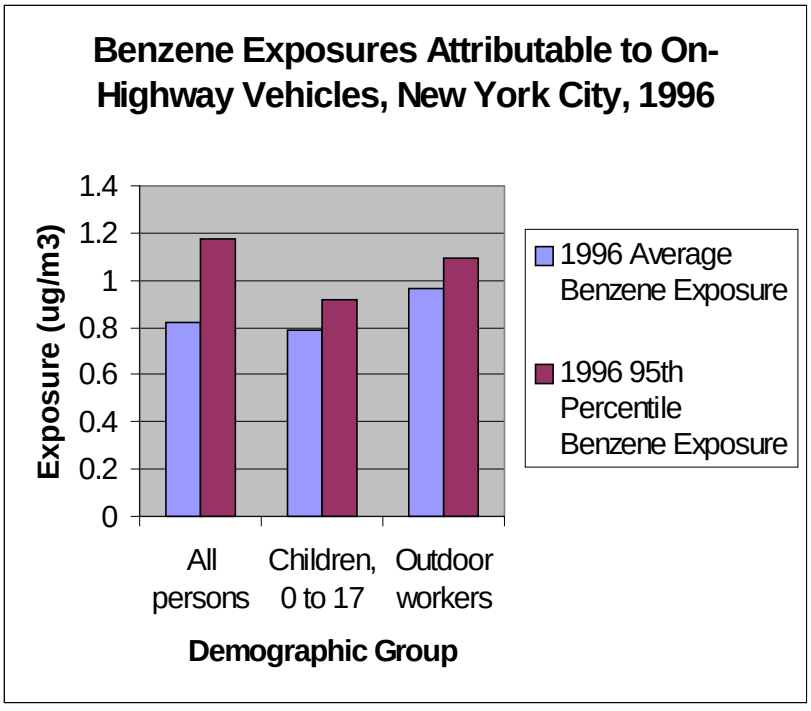
3. Variance in Exposures

a. Distribution of Exposures

HAPEM-MS3 reports average annual and seasonal CO exposures by demographic group, as well as the distribution of exposures around the mean. These distributions for CO exposure can be used to estimate distributions of toxics exposure. As mentioned previously, we believe that HAPEM-MS3 overestimates low-end exposures and underestimates high-end exposures (by about 30% for the 98th percentile). Figure V.B-1 presents the on-highway vehicle portion of both average annual and the 95th percentile annual benzene exposures for the general population in New York City. Exposures for the 95th percentile of the entire population are about 50% higher than the average. Results are presented for children 0 to 17 and outdoor workers as well. The distribution of exposures for children and outdoor workers are not as broad as the distribution for the entire population. This could be due to the smaller database for these demographic groups as well as less variability in activity patterns.

Differences in the amount of time individuals spend in various micro-environments contributes significantly to the overall distribution in annual exposures. For instance, individuals spending the greatest percentage of time in high-exposure micro-environments have annual CO exposures about 30% higher than average. Conversely, individuals spending the greatest amount of time in low-exposure micro-environments have annual CO exposures about 5% lower than average.

Figure V.B-1
Average and 95th percentile benzene exposures
(attributable to on-highway vehicles) in New York City, 1996
[Figure revised to correct error]



b. Variance among demographic groups

We have analyzed average inhalation exposures for three demographic groups -- the overall population, outdoor workers, and children 0 to 17. Since inhalation exposures to air toxics from outdoor sources are typically lower indoors than outdoors, exposures for outdoor workers are somewhat higher than the general population. Exposures for children are similar to the general population, although slightly lower since children spend a little more time indoors than most other demographic groups. Nationwide average inhalation exposures for the three demographic groups in 1996 are presented in Table V.B-4.

Table V.B-4
Highway vehicle portion of nationwide average inhalation exposures to benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and diesel PM for three demographic groups in 1996, based on HAPEM-MS3

Pollutant	Overall Population Exposure ($\mu g/m^3$)	Outdoor Worker Exposure ($\mu g/m^3$)	Children's Exposure ($\mu g/m^3$)
Benzene	0.71	0.82	0.68
1,3-Butadiene	0.08	0.09	0.08
Formaldehyde	0.37	0.46	0.37
Acetaldehyde	0.38	0.45	0.38
Diesel PM	0.76	0.88	0.73

c. Geographic variation

HAPEM-MS3 modeling results indicate that average inhalation exposures vary significantly between geographic locations. Toxics exposures are impacted by ambient temperatures, local fuel properties, age of the in-use fleet, I/M programs, traffic density, demographics, and many other factors. To illustrate, Table V.B-5 presents the on-highway portion of the1996 average annual benzene inhalation exposure estimates for the 10 areas modeled in the EPA 1999 Study, as well as the estimates for urban areas and rural areas nationwide. Among the urban areas modeled, Phoenix had the highest level of annual on-highway vehicle contribution to benzene exposure in 1996. Phoenix had a high level of CO exposure attributable to highway vehicles in 1996 (484 $\mu g/m^3$) combined with an average fuel benzene level of 1.07% in summer and 1.40% in winter (based on AAMA fuel surveys). Average benzene exposure in Phoenix is expected to drop substantially by 2007 due to the adoption of California reformulated gasoline and as a result of more stringent Federal emission standards and fleet turnover. Minneapolis also has high benzene exposure levels relative to other

modeled areas in 1996; this is due to significantly higher than average fuel benzene levels of 1.81% in summer and 1.65% in winter (based on AAMA fuel survey data).

Not surprisingly, individuals in rural areas, which have lower population and traffic density than urban areas, are expected to experience lower benzene-related exposures than individuals in urban areas (Table V.B-5). Moreover, data from the 14 cities modeled in Glen and Shadwick (1998) demonstrate that average CO levels increase proportionally with population density (Table V.B-6). HAPEM-MS3 toxics exposure estimates will follow the same trend, since CO is used as a surrogate for toxics.

Table V.B-5
On-Highway vehicle portion of 1996 benzene exposure estimates for 10 urban areas, and urban and rural areas nationwide, based on HAPEM-MS3 exposure modeling

Urban Area	Average Highway Vehicle Benzene Concentration ($\mu g/m^3$) in 1996
Atlanta	0.87
Chicago	0.50
Denver	0.74
Houston	0.55
Minneapolis	1.15
New York	0.82
Philadelphia	0.53
Phoenix	1.31
Spokane	0.95
St. Louis	0.49
Urban Area Average	0.77
Rural Area Average	0.45

Table V.B-6
Annual Average Ambient CO Levels as a Function of Population Density, 14 Cities

Population Density at Monitor (residents per square mile)	Average CO Levels, 1990 (ppm)
< 300	1.10
300 - 1,000	1.01
1,000 - 2,000	1.19
2,000 - 5,000	1.35
5,000 - 10,000	1.41
> 10,000	1.97
Overall Average	1.32

d. Seasonal variation

Average nationwide exposures to on-highway vehicle air toxics are much higher in winter than in summer (Table V.B-7). This is primarily due to higher cold start emissions in winter.

Table V.B-7
Seasonal Average Nationwide Exposures (µg/m^3) Attributable to On-Highway Vehicle Emissions, for the General Population, 1996

Pollutant	Winter	Spring	Summer	Fall
Benzene	0.90	0.64	0.55	0.74
Acetaldehyde	0.55	0.35	0.29	0.42
Formaldehyde	0.53	0.35	0.30	0.40
1,3-Butadiene	0.13	0.06	0.03	0.08
Diesel PM	0.88	0.74	0.65	0.76

4. Impact of Current On-Highway Vehicle Control Programs on Toxics Exposure

Projected emissions reductions resulting from programs currently in place will result in proportional reductions in inhalation exposure levels attributable to on-highway vehicles. Figure V.B-2 presents exposure estimates for benzene, formaldehyde, acetaldehyde,1,3-butadiene, and diesel PM in 1990, 1996, 2007, and 2020, using the methodology described in Section V.B.1. These estimates assume implementation of Phase II reformulated gasoline, the National Low Emission Vehicle (NLEV) program, Tier 2 emissions standards with 30 ppm sulfur gasoline, 2004 heavy-duty standards, and proposed 2007 heavy duty standards. With these controls, by 2020 we expect that inhalation exposure to benzene attributable to on-highway vehicles will decrease from 1990 levels by 74 percent, exposure to formaldehyde by 75 percent, exposure to acetaldehyde by 65 percent, exposure to 1,3-butadiene by 73 percent, and exposure to diesel PM by 93 percent.

5. Sensitivity Analyses

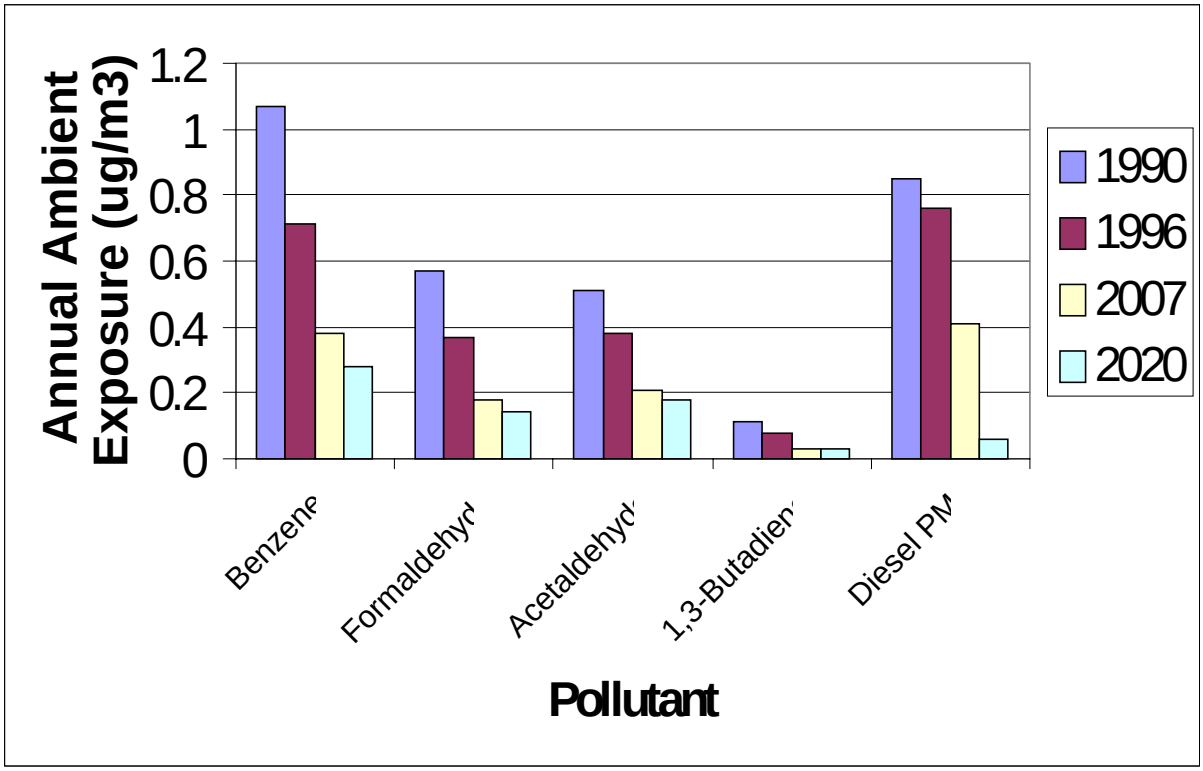
Below is a discussion of the key sources of uncertainty and variability in the models and data affecting exposure. The Agency has conducted some sensitivity analyses to address these issues. However, additional aspects of sensitivity and uncertainty must be explored before we are ready to release results.

1) Representativeness of, and Variability in, Measured Ambient Carbon Monoxide Levels. Toxics exposure estimates are based on measured ambient CO concentrations averaged over 10 selected urban areas. Important limitations of these data relate to variations in CO levels both within and among urban areas, and to the spatial variations in CO levels relative to exposure locations for particular potential highly exposed or sensitive groups (e.g., children, the elderly, ethnic minorities).

2) Estimates of Present and Future HAPs Emissions. The MOBTOX5b and PART5 models include data sets related to vehicle and fleet characteristics, VMT accumulation, climatic conditions, and fuel characteristics. These input data relate to the estimated characteristics of both "current" (1990) and "future" (1996, 2007, and 2020) vehicle fleets.

3) Appropriateness of CO as a Proxy for Estimating Toxics Exposures. Aside from the uncertainties associated with the ambient CO measurements themselves, the inhalation exposure estimates assume that ambient CO concentrations are an appropriate starting point for estimating toxics exposures. This assumption is only valid if (1) the average contributions of mobile sources to ambient CO and toxics exposures levels are similar

Figure V.B-2
Exposure levels for four gaseous toxics and diesel PM under currently planned controls and with 2007 Standards for Heavy Duty Engines (ug/m^3)



across exposure areas, and (2) degradation or secondary generation of toxics due to photochemical and other reactions can be neglected or accurately modeled. Also, HAPEM-MS3 accounts only for exposures to ambient air toxics. Micro-environmental sources of exposure are not accounted for. If the generation patterns or atmospheric fate and transport characteristics of the toxics differ significantly from those of CO, or if there are significant sources of toxics emissions that are not addressed in the current exposure assessment methodology, the toxics exposure estimates may underestimate true exposure.

4) ***Micro-environmental Factors and Time-Activity Patterns.*** The revised ME factors and time use patterns supporting EPA's exposure assessment have been criticized as substantially underestimating exposures to certain potentially sensitive populations and highly exposed demographic groups, and failing to capture important inter-individual variability in behavior and exposure patterns within demographic groups.[36]

5) ***Methods Used to Extrapolate National Urban and Rural Exposures.*** To estimate urban exposures, we developed CO and HAPs emission estimates for urban counties, and scaled them to the estimated average exposure for the 10 modeled urban areas. National average

rural exposures were calculated by assuming that rural HAPs exposures were, on average, a constant fraction of the average urban exposures, scaled to the estimated CO and HAPs emissions in the individual counties. Both of these procedures have some uncertainties.

C. Exposures in Micro-environments

Exposures to air toxics result not only from intrusion of ambient air into micro-environments, but also from emissions of air toxics originating within micro-environments. If the contribution to long-term exposure from mobile sources within micro-environments is large, the above estimates of exposures to ambient concentrations could significantly underestimate the actual risk from exposure to motor vehicle air toxics. Among the most significant micro-environments where such exposures might occur are in vehicles, at service stations during refueling, inside homes with attached garages, and near roadways. Unfortunately, measurements of toxics concentrations within specific micro-environments is very limited, and much of it is dated. We are addressing some of these limitations in the technical analysis plan. This section briefly summarizes what we know about micro-environmental exposures for diesel PM, benzene, 1,3-butadiene, formaldehyde, and acetaldehyde. It should be noted that the information presented here addresses only level of exposure, not the amount of exposure.

1. Diesel PM

Micro-environmental levels of diesel PM can be estimated using elemental carbon as a surrogate. This approach provides some estimates of diesel PM in micro-environments such as in-vehicle concentrations (2.8-36.6 $\mu g/m^3$), near roadways with diesel traffic (diesel PM concentrations are calculated to be 0.7-7.5 $\mu g/m^3$ higher than background), and in schools (0.9-5.5 $\mu g/m^3$).

Recently, elemental carbon measurements were reported for enclosed vehicles driving on Los Angeles roadways.[37] Applying a ratio of diesel PM mass to elemental carbon mass, diesel PM concentrations in the vehicle ranged from approximately 2.8 $\mu g/m^3$ to 36.6 $\mu g/m^3$ depending on the type of vehicle being followed; higher concentrations were observed when the vehicle followed heavy-duty diesel vehicles. CARB also collected elemental carbon near the Long Beach Freeway for four days in May, 1993, and for three days in December, 1993.[38] Using emission estimates from their EMFAC7G model, and elemental/organic carbon composition profiles for diesel and gasoline exhaust, tire wear and road dust, CARB estimated the contribution of the freeway traffic to diesel PM concentrations. For the two days of sampling in December 1993, diesel exhaust from vehicles on the nearby freeway were estimated to contribute average concentrations ranging from 0.7 $\mu g/m^3$ to 4.0 $\mu g/m^3$ excess diesel PM above background concentrations with a maximum 24 hour measurement of 7.5 $\mu g/m^3$.

In a study designed to investigate relationships between diesel exhaust exposure and respiratory health of children in the Netherlands, elemental carbon measurements were collected in 24 schools located from 47 to 377 meters from a freeway.[39] Thirty-two samples were collected

inside schools and 46 samples were collected outside the schools. Preliminary estimates of elemental carbon concentrations indoors and outdoors ranged from 1 $\mu g/m^3$ to 6 $\mu g/m^3$, with an average between 3 and 4 $\mu g/m^3$. These results correspond to diesel PM concentrations ranging from 0.9 $\mu g/m^3$ to 5.5 $\mu g/m^3$ with a mean of approximately 2.7-3.7 $\mu g/m^3$. Total PM2.5 concentrations inside the schools averaged 23.0 $\mu g/m^3$ while PM2.5 outside was only slightly higher (24.8 $\mu g/m^3$), suggesting extensive intrusion of outdoor air into the school environment.

2. Benzene

The information contained in Table V.C-1 summarizes data from several studies that have measured micro-environmental exposures to benzene. These studies are the EPA's Total Exposure Assessment Methodology (TEAM) Study,[40] Commuter's Exposure to Volatile Organic Compounds, Ozone, Carbon Monoxide, and Nitrogen Dioxide,[41] In-Vehicle Air Toxics Characterization Study in the South Coast Air Basin,[42] Air Toxics Micro-environment Exposure and Monitoring Study,[43] a 1998 California EPA study of in-vehicle concentrations (California EPA, 1998), a 2000 study of commuter exposures in Detroit, MI,[44] and a 1993 NIOSH study of concentrations at service stations.[45]

The TEAM Study was planned in 1979 and completed in 1985. The goals of this study were: 1) to develop methods to measure individual total exposure (exposure through air, food and water) and resulting body burden to toxic and carcinogenic chemicals, and 2) to apply these methods with a probability-based sampling framework to estimate the exposures and body burdens of urban populations in several U.S. cities. This was achieved through the use of small personal samplers, a specially designed spirometer (used to measure the chemicals in exhaled breath), and a survey designed to insure the inclusion of potentially highly exposed groups.

The study, Commuter's Exposure to Volatile Organic Compounds, Ozone, Carbon Monoxide, and Nitrogen Dioxide (Chan et al., 1989), focused on the driver's exposure to VOC's in the Raleigh, NC area. The primary objective of this study was to measure driver's exposure to all possible VOC and some combustion gases during one rush-hour driving period (18 sampling days, two trips per day). Factors that could influence drivers' exposure, such as different roadways, car models, vehicle ventilation modes and times of driving were also tested. Car exterior samples were also collected from the exterior of the moving vehicles by setting sampling probes on the middle of the car roof. Another objective was to find the relationships between fixed-site measurements and drivers' exposure (one fixed-site monitor matched per trip). Lastly, the pedestrian's exposure to VOC in urban walking was evaluated with six walking samples.

The study by the South Coast Air Quality Management District (SCAQMD), In-Vehicle Air Toxics Characterization Study in the South Coast Air Basin (Shikiya et al., 1989), was conducted to refine the assessment of health risk due to exposure to toxic air pollutants. This study examined the relative contribution of in-vehicle exposure to airborne toxics to an individual's total exposure by measuring concentrations within vehicle interiors during home-to-work commutes. Other objectives of this study were to develop statistical and concentration

Table V.C-1
Micro-environmental concentrations of benzene (µg/m^3)

Scenarios	In-Vehicle		Service Station		Parking Garage		Office Building	
	Mean	Max.	Mean	Max.	Mean	Max.	Mean	Max.
TEAM Study (EPA, 1987b)	--	40-60[a]	--	3000[b]	--	--	--	--
Raleigh, NC Study[c] (Chan et al., 1989)	10.9	42.8	--	--	--	--	–	--
SCAQMD Study[d] (Shikiya et al., 1989)	42.5	267.1	--	--	--	--	--	--
SCAQMD Study[e] (Wilson et al., 1991)	--	--	--	288	--	67.1	--	16.0
Los Angeles, CA (Cal. EPA, 1998)	10-22	--	--	--	--	--	--	--
Sacramento, CA (Cal. EPA, 1998)	3-15	--	--	--	--	--	--	--
Detroit, MI[f] (Batterman, et. al., 2000)	5.3	--						
NIOSH (Hartle, 1993)	--	--	195	--	--	--	--	--

[a]Maximum benzene concentrations could not be reliably determined because exposures were averaged over a 12-hour period; however, maximum concentrations of 3 to 4 times normal exposures were calculated.
[b]This concentration was estimated, rather than measured directly.
[c]A one-hour measurement was taken for each experimental trip.
[d]The estimated sampling time period was 1.5 hours/round-trip.
[e]The measurements from this study are five-minute levels.
[f]Measurements taken from interiors of urban buses.

measurement methods for a vehicular survey and to identify measures which might reduce commuters' exposure to toxic air pollutants. Vehicles of home-to-work commuters from a non-industrial park were sampled for in-vehicle concentrations of 14 toxic air pollutants, carbon monoxide, and lead.

The second study by SCAQMD, Air Toxics Micro-environment Exposure and Monitoring Study (Wilson et al., 1991), attempted to monitor exposures to motor vehicle emissions in micro-environments other than in-vehicle. The study randomly sampled 100 self-service filling stations and took samples at 10 parking garages and 10 offices nears the garages in Los Angeles, Orange, Riverside, and San Bernadino Counties of Southern California. The study took five-minute samples of 13 motor vehicle air pollutants in each micro-environment and in the ambient environment.

The 1998 California EPA study characterized the concentration of several pollutants inside vehicles during commutes in Sacramento and Los Angeles. A variety of scenarios were assessed, based on such variables as roadway type, traffic congestion, ventilation setting, and vehicle type.

Another recent study conducted in Detroit characterized concentrations of benzene and a number of other compounds inside urban buses and compared them to ambient samples collected on the outside of passenger cars, and to ambient monitor values. The study found that concentrations inside buses were representative of concentrations in the ambient air collected along bus routes. The concentrations inside buses were three to five times higher than concentrations at fixed site monitors in Detroit.

The 1993 NIOSH study assessed benzene and MTBE concentrations and service station attendant exposures at service stations with and without Stage II vapor recovery. The study found that Stage II vapor recovery did not significantly reduce exposure to benzene during refueling. However, the efficiency of Stage II vapor recovery has improved over the years. NESCAUM has suggested that Stage II vapor recovery systems are greater than 90% effective at capturing MTBE and benzene vapors during refueling.[46] These systems would therefore be expected to reduce exposure beyond that shown in this initial exposure assessment.

In general, these micro-environmental exposures are short in duration, and thus are of greater relevance to potential short-term risks rather than potential chronic risks. One micro-environmental source of exposure which could be more significant is inside homes with attached garages. Results of sensitivity analyses on HAPEM-MS3 enable us to estimate how significant exposures from this micro-environmental source might be.[47] HAPEM-MS3 was run with alternative sets of micro-environmental factors. The only factors that differed were those for residential garages and homes with attached garages. The second set of factors were designed to account for evaporative benzene emissions in these micro-environments and were used to adjust CO concentrations upward. However, these factors were developed using data collected before vehicles had evaporative emission controls (circa 1980).[48] Since MOBILE data indicate that in conventional fuel areas with no I/M, evaporative emissions have declined 60% between 1980 and

1990, we scaled the CO concentrations to account for the reduction. The result is average exposure concentrations that are 90% higher than the estimates in Figure V.B-2. Such an estimate assumes all evaporative benzene emissions originating in attached garages are from vehicles, with none from gasoline cans, lawnmowers, snowblowers, solvents, and so on. Thus, this estimate of 90% higher exposure to motor vehicle benzene emissions should be viewed as an upper bound.

3. Acetaldehyde

The only data on micro-environmental exposures to acetaldehyde from motor vehicles are from the In-Vehicle Air Toxics Characterization Study in the South Coast Air Basin (Shikiya et al., 1989), which focused on the driver's exposure to VOC's in the southern California area. The in-vehicle exposure level of acetaldehyde was determined in this study to have a mean of 13.7 $\mu g/m^3$ and a maximum measured level of 66.7 $\mu g/m^3$.

4. Formaldehyde

The information contained in Table V.C-2 is excerpted from three studies that have measured micro-environment exposures to formaldehyde. These two studies are the In-Vehicle Air Toxics Characterization Study in the South Coast Air Basin (Shikiya et al., 1989), Air Toxics Micro-environment Exposure and Monitoring Study (Wilson et al., 1991), and the 1998 California EPA Study of in-vehicle concentrations (California EPA, 1998).

Maximum micro-environment exposure levels of formaldehyde related to motor vehicles were determined in these studies to range from 4.9 $\mu g/m^3$ for exhaust exposure at a service station to 41.8 $\mu g/m^3$ for exhaust exposure at a parking garage.

5. 1,3-Butadiene

There are very few data on micro-environmental exposures to 1,3-butadiene. Some in-vehicle measurements were taken as part of the Commuter's Exposure to Volatile Organic Compounds, Ozone, Carbon Monoxide, and Nitrogen Dioxide (Chan et al., 1989). The in-vehicle exposure level of 1,3-butadiene was determined in this study to have a mean of 3.0 $\mu g/m^3$ and a maximum measured level of 17.2 $\mu g/m^3$. Exterior to the vehicle, the mean was determined to also be 3.0 $\mu g/m^3$ with a maximum level of 6.9 $\mu g/m^3$. The 1998 California EPA study of in-vehicle concentrations (California EPA, 1998) also included 1,3-butadiene measurements. In-vehicle concentrations of 1,3-butadiene ranged from 1 to 4 $\mu g/m^3$ in Sacramento, and 2 to 6 $\mu g/m^3$ in Los Angeles.

Table V.C-2
Micro-environmental exposure to formaldehyde (µg/m^3)

Scenarios	In-Vehicle		Service Station		Parking Garage		Office Building	
	Mean	Max.	Mean	Max.	Mean	Max.	Mean	Max.
SCAQMD Study[a] (Shikiya et al., 1989)	15.4	35.4	--	--	--	--	--	--
SCAQMD Study[b] (Wilson et al., 1991)	--	--	--	4.9	--	41.8	--	44.2
Los Angeles, CA (Cal. EPA, 1998)	<MQL[c] to 22							
Sacramento, CA (Cal. EPA, 1998)	5-14							

[a]The estimated sampling time period was 1.5 hours/round-trip.
[b]The measurements from this study are five-minute levels.
[c]<MQL - below method quantification limit

Appendix 1: Mean Highway and Nonroad Contributions to Nationwide Concentrations of Mobile Source Air Toxics in 1996, from ASPEN Modeling Using the 1996 NTI

Pollutant	Mean Ambient Conc. ($\mu g/m^3$)		
	Highway Contribution (% of Total)	Nonroad Contribution (% of Total)	Total
1,3-Butadiene	0.05 (63)	0.02 (25)	0.08
Acetaldehyde	0.40 (54)	0.27 (36)	0.74
Acrolein	0.05 (45)	0.04 (36)	0.11
Arsenic Compounds	6.5E-07 (0.4)	7.7E-06 (4.8)	1.6E-04
Benzene	0.55 (39)	0.24 (17)	1.39
Chromium Compounds	4.2E-05 (1.3)	1.4E-04 (4.2)	3.3E-03
Dioxins/Furans	2.9E-10	2.0E-10	N.A.
Ethylbenzene	0.32	0.11	N.A.
Formaldehyde	0.38 (30)	0.48 (38)	1.28
Lead Compounds	5.9E-05	3.4E-03	N.A.*
Manganese Compounds	1.7E-05 (0.4)	1.4E-04 (3.3)	4.3E-03
MTBE	0.44	0.29	N.A.
n-Hexane	0.24	0.08	N.A.
Nickel Compounds	3.3E-05	3.2E-04	N.A.
POM (as sum of 7-PAH)	1.2E-04 (10)	2.6E-05 (2.2)	1.2E-03
Styrene	0.04	0.005	N.A.
Toluene	2.18	0.43	N.A.
Xylenes	1.20	0.40	N.A.

*Included in the National Scale Analysis, but nationwide concentrations from all sources not yet available

Appendix 2: HAPEM-MS3 Microenvironmental Factors

Lag=0 indicates current ambient value used,
Lag=-1 indicates previous hour's ambient value used.
All additive terms are set to zero.

Micro-environment	Multiplicative Factor	Lag
In transit, car	0.960	0
In transit, bus	1.650	0
In transit, truck	1.140	0
In transit, van	0.960	0
Indoors, public garage	0.590	-1
Outdoors, in a parking lot	0.570	0
Outdoors, along a roadway	0.850	0
In transit, motorcycle	1.030	0
Indoors, service station	0.870	-1
Outdoors, service station	0.000	0
Indoors, residential garage	0.780	-1
Indoors, in a repair shop	1.320	0
Indoors, home	0.380	-1
Indoors, office	0.380	-1
Indoors, store	0.410	-1
Indoors, restaurant	0.710	-1
Indoors, mfg. facility	0.470	-1
Indoors, school	0.450	-1
Indoors, church	0.280	-1
Indoors, shopping mall	2.110	-1
Indoors, auditorium	0.120	-1
Indoors, health care facility	0.310	-1

Micro-environment	Multiplicative Factor	Lag
Indoors, public building	0.350	-1
Indoors, other location	0.790	-1
Indoors, not specified	0.790	-1
Outdoors, construction	0.960	0
Outdoors, residential grounds	0.550	0
Outdoors, school grounds	0.960	0
Outdoors, sports arena	0.280	0
Outdoors, park/golf course	0.280	0
Outdoors, other location	0.600	0
Outdoors, not specified	0.960	0
In transit, train	0.960	0
In transit, airplane	0.000	0

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Chapter 6: Motor Vehicle-Based Controls of Mobile Source Air Toxics

Introduction

The Chapters 4 and 5 discussed the reductions in toxics emissions and exposure that have already resulted from the Agency's mobile source volatile organic compound (VOC) and diesel particulate matter (PM)) control programs. This chapter presents the rationale for our determination that additional motor vehicle-based controls (beyond those already adopted) not be established at this time under §202(l)(2). This is based on considerations of the technical feasibility and cost of further controls at this time. The first section presents an overview of vehicle-based emission control technologies and their role in reducing air toxics. The second section reviews the Agency's most recent actions to further reduce VOC and PM emissions from on-highway vehicles and engines.

A. Vehicle-Based Technologies that Control Air Toxics

To better understand the nature of mobile source air toxics (MSAT) and their control it is helpful to categorize the MSAT into three groups: gaseous organic toxics, DPM + DEOG (as measured by diesel PM), and metals. For each group, the following sections present an overview of these toxics and the impact of emission control technology on these toxics. It is well documented that the Agency's effort to control criteria pollutants and their precursors through motor vehicle based controls has dramatically reduced VOC emissions. As discussed below and in Chapter 4, these VOC controls have contributed to large reductions in gaseous MSATs. Similarly, other EPA controls have had major impacts on the non-gaseous MSATs as well.

1. Gaseous Organic Toxics

Fifteen of the 21 MSATs are gaseous organics. These 15 gaseous toxics can be further categorized depending on whether they are fuel components or combustion products. Those that are gasoline fuel components may be found in both evaporative and exhaust emissions, while the non-fuel components that are combustion products are found only in exhaust emissions (see Table IV-A.1).

Table VI.A-1
Gaseous MSATs

Gaseous Organic Toxics	Fuel Component	Exhaust Component
Acetaldehyde		YES
Acrolein		YES
Benzene	YES	YES
1,3-Butadiene		YES
Dioxin/Furans		YES
Ethylbenzene	YES	YES
Formaldehyde		YES
n-Hexane	YES	YES
MTBE	YES	YES
Naphthalene	YES	YES
POM	YES	YES
Styrene		YES
Toluene	YES	YES
Xylene	YES	YES

Vehicle-based controls are effective in reducing all 15 of the gaseous air toxics. The toxics that are fuel components may result in vehicle exhaust emissions and evaporative emissions (depending on the volatility of the compound), and can be reduced through exhaust and evaporative emission control as well by controlling the fuel composition directly. For those compounds that are formed during the combustion process, control strategies rely on exhaust emission control technology and changes to fuel composition.

a. Exhaust Controls

This section describes our requirements for vehicle controls that result in the control of exhaust VOC emissions in general. The Agency is not aware of any exhaust emission controls that selectively work only on the gaseous toxics listed in Table VI.A-1. All of the control technologies of which we are aware control other VOCs as well. It is worth noting that since almost all of the compounds listed in Table VI.A-1 contain either oxygen functional groups or unsaturated carbon bonds, the VOC controls that are described here are generally slightly more

effective at oxidizing these toxics than many other exhaust VOCs.[171] Table VI.A-2 shows typical exhaust fractions of MSATs for a gasoline-fueled vehicle operating with a typical fuel. (Note: actual in-use emissions can vary significantly for different vehicles and different fuels.) As is true for this typical case, MSATs can comprise one-quarter of all exhaust VOC emissions from a gasoline-fueled vehicle.

Table VI.A-2
Gaseous MSATs in Typical Gasoline-Fueled Vehicle Exhaust

Toxic	Exhaust Fraction of VOC[172]
Acetaldehyde	0.5 %
1,3-Butadiene	0.5 %
Formaldehyde	1 %
Benzene	4 %
Toluene	10 %
Other Gaseous MSATs	9 %
Total	25%

VOCs are the result of incomplete combustion occurring in a vehicle's engine. Some VOC emissions are unburned fuel and engine oil, some are combustion byproducts from partially-burned fuel and engine oil. This is true for both gasoline-fueled vehicles and diesel vehicles. To reduce both types of VOC emissions from gasoline-fueled vehicles, manufacturers have designed their engines to achieve virtually complete combustion and have installed catalytic converters in the exhaust system. As discussed later in this section, a similar approach can be used with diesel engines. In order for these controls to work well for gasoline-fueled vehicles, it is necessary to maintain the mixture of air and fuel at a nearly stoichiometric ratio (that is, just enough air to completely burn the fuel). Poor air-fuel mixture can result in significantly higher emissions of incompletely combusted fuel. Current generation highway vehicles are able to maintain stoichiometry by using closed-loop electronic feedback control of the fuel systems. As part of these systems, technologies have been developed to closely meter the amount of fuel entering the combustion chamber to promote complete combustion. Sequential multi-point fuel injection delivers a more precise amount of fuel to each cylinder independently and at the appropriate time increasing engine efficiency and fuel economy. Electronic throttle control offers a faster response to engine operational changes than mechanical throttle control can

[171] Siegl, W.O., et al., A Comparison of Conversion Efficiencies of Individual Hydrocarbon Species Across Pd- and Pt-Based Catalysts as a Function of Fuel-Air Ratio, SAE 982549.

[172] EPA Speciate Database http://www.epa.gov/ttnchie1/spec/index.html

achieve, but it is currently considered expensive and only used on some higher-price vehicles. The greatest gains in fuel control can be made through engine calibrations -- the algorithms contained in the powertrain control module (PCM) software that control the operation of various engine and emission control components/systems. As microprocessor speed becomes faster, it is possible to perform quicker calculations and to increase response times for controlling engine parameters such as fuel rate and spark timing. Other advances in engine design have also been used to reduce engine-out emissions of VOCs, including: the reduction of crevice volumes in the combustion chamber to prevent trapping of unburned fuel; "fast burn" combustion chamber designs that promote swirl and flame propagation; and multiple valves with variable-valve timing to reduce pumping losses and improve efficiency. Improvements in the overall efficiency of the vehicle can also reduce emissions by reducing the amount of fuel that is consumed. These technologies are discussed in more detail in the RIA for the Tier 2 FRM.[173]

As noted above, manufacturers are also using aftertreatment control devices to oxidize VOCs emitted by the engine. The primary approach is to use a three-way catalyst (TWC) that simultaneously controls VOCs, CO, and NOx. New three-way catalysts are so effective that once a TWC reaches its operating temperature, VOC emissions are virtually undetectable (0.01 gpm or less).[174] Manufacturers are now working to improve the durability of the TWC and to reduce light-off time (that is, the amount of time necessary after starting the engine before the catalyst reaches its operating temperature and is effectively controlling VOCs and other pollutants). EPA expects that manufacturers will be able to design their catalyst systems so that they light off within less than thirty seconds of engine starting. Other potential exhaust aftertreatment systems that could further reduce cold-start emissions are thermally insulated catalysts, electrically heated catalysts, and HC adsorbers (or traps). Each of these technologies, which are discussed below, offer the potential for VOC reductions in the future. There are technological, implementation, and cost issues that still need to be addressed, and at this time, it appears that these technologies would not be a cost-effective means of reducing toxic emissions on a nationwide basis. However, the cost-effectiveness of these technologies for reducing toxic emissions would depend on the technology costs and emission reductions of these technologies, which remain somewhat uncertain, as well as the costs of traditional exhaust emission control technologies.[175] The cost-effectiveness would also be dependent on the manner in which these costs are apportioned with respect to the other exhaust pollutants (i.e., VOC, NOx, and CO).

Thermally insulated catalysts maintain sufficiently high catalyst temperatures by surrounding the catalyst with an insulating vacuum. Prototypes of this technology have

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[173] http://www.epa.gov/otaq/tr2home.htm#Documents. EPA 420-R-99-023

[174] McDonald, J., L. Jones, Demonstration of Tier 2 Emission Levels for Heavy Light-Duty Trucks, SAE 2000-01-1957.

[175] "Analysis of Cold-Start Emission Controls", November 15, 2000, Docket #A-2000-IV-B-6.

demonstrated the ability to store heat for more than 12 hours.[176] Since ordinary catalysts typically cool down below their light-off temperature in less than one hour, this technology could reduce in-use emissions for vehicles that have multiple cold-starts in a single day. However, this technology would have less impact on emissions from vehicles that have only one or two cold-starts per day.

Electrically-heated catalysts reduce cold-start emissions by applying an electric current to the catalyst before the engine is started to get the catalyst up to its operating temperature more quickly.[177] These systems require a modified catalyst, as well as an upgraded battery and charging system. These can greatly reduce cold-start emissions, but could require the driver to wait until the catalyst is heated before the engine would start to achieve optimum performance.

Hydrocarbon adsorbers are designed to trap VOCs while the catalyst is cold and unable to sufficiently convert them. They accomplish this by utilizing an adsorbing material which holds onto the VOC molecules. Once the catalyst is warmed up, the trapped VOCs are automatically released from the adsorption material and are converted by the fully functioning downstream three-way catalyst. There are three principal methods for incorporating an adsorber into the exhaust system. The first is to coat the adsorber directly on the catalyst substrate. The advantage is that there are no changes to the exhaust system required, but the desorption process cannot be easily controlled and usually occurs before the catalyst has reached light-off temperature. The second method locates the adsorber in another exhaust pipe parallel with the main exhaust pipe, but in front of the catalyst and includes a series of valves that route the exhaust through the adsorber in the first few seconds after cold start, switching exhaust flow through the catalyst thereafter. Under this system, mechanisms to purge the adsorber are also required. The third method places the trap at the end of the exhaust system, in another exhaust pipe parallel to the muffler, because of the low thermal tolerance of adsorber material. Again a purging mechanism is required to purge the adsorbed VOCs back into the catalyst, but adsorber overheating is avoided. One manufacturer who incorporates a zeolite hydrocarbon adsorber in its California SULEV vehicle found that an electrically heated catalyst was necessary after the adsorber because the zeolite acts as a heat sink and nearly negates the cold start advantage of the adsorber. This approach has been demonstrated to effectively reduce cold start emissions.

Historically, control of VOC emissions from diesel engines has relied primarily on technologies that improve combustion. Because diesel engines are designed to operate with very high air/fuel ratios (that is, with excess oxygen), they have inherently lower VOC emissions than gasoline-fueled engines. Nevertheless, since combustion is not always complete, diesels do have significant VOC emissions. Recent efforts to lower PM emissions have led to much more

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[176] Burch, S.D., and J.P. Biel, SULEV and "Off-Cycle" Emissions Benefits of a Vacuum-Insulated Catalytic Convert, SAE 1999-01-0461.

[177] Laing, P.M., Development of an Alternator-Powered Electrically-Heated Catalyst System, SAE 941042.

complete combustion for diesel engines being produced today. This has resulted in VOC emissions from diesel engines being about 0.2-0.3 g/bhp-hr. It is possible to achieve even lower VOC emission levels by incorporating an oxidation catalyst in the exhaust. Since these catalysts are generally used to control PM, they are discussed in more detail in that section.

b. Evaporative Controls

Evaporative emissions occur when fuel evaporates and is vented to the atmosphere. They can occur during refueling, while the vehicle is operating, or while it is parked. This section describes the control of evaporative VOC emissions from on-highway gasoline vehicles. Diesel vehicles do not have significant evaporative emissions because diesel fuel has such a high boiling point (over 300°F). Table VI.A-3 shows typical evaporative fractions of MSATs for a gasoline-fueled vehicle operating with a typical fuel. MSATs can comprise one-tenth of all evaporative VOC emissions from a gasoline-fueled vehicle.

Table VI.A-3
Gaseous MSATs in Typical Gasoline-Fueled Evaporative Emissions

Toxic	Evaporative Fraction of VOC[178]
Acetaldehyde	0 %
1,3-Butadiene	0 %
Formaldehyde	0 %
Benzene	2 %
Toluene	4 %
Other MSATs	5 %
Total	11%

In general, evaporative emission control is accomplished by sealing the fuel system, and forcing all vented vapors to go through a charcoal canister, which adsorbs any fuel vapors present. If the canister is sufficiently large, evaporative emissions from venting can be virtually eliminated for normal operating conditions. The canister is occasionally flushed with fresh air which "purges" the fuel vapors from the canister. These purged gases are then routed into the engine air intake system so that they can be combusted in the engine. Since using the purge gases for intake air instead of fresh air changes the air/fuel ratio, it must be coordinated with the fueling control to ensure that the engine does not run rich. However, with modern electronic controls, this is relatively straightforward.

[178] EPA Speciate Database http://www.epa.gov/ttnchie1/spec/index.html

Other sources of evaporative emissions are fuel vapor venting, fuel spit-back, and post-fill drip during refueling; fuel permeation through the fuel tank and fuel lines; and leaking connections. The refueling emissions can be controlled by optimizing the nozzle-vehicle interface, including the fill neck design, and capturing the vapors with a charcoal cannister. These systems are called onboard refueling vapor recovery (ORVR) systems. For design efficiency, ORVR systems are integrated with the evaporative systems. The refueling vapors are vented to the same cannister that is used for other evaporative emissions and are purged into the engine along with the other vapors. The primary physical difference between an evaporative control system and an ORVR system is the fillneck seal. In most cases, the fillneck seal is achieved using a liquid seal. The liquid seal can be as simple as incorporating a "J-tube" into the fillneck, much like the trap in a sink drain. ORVR systems also include anti-spitback valves to control fuel spillage during refueling.

Permeation and leaks can be greatly reduced by reducing the number of hoses, fittings and connections, and by using less permeable hoses and lower loss fittings and connections. Fluoropolymer materials can be added as liners to hose and component materials to yield large reductions in permeability over such conventional materials as monowall nylon. In addition, fluoropolymer materials can greatly reduce the adverse impact of alcohols in gasoline on permeability of evaporative components, hoses and seals. Manufacturers are also beginning to incorporate “returnless” fuel injection systems. These systems use more precise fuel pumping and metering to eliminate the return of heated fuel from the injectors, which is a significant source of fuel tank heat and vapor generation. The elimination of return lines also reduces the total length of hose on the vehicle and also reduces the number of fittings and connections that can leak.

The test procedures and requirements associated with EPA's evaporative and refueling emission standards essentially require manufacturers to design for zero emission levels. Upgrades to evaporative emission requirements and expansion of the refueling standard coverage as part of recent and proposed EPA rules (see Section B) have gone even further towards the goal of eliminating gasoline evaporation as a potential source of MSATs. More information on evaporative control technologies is contained in the RIAs for the 1994 "Refueling Emissions Regulations for Light-duty Vehicles and Trucks and Heavy-duty Vehicles" and the recent Tier 2 Rulemaking.

2. Diesel Particulate Matter and Diesel Exhaust Organic Gases

Diesel exhaust is a complex mixture of carbon particles and associated organics and inorganics, and it is not known what fraction or combination of fractions cause the health effects discussed in Chapter 3 that have been observed with exposure to diesel exhaust. Gaseous components of diesel exhaust include benzene, formaldehyde, acetaldehyde, 1,3-butadiene, acrolein, and semi-volatile organic compounds, some of which are nitrated. Diesel PM has historically been divided into three primary constituents: unburned elemental carbon particles (or "soot"), which make up the largest portion of the total PM; the soluble organic fraction (SOF),

which consists of unburned hydrocarbons (including polycyclic organic matter and dioxins) that have condensed into liquid droplets or have condensed onto unburned carbon particles; and sulfates, which result from oxidation of fuel-borne sulfur in the engine's exhaust. Diesel exhaust also includes metals, which are discussed in the next section. EPA and engine manufacturers have historically focused on control of PM emissions rather than gas-phase organics for two reasons. First, PM-reducing technologies generally also reduce gas-phase organic emissions. Second, controlling PM emissions has proven to be significantly more challenging than controlling gas phase organic emissions from diesel engines. Thus, the remainder of this section describes PM emission controls. The extent to which diesel exhaust organic gas emissions are controlled by VOC or other hydrocarbon controls is discussed above in section 6.A.1.

Diesel engines have made great progress in lowering engine out emissions from uncontrolled levels between 0.8 and 1.0 g/bhp-hr PM to 0.1 g/bhp-hr PM for current engines. These reductions came initially with improvements to combustion and fuel systems. Several exhaust aftertreatment devices have also been developed to control PM. They generally fall into two categories: diesel oxidation catalysts (DOCs) and particulate filters (or traps). DOCs have been shown to be durable in-use, but they control only a relatively small fraction of the total PM mass (mostly the soluble fraction, which is typically less than 30 percent of the total). Nevertheless, DOCs have been shown to significantly reduce the emissions of toxic organics from diesel engines.[179, 180]

PM traps work by passing the exhaust through a ceramic or metallic filter to collect the PM. The collected PM must then be burned off the filter before the filter becomes plugged. This burning off of collected PM is referred to as "regeneration," and can occur either: on a periodic basis by using base metal catalysts or an active regeneration system such as an electrical heater, a fuel burner, or a microwave heater; or, on a continuous basis by using precious metal catalysts. Uncatalyzed diesel particulate traps demonstrated high PM trapping efficiencies many years ago, but the regeneration characteristics were not dependable. As a result, some systems employed electrical heaters or fuel burners to improve upon regeneration, but these complicated the system design and still did not provide the durability and dependability required for HD diesel applications.

Catalyzed diesel particulate traps have the potential to provide the same reductions in diesel PM emissions and provide the durability and dependability required for diesel applications. They have lower average backpressure than other traps and they need no extra burners or heaters. Most importantly, however, they are highly efficient at trapping all forms of diesel PM and are reliably regenerated under normal operating conditions typical of a diesel

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[179] McClure, B'T', et al., The Influence of an Oxidation Catalytic Converter and Fuel Composition on the Chemical and Biological Characteristics of Diesel Exhaust Emissions, SAE 920854.

[180] Pataky, G.M., et al., Effects of an Oxidation Catalytic Converter on Regulated and Unregulated Diesel Emissions, SAE 940243.

engine. These catalyzed PM traps are able to provide in excess of 90 percent control of diesel PM. More than one aftertreatment manufacturer is developing these precious metal catalyzed, passively regenerating PM traps. In field trials, they have demonstrated highly efficient PM control and promising durability. A recent publication documents results from a sample of these field test engines after years of use in real world applications. The sampled filters had on average four years of use covering more than 225,000 miles in applications ranging from city buses to garbage trucks to intercity trains. When tested on the U.S. Heavy-Duty Federal Test Procedure (HD FTP), these in-use engines demonstrated PM reductions in excess of 90 percent.[181] It should be noted, however, these catalyzed traps work well only with diesel fuel with very low sulfur content.

Modern catalyzed PM traps have been shown to be very effective at reducing not only PM mass, but overall number of particles emitted. Hawker, et. al., found that a modern catalyzed PM trap reduced the particle count by over 95 percent, including ultrafine particles (< 50 nm), at most tested operating conditions.[182] Particles smaller than 1,000 nanometers (nm) comprise more than 90 percent of PM mass. Of these particles, approximately half of the mass is from particles smaller than 100 to 200 nm. PM traps have very high particle capture efficiencies. Smaller particles (<200 to 300 nm) are captured primarily by diffusional deposition to surfaces within the trap walls. Capture efficiency of primary PM by diffusion actually increases for decreasing particle size. Larger particles are captured primarily by inertial impaction on surfaces due to the tortuous path of the exhaust gases as they pass through the porous trap walls. Capture efficiencies for the elemental carbon fraction (soot) of diesel PM nearing 100 percent are possible with PM traps, with the only remaining PM downstream of the trap being sulfate and a small amount of organic material.

3. Metals

Mobile source toxics include compounds of six metals: arsenic, chromium, lead, manganese, mercury, and nickel. The source of these toxic emissions are trace amounts of these compounds in engine oil or fuel that come from additives, impurities, and products of engine wear. From a vehicle or engine perspective, the primary methods of reducing emission of toxic metals would be to reduce engine wear and oil consumption. (Metal products of engine wear collect in the engine oil, and can be emitted if the oil is burned in the cylinder or otherwise enters the exhaust stream.) Manufacturers already have a strong incentive to reduce both engine wear and oil consumption due to consumer demand. Moreover, manufacturers must limit oil consumption to very low levels in order to comply with the existing PM standards.

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[181] Allansson, et al, European Experience of High Mileage Durability of Continuously Regenerating Diesel Particulate Filter Technology. SAE 2000-01-0480.

[182] Kleeman, M.J., Schauer, J.J., Cass, G. R., 2000, Size and Composition Distribution of Fine Particulate Matter Emitted From Motor Vehicles, Environmental Science and Technology, Vol. 34, No. 7.

It is also worth noting that some engines are equipped with devices that intentionally introduce small amounts of used crankcase oil into the fuel system as a means of disposing of the used oil and capturing its energy content. The effects of using this type of system are essentially the same as the effects of ordinary oil consumption. To the extent that there are toxic metals in the oil, they would be emitted in the exhaust (generally associated with the PM emissions). We currently require that these systems be certified to ensure that they do not cause noncompliance with our standards. The very low heavy-duty on-highway engine PM standards that have been proposed for 2007 may effectively preclude the use of these devices.

B. Emission Control Requirements

The previous section described the emission control technologies that are currently being used as well as others that are projected to be used in the near future. This section describes regulatory requirements that will force the introduction of these projected technologies in future model years, and the impacts that these requirements will have on toxic emissions.

1. Tier 2 Standards for Light-Duty Vehicles

On February 10, 2000, EPA published new "Tier 2" emissions standards for all passenger vehicles, including sport utility vehicles (SUVs), minivans, vans and pick-up trucks. The new standards will ensure that exhaust VOC emissions be reduced to less than 0.1 g/mi on average over the fleet, and that evaporative emissions be reduced by at least 50 percent. ORVR requirements were also extended to medium-duty passenger vehicles. By 2020, these standards will reduce VOC emissions from light-duty vehicles by more than 25 percent of the projected baseline inventory. (See Chapter 4 for a more detailed discussion of the impact of the Tier 2 FRM on VOC inventories.) To achieve these reductions, manufacturers will need to incorporate nearly all available emission controls, including: larger and improved close-coupled catalysts, optimized spark timing and fuel control, improved exhaust systems, and improved evaporative controls. However, the Tier 2 standards will be achievable without using HC traps or electrically heated catalysts. In the Tier 2 rulemaking, EPA determined that these technologies were not likely to be cost-effective in time to meet the standards.

The Tier 2 FRM also included the first federal formaldehyde emission standards for light-duty vehicles. However, it is actually the VOC controls that are expected to provide the toxic emission reduction. According to the Tier 2 RIA, these controls will reduce benzene emissions by more 20,000 tons per year, acetaldehyde by 2,000 tons per year, formaldehyde by 4,000 tons per year, and 1,3-butadiene by more than 2,000 tons per year. Although not calculated for that rule, the standards will also significantly reduce emission of the other gaseous toxics since it will require reductions of exhaust and evaporative VOC emissions in general.

2. Heavy-Duty Engines and Vehicles

EPA recently set new emission standards that will significantly reduce VOC emissions

from heavy-duty vehicles (65 FR 59896, October 6, 2000), and proposed to set more agressive standards (65 Federal Register 35430, June 2, 2000**)**. In the first rulemaking, we set new standards for 2005 and later heavy-duty gasoline vehicles that are projected to reduce exhaust VOC emissions by two-thirds or more. To comply with these 2005 standards, manufacturers are expected to optimize existing emission controls, but are not expected to need to use the more sophisticated controls projected for Tier 2 vehicles. The October 6, 2000 FRM will require all complete vehicles under 10,000 pounds GVWR to comply with the ORVR requirements.

The other rulemaking includes new emission standards that would begin to take effect in 2007, and would apply to all heavy-duty highway engines and vehicles. These proposed standards would require the use of high-efficiency catalytic aftertreatment devices as well as advanced engine technologies. For diesel engines, manufacturers are expected to incorporate catalyzed PM traps that could virtually eliminate both organic PM (elemental carbon and SOF) and VOC emissions from diesel engines during normal operation. However, the engines will still emit some sulfate PM, and can also emit some VOCs during start-up operation where the catalysts are below minimum functional temperature. Fortunately, since diesels are used mostly in commercial applications, almost all operation will occur with a warm catalyst. For gasoline engines manufacturers are expected to incorporate the technologies similar to those that will be used to comply with the Tier 2 light-duty standards. This will include improved fuel injection, fast electronic throttle controls, reduction of crevice volumes, "fast burn" combustion chamber, and improved three-way catalysts. The heavy-duty standards that we proposed are projected to reduce PM emissions by 83,000 tons and VOC emissions by 230,000 tons.

C. Potential for Further Reductions from New Vehicle Standards

Given the technology-forcing nature of the recently finalized Tier 2 emission standards for light-duty vehicles and the new emission standards for heavy-duty vehicles and engines, it is not feasible that manufacturers would be able to further reduce toxic emissions significantly at this time. For both gasoline and diesel vehicles, these standards will result in near-zero exhaust VOC emissions for all operation other than engine starting. Since the only significant exhaust emissions will occur within the first minute after engine starting, further reductions would require manufacturers to develop technologies specifically for this very short window. While start-up controls such as HC traps and electrically-heated catalysts exist, the issues of technological feasibility and cost are significant enough that EPA does not believe it would be appropriate at this time to promulgate more stringent standards based on these technologies.[183] For diesels, carbonaceous PM emissions are expected to be near-zero for all operation, including engine starting. The only PM that is expected to occur in significant amounts will be in the form of sulfate, which cannot be reduced by vehicle-based controls.

Similarly, for evaporative emissions, manufacturers are already required to design their

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[183] "Analysis of Cold-Start Emission Controls", November 15, 2000, Docket #A-2000-IV-B-6.

Tier 2 vehicles to have very low evaporative emissions, and must also account for the effect of alcohol in the fuel on fuel line permeability and in-use performance. For an in-use Tier 2 vehicle, the primary evaporative emissions that are expected to occur would be the result of an occasional leaking connection in the fuel or evaporative control system, or abnormal vehicle operation (e.g. vehicles parked for several days without being driven). While manufacturers are expected to minimize the number of fuel connections used in the vehicle and to use designs that are not prone to leaking, some leaks are still likely to occur in use.

Since metal emissions are primarily the result of the combination of engine wear and oil consumption, manufacturers already have a very strong incentive to minimize metal emissions. Those metal emissions that are not a result of engine wear and oil consumption are caused by contaminants or additives in the fuel, and are thus beyond a vehicle or engine manufacturer's control. An EPA standard for metals set at a level that is feasible for manufacturers would only enforce existing engine designs. Such a standard would not justify the administrative and testing burden that it would cause. As discussed earlier, EPA is addressing in the 2007 heavy-duty rulemaking the issue of blending used oil into diesel fuel.

D. Potential for Further Reductions from In-Use Vehicles

As described in the Response to Comment document, we are not pursuing in-use controls in this rulemaking. Nevertheless, they could be an important part of our future attempts to achieve further reductions in toxic emissions. With the new standards that are coming into effect, manufacturers will be designing and building their vehicles and engines to have very low toxic emissions. However, this does not guarantee that all of these vehicles and engines will have low toxic emissions in use. Malmaintenance of and/or tampering with the emission controls could result in increased toxic emissions. EPA is continually working to improve in-use maintenance and enforce the tampering prohibition. To address the malmaintenance issue, EPA has established onboard diagnostic (OBD) requirements for manufacturers.[184] These OBD provisions require that vehicle manufacturers install dashboard indicators that alert drivers to the need for emission-related maintenance, and electronic monitors that store codes in the vehicle's computer to assist mechanics in the diagnosis and repair of the malfunction. To address both the malmaintenance and tampering issues, EPA is working with states to develop and optimize inspection and maintenance (I/M) programs that monitor the emission performance of in-use vehicles. Historically, these programs have relied on tailpipe testing to identify high-emitting vehicles. However, these programs have begun to rely more on the OBD systems to identify the high-emitting vehicles, as well as the cause of the emission problem.

For heavy-duty vehicles, I/M is more difficult because it is not as easy to test the larger vehicles. However, we recently established new OBD requirements for heavy-duty gasoline vehicles under 14,000 pounds GVWR, and expect to propose similar requirements for all other heavy-duty vehicles in the near future. As these heavy-duty OBD programs take effect, it will be

[184] 58 FR 9467, February 19, 1993.

much more practical to develop heavy-duty I/M programs that will achieve real emission reductions, especially for PM.

Another potential approach that could reduce emissions from existing vehicles is to retrofit these vehicles with improved emission control devices. This could be especially beneficial with heavy-duty vehicles, which can have very long useful lives. A heavy-duty retrofit program is thus appealing because the slow turnover of the diesel fleet makes it difficult to achieve near-term air quality goals through new vehicle standards alone. Some of the exhaust aftertreatment technologies being considered in the recent heavy-duty diesel rule are especially appealing for use in retrofits because they can be fitted to an existing vehicle as add-on devices without major engine modifications, although some of the more sophisticated systems that require careful control of engine parameters may be more challenging. We believe that large diesel PM emission benefits from the existing fleet could be realized as a result of retrofits. However, these technologies will require changes to the fuel. Therefore, we think that where we can consider future fuel and technology changes it is reasonable to defer our analysis on the long-term feasibility of these controls until a later action.

Finally, MSATs can be reduced through Transportation Control Measures (TCMs), which have been in use for several decades to control traffic congestion on major streets and highways. TCMs include a wide variety of measures used to reduce motor vehicle emissions, primarily by reducing the total amount of vehicle miles of travel in an area. A few examples of TCMs include mass transit improvements, ridesharing arrangements, telecommuting and work schedule changes, parking management, and roadway tolls. As noted earlier, most emissions from cars occur just after starting, when the emissions control devices are not fully warmed up. Therefore, transportation control strategies that reduce short trips have the most emissions benefits. Although we have no rules or regulations requiring that states or cities implement TCMs, we are developing guidance that will help more areas implement such programs that should reduce MSAT emissions.

Chapter 7: Fuel Controls

This Chapter contains background information and analyses supporting our anti-backsliding program. We first summarize the refining industry's current gasoline production and the reasons for the current overcompliance with the reformulated gasoline and anti-dumping requirements. We then discuss the means through which we expect the industry to comply with our anti-backsliding requirement. A description of the costs and emission benefits of our program follow, and we end with a discussion of incremental volumes, compliance margins, and the reasons we have excluded California gasoline from our anti-backsliding requirements.

A. Industry and product characterization

1. Description of entities subject to the toxics anti-backsliding requirements

Our toxics anti-backsliding program will apply to every domestic refinery that produces gasoline, every importer of gasoline to the U.S., and every foreign refiner of gasoline that exports to the U.S. There are no new standards applicable to parties who buy, sell, and/or trade gasoline downstream of refineries.

a. Domestic refiners

Approximately 146 domestic refineries collectively produced approximately 2.9 billion barrels of gasoline in 1998 (however, not all refineries produce gasoline). This amounts to approximately 7.9 million barrels per calendar day on average[185]. Refineries are often identified by the Petroleum Administration for Defense District (PADD) in which they reside. PADDs are shown in Figure VII.A-1, while the number of refineries in each PADD, along with gasoline production and consumption in that PADD, are shown in Table VII.A-1. Although a refinery may be located in a particular PADD, the gasoline produced at that refinery may not necessarily be distributed in that same PADD. For example, most of the gasoline consumed in PADD I is produced by PADD III refineries.

[185] *Petroleum Supply Annual 1998*, Energy Information Administration, June 1999. Table 2.

Figure VII.A-1
Location of PADDs in the Contiguous U.S.

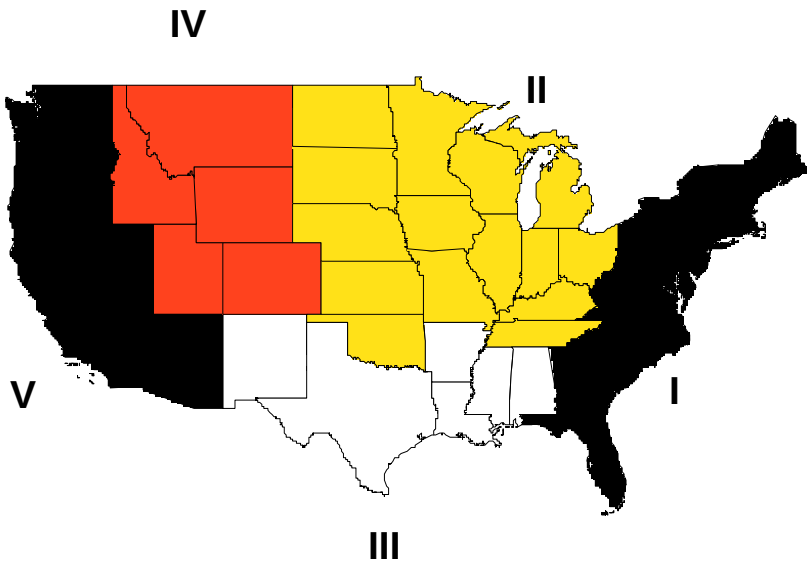


Table VII.A-1
Refinery Count and 1998 Gasoline Volumes by PADD

PADD	Number of operating refineries as of 1/1/2000	Gasoline production (million barrels)[186]	Gasoline consumption (million barrels)[187]
I	14	354	1092
II	30	673	886
III	59	1270	418
IV	15	94	96
V	28	490	521

[186] *Petroleum Supply Annual 1998, Volume 1*, Energy Information Administration, June 1999.

[187] *Petroleum Marketing Annual 1998*, Energy Information Administration, May 1999.

b. Gasoline importers

In 1998, approximately 113 million barrels of gasoline were imported into the U.S. [188] Gasoline is imported into each coast, as well as into Alaska and Hawaii. Gasoline imports by PADD are shown in Table VII.A-2.

Table VII.A-2
1998 Imported Gasoline

	Millions of barrels
PADD I	104
PADD II	1
PADD III	6
PADD IV	0.2
PADD V	2

c. Foreign refiners

Only a few foreign refineries have approved individual anti-dumping baselines. We approved some of these baselines in 1999, and some in 2000. Shipments of gasoline (conventional only) subject to the individual baselines began late in 1999.

2. Toxics emissions variations

a. Differences in 1997-99 data

Our analysis of refiner reports submitted for the years 1997 through 1999 showed little difference in fuel benzene levels between the years, or for either type of gasoline, RFG or conventional. The lack of variation between these years suggests that the results for even one of these years would be representative of all years. Table VII.A-3 shows the results of our analysis.

[188] *Petroleum Supply Annual 1998, Volume 1*, Energy Information Administration, June 1999.

Table VII.A-3
Annual Average Refinery Fuel Benzene Content (vol%)

	1997	1998	1999
RFG	0.64	0.65	0.67
CG	1.12	1.11	1.10

Accurate data on annual average toxics performance was not available for years other than 1998, so year-to-year variations in toxics performance could not be directly evaluated. However, since fuel benzene content has such a large effect on toxics emissions (up to 70 percent), we believe that the values in Table VII.A-3 suggests that toxics performance levels also varied little between 1997 and 1999.

b. Default anti-backsliding baseline

Under our anti-backsliding program, each refinery and importer will be subject to a toxics performance baseline which is based on their annual average production of RFG and CG over the 1998 - 2000 baseline period. Under some conditions, described more fully in the preamble, a refinery or importer will not be able to establish a toxics performance baseline that is specific to their facility. In this case there must be a default baseline available.

We have calculated the default anti-backsliding toxics performance baseline using 1998 - 1999 batch report data submitted to EPA by refiners under the RFG and anti-dumping programs. We are unable at this time to develop a default baseline using data for the full baseline period of 1998 - 2000 because year 2000 production data is not yet available. However, given the yearly consistency exhibited in toxics emissions in Table VII.A-3, we believe that a default baseline which is based on 1998 - 1999 data will be very close to one that is based on 1998 - 2000 data. Once complete year 2000 data becomes available, we will re-evaluate our default baseline and will publish a revised version. .

Though 1998 and 1999 were Phase I RFG years, we have used the Phase II version of the Complex Model to develop our default baseline. This is consistent with the manner in which toxics performance requirements for every refinery and importer will be established, since compliance with the anti-backsliding program will be determined using the Phase II version of the Complex Model. Thus, the 1998-1999 refinery information was evaluated using the Phase II Complex Model, and volume-weighted to determine the values listed in Table VII.A-4 below. We have also added a compliance margin to assure that refiners need go no further than their 1998 - 2000 production in terms of toxics performance levels. Compliance margins are discussed more fully in Section VII.E below.

Table VII.A-4
Toxics Anti-backsliding Default Baseline

Type of gasoline	Reformulated	Conventional
Form of toxic emissions	Total	Exhaust
Units	% reduction form statutory baseline	mg/mile
1998-1999 average value	26.01 % reduction	92.14 mg/mile
Compliance margin	0.7 % reduction	2.5 mg/mile
Default baseline	26.71 %	94.64 mg/mile

3. Refinery Production of Benzene

Though we are finalizing toxics anti-backsliding performance requirement, and not a benzene content requirement as proposed, the impacts of the petrochemical benzene market and the associated refiner actions are still critical to a toxics overcompliance discussion because benzene emissions constitute 70 percent of toxics emissions on a mass basis, and fuel benzene content is the fuel parameter with the largest effect on benzene emissions. Therefore, our discussion of current overcompliance with the current RFG and anti-dumping requirements must begin with a presentation of chemical-grade benzene production at refineries at demand for benzene in the petrochemicals industry.

To better understand the market forces that have lead to the significant fuel benzene content reductions we have seen in the last several years, EPA oversaw a study conducted by ICF Consulting to evaluate the benzene industry[189]. Specifically, the study considered the technical and economic factors associated with benzene production and disposition. The work assignment was designed to provide information on the following:

1) Technologies and economics for benzene reduction at refineries
2) Benzene use in petrochemical complexes
3) Benzene market (production, use transportation, costs)
4) Locations of refineries and petrochemical complexes
5) Technical and economic inputs on benzene reductions in small refineries

[189] "Economic Reasons for U.S. Refineries Overcompliance with Current Benzene Fuel Content in Reformulated and Conventional Gasoline, and Toxic Emissions Performance Standards," ICF Consulting, prepared for U.S. EPA under Contract 68-C-98-170. December, 2000.

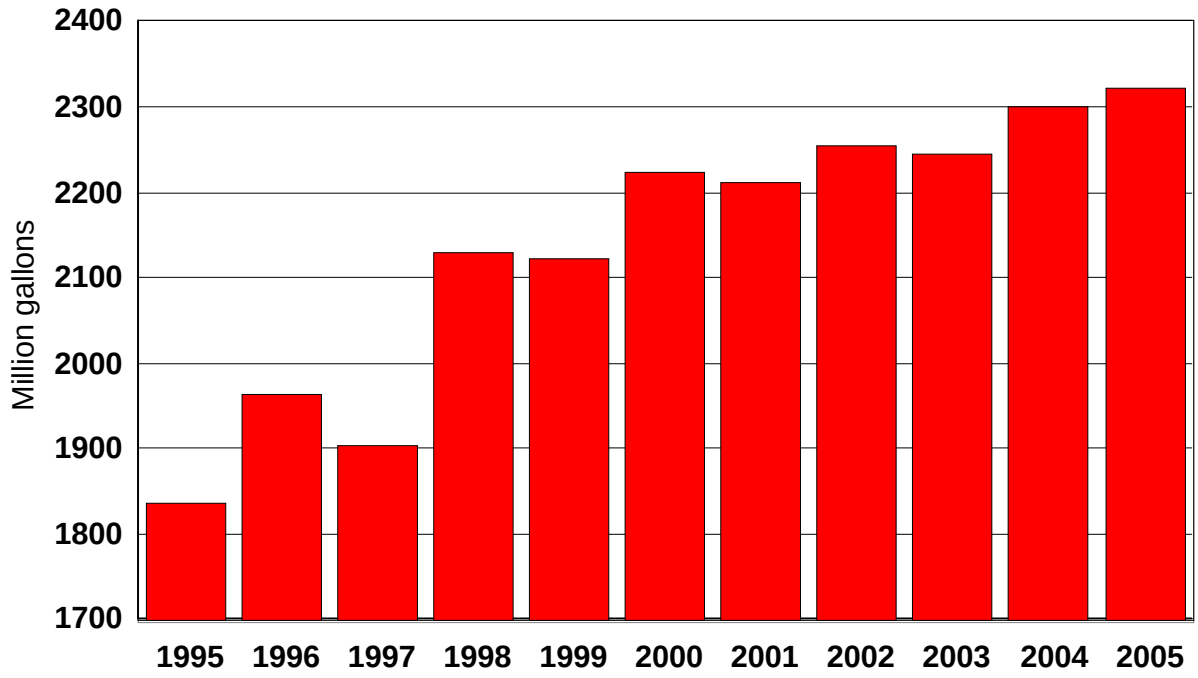
As discussed in the study, the factors that determine whether a refinery sells benzene in the petrochemical market or includes it in its gasoline are based on the relative economic benefits of the petrochemical market for benzene versus the incremental processing costs associated with continuing to allow benzene to be included in gasoline which meets all applicable gasoline composition requirements. The report states that, currently, refineries which extract benzene for the purposes of selling it to the petrochemicals industry do so because it appears more profitable to extract benzene than to include it in gasoline. This situation appears to be motivated largely by regulatory benzene content limits. As stated in the report:

> *The incremental cost to extract more benzene in a refinery is insignificant compared to the base cost to extract benzene down to the RFG limit. Therefore, once the investment in benzene extraction is made, the decision on whether to sell benzene to the petrochemical market is made by comparison of the petrochemical feedstock net-back value to the octane-barrels gasoline blending value.*

Benzene Consumption (Demand) Demand for styrene, which is the primary petrochemical product that uses benzene as a feed, is expected to increase at 4.5 percent annually. World consumption of benzene is expected to increase by 2-4 percent annually. In the U.S., benzene consumption is predicted to increase by about 1-3 percent annually. Future forecasts from both Dewitt & Company, Inc. and Honeywell Hi-Spec Solutions show continued increases for the next several years. See Figure VII.A-2[190]. Also, the U.S. is a net importer of benzene, and no change is expected in this status.

[190] Based on an analysis from Honeywell Hi-Spec Solutions (formerly Bonner & Moore), based on 1999 NPRA Petrochemical Survey for benzene. See EPA Air Docket A-2000-12, "Bonner & Moore Associates U.S. Benzene Supply/Demand."

Figure VII.A-2
Chemical Benzene Demand in the U.S. (Honeywell Hi-Spec Solutions forecast)



Benzene production(supply) The petrochemical industry obtains benzene from a number of sources, depending on company location, transport issues and supply-demand characteristics. These sources include:

1) A refinery owned by the same company as the petrochemical facility
2) Another refining company
3) Benzene extraction units of the petrochemical company
4) Benzene imports

Within a refinery, chemical-grade benzene can be produced from a number of sources. Table VII.A-5 shows a typical refinery benzene supply pattern.

Table VII.A-5
Typical Refinery Benzene Supply Pattern

Refinery Process Unit	Source of Benzene Supply, vol%
Reformer/Straight Run Gasoline	33
Ethylene Unit/Pyrolysis Gasoline	39
TDA/TDP	22
FC Gasoline/Hydrocrackate/Coker Gasoline	6

Benzene Price Benzene price has ranged from $0.70 to $2.25 per gallon since 1981. Currently benzene is selling for about $1.50 per gallon.

Benzene Transportation Benzene can be transported by pipeline, barge, rail and tank truck. Benzene freezes at a relatively high temperature, thus pipeline transport is typically between a refinery and a nearby petrochemical plant. Long distance pipeline transport is precluded because of the high potential for freezing en route. Rail transport is increasingly the travel mode of choice for benzene because of lower cost per volume and because inventories can be reduced due to the availability of nearby rail access. Tank truck is not a favorable mode due to hazardous materials (HAZMAT) restrictions. Barge is favored only if waterway transport is convenient. The ICF report notes that:

> *...margins on benzene sales are not high enough to justify significant transportation costs ....transportation is optimized to minimize cost.*

The report states that economists have predicted that the transportation infrastructure may significantly influence the benzene market in the future, providing an edge to companies with the best transportation "logistics". Thus benzene production is likely to be most favorable in situations where transportation between a production source and the point of consumption is shortest.

B. Toxics inventory and overcompliance

In this Section we present a brief discussion of the toxics emission inventories and the reductions in those inventories that have resulted from overcompliance with the RFG and anti-dumping requirements. We are finalizing a toxics performance requirement which covers the five toxics identified in the Clean Air Act's RFG and anti-dumping provisions. These include benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and polycyclic organic matter (POM).

Chapter IV presented our estimates for toxics inventories for selected years. For toxics compounds which are included in the RFG and anti-dumping programs, these inventories represent current gasoline production including all overcompliance with the RFG and anti-dumping requirements. In order to determine the effect that this overcompliance has had on toxics inventories, it was necessary for us to estimate a hypothetical case in which there was no overcompliance (i.e. RFG and conventional gasoline (CG) exactly meet the applicable toxics emission requirements). To do this, we relied on estimates of gasoline fuel properties and of toxics emissions as predicted by the Complex Model.

The Complex Model was created to permit users to estimate the percent change in emissions that would result from changes in various gasoline fuel properties. The Complex Model also predicts emissions in mg/mi for a given fuel formulation to permit calculation of percent change effects for the sum of all toxics. As a result, it is possible to use the Complex Model to determine how the mg/mi values for total toxics emissions would change if the fuel exactly met the requirements of the RFG and anti-dumping programs. The change in mg/mi values can then be correlated with the inventories presented in Chapter IV to determine the net reduction in toxics inventories that has resulted from overcompliance with the RFG and anti-dumping requirements.

In order to estimate the change in mg/mi emissions as predicted by the Complex Model that results from overcompliance, we must first have fuel property data on actual RFG and CG produced and sold in recent years to establish "baseline" mg/mi levels with the Complex Model. EPA collects batch reports from all refiners to ensure compliance with the RFG and anti-dumping regulations. The most recent database that was available for use in our analysis was for calender year 1998. This database includes volumes and measured properties for all Complex Model inputs. Using this database, we volume-weighted all values to determine the annual average fuel properties for CG and RFG for both summer and winter. The one exception to this was for oxygen content. The dataset for oxygenates used in CG appeared to have contained blendstock and/or RBOB (Reformulated gasoline Blendstock for Oxygenate Blending) data, and was therefore deemed unsuitable. Therefore, we based the CG oxygen levels on the 1996 API/NPRA survey.[191] The 1998 average fuel properties are shown in Table VII.B-1.

[191] "1996 American Petroleum Institute/National Petroleum refiners Association Survey of Refining Operations and Product Quality," Final report, July 1997.

Table VII.B-1
Average fuel properties from 1998 annual reports

	Conventional gasoline		Reformulated gasoline	
	Summer	Winter	Summer	Winter
Oxygen, wt%[a]	0.20	0.20	2.17	2.28
Sulfur, ppm	306	299	203	200
RVP, psi	8.31	12.17	7.69	12.72
E200, vol%	44.65	50.07	48.97	56.07
E300, vol%	80.89	83.22	82.46	84.77
Aromatics, vol%	27.30	24.49	22.58	19.58
Olefins, vol%	11.75	11.51	11.73	11.91
Benzene, vol%	1.13	1.09	0.67	0.63
Volume fraction	0.324	0.386	0.133	0.157

[a] Oxygen was composed of approximately 70% MTBE, 30% ethanol.

The most recent inventory is for 1996. Ideally, then, we would also have annual average fuel properties for 1996. However, 1998 is the most recent year for which we have accurate fuel property data. As shown in Table VII.A-3, we believe that fuel properties did not, on average, vary significantly from year to year. Therefore, we considered the values in Table VII.B-1 to be representative of gasoline produced in 1996. However, it may also be instructive to have a set of representative fuel properties which represent gasoline production in 2007. Therefore we adjusted the values in Table VII.B-1 to take into account the introduction of Phase II RFG in 2000 and low sulfur gasoline in 2004. First, we made use of the presumed fuel properties for summer-grade Phase II RFG developed by the Phase II RFG Implementation Team in 1997 (winter-grade Phase II RFG was expected to be substantially similar to winter-grade Phase I RFG).[192] Second, we changed the sulfur levels of all gasoline to 30 ppm to reflect compliance with the low sulfur requirements. For this analysis we have assumed that corresponding changes in other fuel properties are negligible. Since we are doing a comparative and not an absolute analysis as described more fully below, we do not believe that this assumption substantively affects the results. Note that actual production and/or batch data on gasoline produced in year 2000 was not available at the time of this analysis. The resulting set of representative 2007 fuel properties are given in Table VII.B-2.

[192] "Phase II RFG Report on Performance Testing," April 5, 1999. U.S. EPA, Office of Transportation and Air Quality. See EPA Air Docket A-2000-12.

Table VII.B-2
Representative 2007 Fuel Properties

	Conventional gasoline		Reformulated gasoline	
	Summer	Winter	Summer	Winter
Oxygen, wt%[a]	0.20	0.20	2.17	2.28
Sulfur, ppm	30	30	30	30
RVP, psi	8.31	12.17	6.80	12.72
E200, vol%	44.65	50.07	52.00	56.07
E300, vol%	80.89	83.22	84.00	84.77
Aromatics, vol%	27.30	24.49	22.58	19.58
Olefins, vol%	11.75	11.51	11.50	11.91
Benzene, vol%	1.13	1.09	0.67	0.63
Volume fraction	0.324	0.386	0.133	0.157

[a] For calculation purposes, all oxygen was assumed to be MTBE.

The fuel properties from Tables VII.B-1 and VII.B-2 were then used as inputs into the Complex Model to estimate total toxics emissions in terms of mg/mi. These emission estimates are the per-vehicle equivalents of the inventories presented in Chapter IV, and are shown in Table VII.B-3. Note that the 1996 values were derived from the Phase I version of the Complex Model, which predicts significantly lower emissions than the Phase II version of the Complex Model used to estimate the values for 2007.

Table VII.B-3
Baseline Toxics Emission Estimates From the Complex Model (mg/mi)

	Conventional gasoline		Reformulated gasoline		Weighted average
	Summer	Winter	Summer	Winter	
1996	39.96	51.97	30.71	42.73	43.80
2007	68.67	98.96	55.87	83.78	81.03

In order to determine the effect of overcompliance on toxics emissions, we then estimated the mg/mi emissions that would result if both RFG and CG exactly met their respective standards with respect to toxics. For RFG, this simply entailed applying the toxics standard given in 40 CFR §80.41 to the annual average statutory baseline for toxics to generate a "no overcompliance" estimate of toxics emissions. For CG, we first calculated the average fuel properties for all individual refinery 1990 baselines which are used as the foundation of compliance with anti-dumping, and then calculated the associated mg/mi values with the Complex Model. The results are shown in Table VII.B-4.

Table VII.B-4
Total Toxics Emissions With and Without Overcompliance (mg/mi)

	With Overcompliance	Without Overcompliance	Ratio
1996	43.80	49.77	1.136
2007	81.03	95.84	1.183

Since the mg/mi values in Table VII.B-4 are the per-vehicle equivalents of the inventory estimates given in Chapter IV for gasoline-powered highway vehicles, the fractional increase in mg/mi values due to removing the overcompliance should be the same as the fractional increase in the inventories due to removing overcompliance. In other words, by applying the ratios given in Table VII.B-4 to the inventories in Chapter IV, we can estimate the toxics inventories that would have been produced had there been no overcompliance with the RFG and anti-dumping requirements.

The estimated toxics inventories given in Table IV.B-3 include emissions from both gasoline and diesel highway vehicles. Since the issue of overcompliance is limited to RFG and CG, we used only the estimated tons for gasoline-powered vehicles from Sierra Research, Inc.[193] These values are shown in Table VII.B-5, including our estimate of the effect of current overcompliance on these inventories. Note that the study conducted by Sierra Research, Inc. did not model POM. Therefore, the 1996 POM values in Table VII.B-5 were taken instead from the 1996 National Toxics Inventory, shown in Table IV.A-1. The reduction in the toxics inventories between 1996 and 2007 is due to continuing reductions in the VOC inventory that result from the compounding effects of existing control programs.

[193] "Analysis of the impacts of control programs on motor vehicle toxics emissions and exposure in urban areas and nationwide," U.S. EPA report number EPA420-R-99-030, November 1999

Table VII.B-5
Annual Emissions Inventory for Complex Model Toxics for the Total U.S. Gasoline On-Highway Vehicles Only
(thousand short tons per year)

Compound	1996 Emissions			2007 Emissions		
	Actual	Without over-compliance	Reduction	Actual	Without over-compliance	Reduction
Benzene	168	191	23	87	103	16
Acetaldehyde	21	24	3	9	11	2
Formaldehyde	66	75	9	22	26	4
1,3-Butadiene	22	25	3	10	12	2
POM	0.04	0.05	0.01	0.02	0.02	0.00
Total	277	315	38	128	151	23

Thus we estimate that, as a result of overcompliance with the RFG and anti-dumping requirements, the on-highway toxics inventories are approximately 38,000 and 23,000 tons lower than would otherwise be the case for the 1996 and 2007 inventories, respectively.

We do not have an accurate means for estimating the effects of overcompliance on toxics emissions from nonroad engines. However, since a significant amount of toxics emissions arise from nonroad engines, it nevertheless seems appropriate to calculate a rough estimate of the effects of overcompliance on nonroad toxics inventories. In lieu of an emissions model specific to nonroad engines, we applied the ratios from Table VII.B-4 to the nonroad inventories for gasoline-powered vehicles. The nonroad inventories given in Table VIII.B-1 include both gasoline and diesel sources of toxics emissions, so it was necessary to draw directly from the 1996 NTI study. Values for 2007 were adjusted on the basis of VOC inventories for nonroad in 1996 and 2007, consistent with the approach taken in developing the values in Table VIII.B-1. As before, the absent POM values were taken from Table IV.A-1. The results are shown in Table VII.B-6.

Table VII.B-6
Annual Emissions Inventory for Complex Model Toxics for the Total U.S. Gasoline Nonroad Vehicles Only
(thousand short tons per year)

Compound	1996 Emissions			2007 Emissions		
	Actual	Without over-compliance	Reduction	Actual	Without over-compliance	Reduction
Benzene	176	200	24	70	83	13
Acetaldehyde	13	15	2	5	6	1
Formaldehyde	28	32	4	11	13	2
1,3-Butadiene	20	23	3	8	9	1
POM	0.02	0.02	0.00	0.02	0.02	0.00
Total	237	269	32	94	111	17

Our estimate of the total tons reduced as a result of overcompliance with the RFG and anti-dumping requirements is shown in Table VII.B-7.

Table VII.B-7
Total Tons Reduced Due to Overcompliance (thousand tons)

Compound	1996 Inventory Reductions			2007 Inventory Reductions		
	Highway	Nonroad	Total	Highway	Nonroad	Total
Benzene	23	24	47	16	13	29
Acetaldehyde	3	2	5	2	1	3
Formaldehyde	9	4	13	4	2	6
1,3-Butadiene	3	3	6	2	1	3
POM	0.01	0.00	0.01	0.00	0.00	0.00
Total	38	32	70	23	17	40

Note that the derivation of the ratios in Table VII.B-4 and their application to the toxics inventories was necessarily based on total toxics emissions, not emissions of the individual pollutants benzene, acetaldehyde, formaldehyde, 1,3-butadiene, and POM. This is due to the fact that compliance with the RFG and anti-dumping requirements is done on the basis of the sum of toxics. As a result, we can place confidence only in the total tons reduced as given in Table VII.B-7, not the tons reduced for the individual pollutants. We have provided estimates of tons reduced for individual pollutants only to illustrate the relative reductions in each that we believe are most likely. In fact the distribution of tons reduced for the individual pollutants could be different in reality even though the totals remain the same. The analyses of the refinery modeling has given us a solid indication of what the trends will be for fuel quality in the future as a result of the RFG Phase II program, Tier 2 gasoline sulfur program and the ultra low sulfur diesel program.

C. Costs and Benefits of the Program

1. Technological Feasibility

a. Toxics Performance of RFG Program

The 1990 Clean Air Act Amendments directed EPA to issue regulations that require gasoline to be "reformulated" to significantly reduce vehicle emissions of ozone-forming and toxic air pollutants. This cleaner burning gasoline is called reformulated gasoline (RFG). RFG contains the same components as conventional gasoline, but is blended to reduce air pollution. RFG is required to be used in the ten major metropolitan areas of the U.S. with the worst ozone air pollution problems. Other areas with ozone levels exceeding the public health standard have voluntarily chosen to use RFG. Phase I of the RFG program began in January of 1995 and Phase II began in January 1, 2000. RFG has significantly reduced toxic emissions, including benzene. Analysis of fuel data submitted to EPA by industry for compliance purposes shows that emission reductions from the RFG program have been more than the program requires each year since the program's introduction in 1995. Table VII.C-1 gives a comparison of the average toxics emission reductions standards of RFG Phase I and Phase II, summarised from the standards given in 40 CFR §80.41.

Table VII.C-1
Comparison of Average Emission Reductions Standards of RFG Phase I and RFG Phase II

	RFG Phase I	RFG Phase II
Volatile Organic Compounds	17%	27%
Nitrogen Oxides	2%	7%[a]
Toxics	17%	22%

[a.] Summertime only.

The CAA also requires EPA to establish anti-dumping standards applicable to conventional gasoline used in the rest of the country. The Administrator signed the final RFG and anti-dumping regulations on December 15, 1993[194] and these regulations became effective in January 1995.

b. What is expected to happen with Air Toxics Performance due to RFG Phase II Implementation?

Phase II RFG requirements began on January 1, 2000. However, since the compliance period (the full calender year) has not been completed at the time of this writing, we do not yet have complete information about the means refiners are using to comply with the new requirements. Even so, we have information from refiners' compliance with the Phase I requirements and their own voiced expectations regarding Phase II compliance that lead us to gauge the means for complinace with the Phase II requirements. For instance, refiners are expected meet the more stringent Phase II RFG VOC requirements by lowering the RVP of Phase II RFG. To accomplish a lower RVP in Phase II RFG, one cost-effective option that refiners may use is to blend less butane in Phase II RFG gasoline than Phase I RFG. The reduction in butane will affect the toxics content of gasoline because butane dilutes toxics components such as benzene, aromatics and olefins. In addition, blending less butane in gasoline typically results in lower octane. To make up for the lost octane, refiners may rely on increased reformate, which contains aromatics and benzene.

To check the assumption that fuel benzene properties are likely to increase as a result of compliance with Phase II RFG's more stringent VOC standards, EPA performed a preliminary analysis of second quarter benzene properties for 1998, 1999 and 2000 for those refineries in three PADDs with available 2000 data. This analysis found that, contrary to EPA expectations, that benzene content declined slightly from Phase I to Phase II RFG.[195]

Oxygenates are added to RFG in order to meet the oxygen mandate of 2.0 weight percent oxygen as mandated for RFG by the Clean Air Act. Methyl Tertiary Butyl Ether (MTBE) is the most common oxygenate used in RFG to meet the oxygen mandate. Ethanol is the second most common oxygenate used in RFG to meet the oxygen mandate. MTBE's blending RVP is 8 psi,

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[194] "Regulation of Fuels and Fuel Additives: Standards for Reformulated and Conventional Gasoline - Final Rule," 59 FR 7812 (February 16, 1994). See 40 CFR part 80 subparts D, E, and F.

[195] "Negligible Cost Justification for Total Toxics Anti-Backsliding Program During the Phase II RFG Program," Memo to the Docket (A-2000-12) by Joe Sopata (USEPA), November 17, 2000.

whereas Ethanol's blending RVP is 18 psi.[196] Ethanol's higher blending RVP requires that more light-end hydrocarbons like butane and pentane be removed from the base gasoline blend stock in the manufacturing of RFG, whereas, MTBE's lower blending RVP may only require the removal of some butanes in the manufacturing of RFG.

c. Tier 2 Gasoline Sulfur Program

Recently, EPA issued regulations establishing lower sulfur content requirements for all gasoline[197] and establishing stricter tailpipe emissions standards for all passenger vehicles, including sport utility vehicles (SUVs), minivans, vans and pick-up trucks (the "Tier 2" rule). The low sulfur program will be phased-in beginning in 2004, and, in general, refiners must meet a refinery average sulfur standard of 30 ppm beginning in 2005 and a per gallon cap standard of 80 ppm beginning in 2006 (with the exception of challenged refiners, and gasoline sold in certain western states subject to geographic phase-in). Since less sulfur will be in gasoline as a result of the Tier 2 gasoline sulfur rule, the vehicle emission control system will work more efficiently, thus providing reductions in toxic pollutants.[198] Some refiners will be generating credits early under this program which will result in a decrease in toxic pollutants before 2004.

d. Summary of Refinery Modeling

In the Notice of Proposed Rulemaking, we proposed a benzene content requirement in order to capture the significant amount of overcompliance above and beyond the requirements of the federal reformulated gasoline and anti-dumping programs. The Agency asked for comment on two other forms of the today's rule: benzene emissions performance and toxics emissions performance.

In response to these comments, and based on refinery modeling performed for this rule, the Agency finalized a toxics performance requirement instead of a benzene content requirement. The Agency's general rationale is twofold: a toxics performance requirement captures a larger amount of the overcompliance with the existing standards while imposing less costs on the refining industry than the proposed benzene content requirement.

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[196] U.S. Petroleum Refining, Assuring the Adequacy and Affordability of Cleaner Fuels, National Petroleum Council, Committee on Refining, Lee R. Raymond, Chair, June 2000

[197] "Control of Air Pollution from New Motor Vehicles: Tier 2 Motor Vehicles Emissions Standards and Gasoline Sulfur Control Requirements - Final Rule," 65 FR 6698 (February 10, 2000). See also 40 CFR part 80 subpart H for regulations applicable to gasoline sulfur.

[198] Regulatory Impact Analysis - Control of Air Pollution from New Motor Vehicles: Tier 2 Motor Vehicle Emission Standards and Gasoline Sulfur Control Requirements, EPA420-R-99-023, December 1999.

As described in the preamble, the new toxic emissions performance requirement will require, beginning with calendar year 2002, that a refinery's or importer's annual average total toxics emissions performance for its reformulated gasoline (RFG) not exceed its 1998-2000 baseline RFG total toxics emissions performance. Likewise for conventional gasoline (CG), this rule will require that the exhaust toxics emissions performance of a refinery's or importer's CG not exceed its 1998-2000 baseline exhaust toxics emissions performance for CG. Incremental volumes of either RFG or CG will be excluded from these requirements.

The 1998-2000 baseline RFG or CG toxics emissions performance value is the average performance of the gasoline produced at the refinery (or imported) over the three year period 1998 through 2000. Emission values are determined using the Complex Model[199], and compliance with the program is determined separately for RFG and CG. Though this program does not require new capital investments or changes in refinery operations, and thus should pose no additional burden on refiners, we have included a number of compliance flexibilities, such as deficit and credit carryforward, and a compliance margin, to offset unexpected or unusual variances in the gasoline quality of a refinery (or importer).

The refinery modeling costs analysis is provided below.

Estimated Costs of Compliance with Toxics Emissions Performance Requirement

The Agency oversaw refinery modeling to investigate what impact the RFG Phase II program, the Tier 2 sulfur gasoline program, and the proposed ultra low sulfur diesel fuel program would have on the complex model fuel qualities for both conventional gasoline and reformulated gasoline. We chose 1999 to represent the base case because it is the most recent year to which we could calibrate the refinery model, and because it was the middle year of our finalize baseline period (1998-2000). The modeling then compared the base case with what's known as a "reference case" to account for expected changes in fuel properties due to Phase II RFG, Tier 2 low sulfur gasoline, and 15 ppm low sulfur diesel fuel (based on the proposed heavy-duty 2007 rule). Changes to diesel fuel quality will not directly affect gasoline quality, but the increased hydrogen demand could put pressure to operate the reformer unit harder to provide that needed hydrogen. Operating the reformer harder results in more benzene and other aromatics in the gasoline.

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[199] The Complex Model is a regulatory tool for estimating emissions for the reformulated gasoline and anti-dumping programs. The Complex Model inputs are eight specified fuel parameters: benzene, oxygen content (by oxygenate type), sulfur, Reid Vapor Pressure, aromatics, olefins and the percents evaporated at 200°F and 300°F (E200 and E300). Complex Model outputs are the estimated emissions (VOC, toxics, NOx) resulting from the fuel parameters specified. The Complex Model also calculates percent reductions of the input slate of fuel parameters and resulting emissions compared to a base set of fuel parameters and resulting base emissions.

Table VII-C.2 lists 1999 baseline summer and winter conventional gasoline fuel quality parameters and the volume fractions for PADDs I, II and III. Table VII-C.3 lists 1999 baseline summer and winter RFG fuel quality parameters and the volume fractions for PADDs I, II and III. Table VII-C.4 lists the projected 2005 summer and winter conventional gasoline fuel quality parameters and the volume fractions for PADDs I, II and III. Table VII-C.5 lists the projected 2005 summer and winter RFG fuel quality parameters and the volume fractions for PADDs I, II and III. The projected fuel quality parameters and volume fractions in 2005 account for the implementation of the Phase II RFG program, the Tier 2 sulfur gasoline program and the proposed ultra low sulfur diesel fuel program. Although PADD IV refiners and refiners outside of California in PADD V were not modeled, these results would be expected to carry through for these refiners because the effect of meeting the Tier 2 gasoline sulfur program should be the same in these PADDs as they would be for PADDs I, II, and III.

Table VII.C-2
Baseline (1999) conventional gasoline (summer & winter) fuel quality parameters and volume fraction in PADDs I, II and III.

Complex Model Parameter	Summer CG PADD I	Winter CG PADD I	Summer CG PADD II	Winter CG PADD II	Summer CG PADD III	Winter CG PADD III
MTBE (wt% oxygen)	0	0	0	0	0	0
Ethanol (wt% oxygen)	0	0	0.4	0.4	0	0
Sulfur (ppm)	333	288	175.5	162.9	316	278
RVP (psi)	8.5	12.5	8.0	12.9	7.3	11.7
E200 (%)	40	45	47.1	52.3	40	46
E300 (%)	76	77	81.3	82.2	74	76
Aromatics (vol%)	35.4	30.0	25.9	25.8	30.6	28.4
Olefins (vol%)	18.8	15.5	9.7	9.8	14.2	12.1
Benzene (vol%)	0.90	1.4	1.4	1.3	0.90	1.1
Volume Fraction	0.4913	0.5087	0.5076	0.4924	0.5048	0.4952

Table VII.C-3
Baseline (1999) RFG (summer & winter) fuel quality parameters and volume fraction in PADDs I, II and III.

Complex Model Parameter	Summer RFG PADD I	Winter RFG PADD I	Summer RFG PADD II	Winter RFG PADD II	Summer RFG PADD III	Winter RFG PADD III
MTBE (wt% oxygen)	2.1	2.1	0	0	2.1	2.1
Ethanol (wt% oxygen)	0	0	2.2	2.4	0	0
Sulfur (ppm)	200	164	209.1	244.5	117	282
RVP (psi)	7.9	12.5	7.8	12.9	6.9	11.7
E200 (%)	51	57	43.6	48.4	59	60
E300 (%)	80	83	77.1	80.5	89	88
Aromatics (vol%)	24.4	21.5	32.0	23.6	16.7	13.6
Olefins (vol%)	10.0	8.6	14.6	11.5	1.7	11.8
Benzene (vol%)	0.7	0.8	1.0	1.0	0.5	0.4
Volume Fraction	0.4984	0.5016	0.5092	0.4908	0.5167	0.4833

Table VII.C-4
Projected (2005) conventional gasoline (summer & winter) fuel quality parameters and volume fraction accounting for Phase II RFG, Tier 2 Sulfur Gasoline and Ultra-Low Sulfur Diesel in PADDs I, II and III.

Complex Model Parameter	Summer CG PADD I	Winter CG PADD I	Summer CG PADD II	Winter CG PADD II	Summer CG PADD III	Winter CG PADD III
MTBE (wt% oxygen)	0	0	0	0	0	0
Ethanol (wt% oxygen)	0	0	0.4	0.4	0	0
Sulfur (ppm)	40	29	32.1	26.7	38	25
RVP (psi)	8.5	12.5	8.0	12.9	7.3	11.7
E200 (%)	43	49	42.8	51	40	47
E300 (%)	76	80	76.5	81	74	77
Aromatics (vol%)	32.6	31.3	33.0	28.6	33.7	30.0
Olefins (vol%)	11.4	6.6	6.6	3.8	11.5	6.1
Benzene (vol%)	2.0	1.6	1.6	1.3	1.4	1.1
Volume Fraction	0.4498	0.5502	0.5118	0.4882	0.5048	0.4952

Table VII.C-5
Projected (2005) RFG (summer & winter) fuel quality parameters and volume fraction accounting for Phase II RFG, Tier 2 Sulfur Gasoline and Ultra-Low Sulfur Diesel in PADDs I, II and III.

Complex Model Parameter	Summer RFG PADD I	Winter RFG PADD I	Summer RFG PADD II	Winter RFG PADD II	Summer RFG PADD III	Winter RFG PADD III
MTBE (wt% oxygen)	1.8	2.4	0.52	0.54	2.1	2.1
Ethanol (wt% oxygen)	0	0	1.38	1.77	0	0
Sulfur (ppm)	37	28	40	24.8	32	11
RVP (psi)	6.8	12.5	6.7	12.9	6.7	11.7
E200 (%)	50	58	57.5	56.3	62	62
E300 (%)	80	82	93.4	86.7	94	93
Aromatics (vol%)	24.1	19.3	7.4	17.5	4.8	6.8
Olefins (vol%)	11.2	5.4	2.5	5.4	4.1	3.6
Benzene (vol%)	0.9	0.8	0.4	0.70	0.3	0.4
Volume Fraction	0.5128	0.4872	0.5086	0.4914	0.5167	0.4833

Even though the ultra low-sulfur diesel fuel requirements were not proposed to begin until 2006, we have assumed that they began in 2005 for the purposes of modeling.

In order to determine the impact of the RFG Phase II program, the Tier 2 gasoline sulfur program and the ultra low sulfur diesel program on exhaust and total toxic emissions, the fuel quality parameters listed above were entered into the complex model for the 1999 baseline and the projected year 2005 case separately for conventional gasoline and RFG. Table VII.C-6 lists the results of the analysis for conventional gasoline on a PADD basis for exhaust toxic emissions. Table VII.C-7 lists the results of the analysis for RFG on a PADD basis for total toxic emissions.

Table VII.C-6
Conventional gasoline (CG) exhaust toxic emissions performance for the 1999 base case and projected year 2005 case on a PADD basis

CG Case	PADD I, Exhaust Toxic Emissions (mg/mi)	PADD II, Exhaust Toxic Emissions (mg/mi)	PADD III, Exhaust Toxic Emissions (mg/mi)
Base	100.27	86.70	93.28
Reference	100.23	87.85	88.52

For conventional gasoline (Table VII.C-6), the complex model analysis of predicted fuel properties for PADDs I, II and III demonstrated that toxic emissions are likely to remain at or below 1999 levels despite significant changes necessary to comply with future fuel regulations. The refinery modeling predicted a slight increase in toxic emissions in PADD II due to compliance with future fuel regulations, but that increase is only 1.15 mg/mi, or about 1.3 percent above the 1999 base. The compliance margin established for conventional gasoline is 2.5 mg/mi, and thus the predicted increase is well within the compliance margin. Although PADD IV refiners and refiners outside of California in PADD V were not modeled, these results would be expected to carry through for these refiners because the effect of meeting the Tier 2 gasoline sulfur program should be the same in these PADDs as they would be for PADDs I, II, and III.

The model did not include any limits on the type of desulfurization technology to be used. In implementing the Tier 2 gasoline sulfur requirements, we believe that refiners may choose desulfurization technologies such as Mobil Octgain or Scanfining which has the ability to both recover lost octane and reduce olefins, at the same time that sulfur is being removed from FCC Naptha. This type of technology would actually result in lower exhaust toxic emissions for PADD II than predicted by the model on average. Even though the refinery modeling did not assume these more advanced technologies, based on the refinery modeling and complex model analysis presented here, today's rule is expected to impose only negligible costs on the refining industry.

Table VII.C-7
RFG exhaust toxic emissions performance for the 1999 base case and projected year 2005 case on a PADD basis

RFG Case	PADD I, Total Toxic Emissions (mg/mi)	PADD II, Total Toxic Emissions (mg/mi)	PADD III, Total Toxic Emissions (mg/mi)

Base	75.12	86.74	67.42
Reference	71.89	66.82	60.22

For RFG, the estimated emissions resulting from base and future fuel properties predicted by the refinery modeling show that refineries are likely to be able to meet or exceed the new baseline requirements even after implementing changes to comply with future fuel regulations. Table VII.C-7 lists the results of the analysis for RFG on a PADD basis for total toxic emissions. Based on the refinery modeling and analysis presented here, we expect that the new toxic emissions performance requirement finalized in today's action will impose only negligible costs on the refining industry for RFG and for conventional gasoline.

These fuel quality trends are averaged over PADD I, PADD II and PADD III separately for RFG and conventional gasoline. Because we did not model PADD IV and PADD V and also because we are not able to predict what each individual refiner will do to meet the fuels programs in the year 2005, to help ensure that this toxic emissions anti-backsliding program will be a negligible cost program for refiners we are also implementing several flexibilities for refiners. These flexibilities are a deficit and credit carry forward flexibility, a compliance margin flexibility and an expanded baseline period flexibility.

Estimated Cost of Proposed Benzene Fuel Content Requirement

The Agency oversaw refinery modeling to investigate the costs of the proposed benzene fuel content requirement, where benzene levels were not allowed to above a 1998-99 baseline for an average refinery in PADDs I, II and III. This analysis was similar to the analysis performed for the toxic emissions performance requirement.

Table VII.C-8
Annual Aggregate Cost Increase and Cent per Gallon Cost of a Benzene Anti-backsliding Program for PADDs I, II and III and Nationally

	Annual Aggregate Cost Increase (including amortized capital and all the operating costs) $MM	Cost (Cents/Gallon)
PADD I	0.9	0.01
PADD II	32	0.10
PADD III	41	0.07
Aggregate Costs	74	0.07 (volume-weighted)

Table VII-C.8 lists the annual aggregate cost increase (including amortized capital and all the operating costs), and the cents per gallons cost of a benzene fuel content requirement for PADDs I, II, and III. According to 1999 Energy Information Administration (EIA) data on refinery motor gasoline volumes, PADDs I, II and III represent approximately 91 percent of the gasoline produced in the United States, excluding California gasoline. PADD IV and refiners outside of California in PADD V represent approximately 9 percent of the gasoline produced in the Unites States. National gasoline domestic production in 1999 was estimated by EIA at 99 billion gallons. Since the aggregate costs in Table VII.C-8 represents about 91 percent of the national gasoline supply, if these costs are extrapolated out to the rest of the United States, excluding California, the national annualized aggregate cost increase would be $81 million.

D. Treatment of Incremental Volumes

There are several situations in which a refinery's production of gasoline could increase above the annual average volumes of RFG and CG for that refinery in the 1998 - 2000 baseline period. Incremental volumes are defined here as those volumes of RFG or CG that exceed the volumes produced 0Examples of these situations include entering the RFG market when heretofore the refinery has produced no RFG, shifting the balance from RFG to CG production or vice-versa due to changes in the geographic applicability of the RFG program, or increasing production volumes simply to keep up with growing demand.

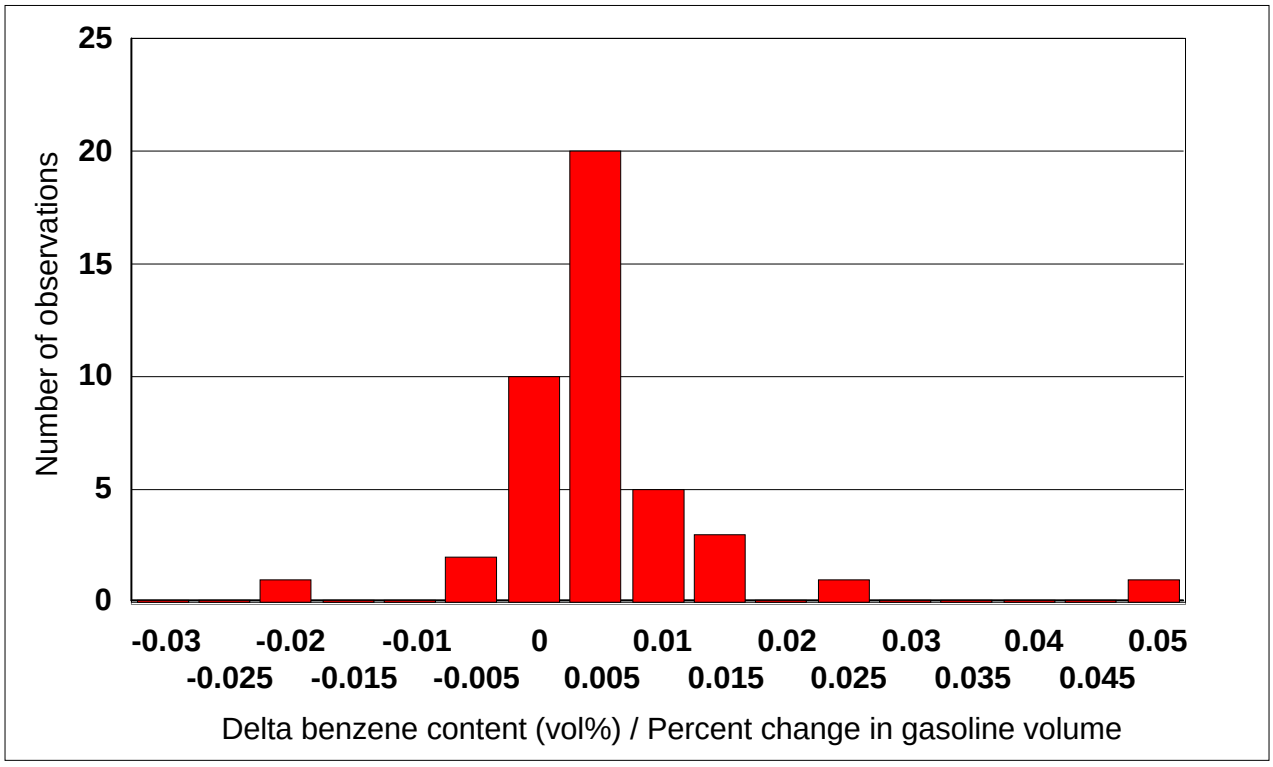
In the NPRM we raised the question of whether the "incremental volumes" of RFG would be likely to contain higher concentrations of benzene than the baseline volumes. Higher benzene concentrations could theoretically arise if refineries producing greater volumes of gasoline did so by converting a larger portion of their crude oil into gasoline instead of simply processing more crude oil. In converting a larger portion of their crude into gasoline, they would be processing that portion of crude oil which tends to be incrementally heavier and more aromatic than the portion of crude oil used to produce gasoline up to their 1998 - 2000 baseline volume. As a result, the benzene concentration of gasoline could increase.

At the time of the NPRM, we did not have any data or modeling that suggested higher benzene concentrations would occur for incremental volumes of RFG. But the potential for higher benzene concentrations in incremental volumes was theoretically great enough to warrant taking comment on the issue. However, the comments we received in response to the NPRM provided essentially no data on this subject, though most of the regulated community did agree with the need to exclude incremental volumes from the anti-backsliding program.

In response to the need for a more quantitative analysis on this subject, we reviewed historical data on RFG production volumes and benzene concentrations to determine if there was a correlation between the two. By-refinery annual average volumes and benzene concentrations were culled from EPA's batch report database for 1997, 1998, and 1999 for RFG. For any case in

which total production volumes increased from 1997 to 1998, or from 1998 to 1999, the ratio of the change in benzene concentration to the percent increase in gasoline volume was calculated. A distribution of these values for can be seen in Figures VII.D-1.

Figure VII.D-1
Effect of Incremental Volumes on Benzene for Reformulated Gasoline



The majority of observations indicate that fuel benzene content did in fact increase when total volume increased, though the reasons (processing more crude, greater use of FCC or reformer units) are unclear. In addition, there was a statistically significant (at the 95 percent confidence level) average increase in benzene concentration when total RFG production volume increased. Although the average increase was small, 0.003 vol% benzene for a one percent increase in gasoline volume, these results do suggest that, at least in the past, increases in RFG production volume were in fact accompanied by increases in benzene content.

We also repeated the analysis for CG. The results are shown in Figure VII.D-2. Even though a majority of the observations were above zero, and there was no statistically significant average effect.

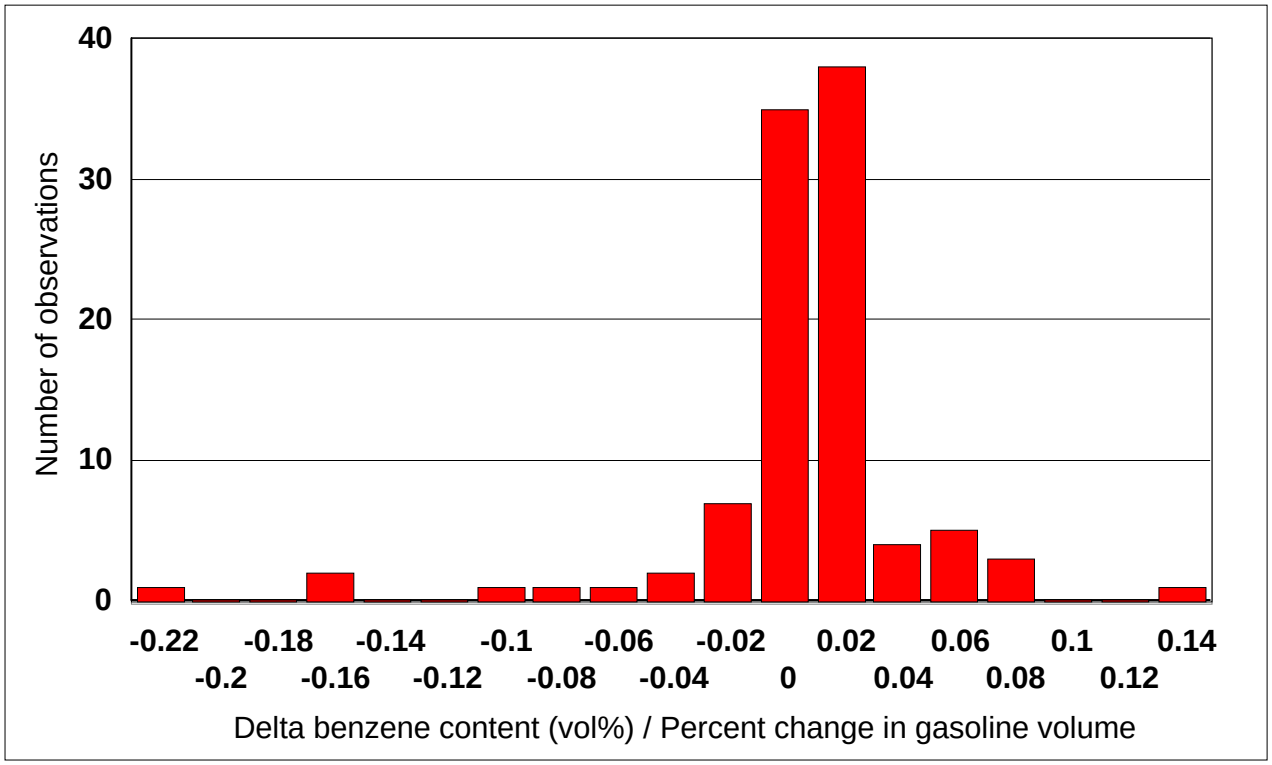


Figure VII.D-2
Effect of Incremental Volumes on Benzene for Conventional Gasoline

Since we are finalizing a toxics performance requirement instead of the benzene content standard we proposed in the NPRM, it seemed prudent to determine how potential increases in RFG benzene content for incremental volumes might impact toxics emissions. To do this we used the RFG fuel properties from Table VII.B-1 as representative of the average refinery baseline, and increased both benzene content and total aromatics content by an amount equal to the average expected increase in RFG benzene content derived above. The increase in benzene content was calculated for a total RFG pool volume increase of approximately 14 percent, which represents the expected growth in RFG demand between the baseline period and 2008. Calender year 2008 was chosen because by this time the Tier 2 low sulfur standard will be phased-in. However, other years could also have served to illustrate how potential increases in benzene content due to increasing pool volumes might affect total toxics emissions. The resulting increase in benzene content was approximately 0.04 vol%. When this benzene content increase was added to the baseline RFG and the results evaluated with the Complex Model, the result was that total toxics emissions in terms of percent change from the statutory baseline increased by 0.5 percent. Given that the compliance margin, as described more fully in Section VII.E below, is only 0.7 percent, it would appear that potential future increases in RFG benzene content which are associated with incremental volumes may in fact erode the cushion that the compliance margin is intended to establish.

Our analysis focused only on fuel benzene content. There is some reason to believe that other changes in fuel properties which are associated with incremental volumes may also contribute to toxics emissions. As described in the preamble, these could include greater volumes of imports from foreign refiners to satisfy growing demand for gasoline, or greater use of existing domestic reformer and FCC units. In addition to increases in aromatic and benzene content, the increased use of these units may also lead to increases in sulfur or olefin content which can also increase toxics emissions. The impact that these incremental volumes could have on fuel properties and, thus, toxics emissions may be just as likely for CG as for RFG, since incremental volumes are a function primarily of growth in demand for gasoline which affects both CG and RFG.

Requiring refiners, however, to meet a more stringent toxics standard for these incremental volumes could require additional capital investment and thereby impose a constraint on incremental gasoline production. As a result, we have decided to exclude incremental volumes from our anti-backsliding program for both RFG and CG.

Exclusion of incremental volumes from the compliance calculations is not straightforward, because the incremental volumes are not specific gallons produced during the last few weeks of the year, but instead arise as increased production volume throughout the year. As a result it is impossible to identify the specific gallons, and their associated fuel properties, that can be labeled as incremental volumes and thus excluded from the compliance calculations. Instead, it was necessary to adjust the compliance calculations in such a way that incremental volumes have no impact on a refinery's compliance with its 1998 - 2000 toxics performance baseline.

In determining compliance, a refinery must compare its annual average toxics performance for any given year to its baseline toxics performance. If the annual average toxics performance exceeds the baseline toxics performance, the refinery is out of compliance (credit carryovers and other flexibilities in our final rule notwithstanding). , Consistent with the approach described above, the baseline must be adjusted so that incremental volumes, as part of a refinery's total annual volume, are compared only to the existing RFG or anti-dumping toxics standards. Using this approach, incremental volumes remain as part of the calculation of annual average toxics performance according to the following equation:

$$T_a = \frac{\sum_{i=1}^{n} (V_i \times T_i)}{\sum_{i=1}^{n} V_i} \qquad (1)$$

Where:

T_a = The refinery or importer annual average toxics value, as applicable. Units are mg/mi for CG and percent reduction from statutory baseline for RFG.

V_i = The volume of applicable gasoline produced or imported in batch i.

T_i	=	The toxics value of batch i. Units are mg/mi for CG and percent reduction from statutory baseline for RFG.
n	=	The total number of batches of applicable gasoline produced or imported during the averaging period.
i	=	Individual batch of gasoline produced or imported during the averaging period.

However, even though incremental volumes remain as part of the calculation of annual average toxics performance, the baseline to which the annual average is compared is adjusted to eliminate the effect of the incremental volumes. To do this we developed a "compliance baseline" which combines a refinery's 1998 - 2000 toxics baseline and the applicable RFG or CG toxics standard:

$$T_{CBase} = \frac{T_{Base} \times V_{Base} + T_{Exist} \times V_{inc}}{V_{Base} + V_{inc}} \qquad (2)$$

where

T_{CBase}	=	Compliance baseline toxics value. Units are mg/mi for CG and percent reduction from statutory baseline for RFG.
T_{Base}	=	Baseline toxics value for the refinery or importer. Units are mg/mi for CG and percent reduction from statutory baseline for RFG.
V_{Base}	=	Baseline volume, equal to the annual average volume of gasoline produced or imported during January 1, 1998 through December 31, 2000.
T_{Exist}	=	Existing toxics standard. For RFG, this value is 21.5 percent. For CG, this value is equal to the refinery's or importer's CG standard in mg/mi.
V_{inc}	=	Volume of gasoline produced over and above V_{Base}

If a refinery's gasoline production for either RFG or CG does not exceed its 1998 - 2000 annual average production, then V_{inc} in equation (2) is zero and the compliance baseline reverts to the refinery's 1998 - 2000 toxics baseline. T_{Base} is a refinery's 1998 - 2000 toxics baseline calculated from:

$$T_{Base} = \frac{\sum_{i=1}^{n} (V_i \times T_i)}{\sum_{i=1}^{n} V_i} + M \qquad (3)$$

Where:

T_{Base}	=	Baseline toxics value. Units are mg/mi for CG and percent reduction from statutory baseline for RFG.
V_i	=	Volume of gasoline batch i produced or imported during January 1, 1998 through December 31, 2000.
T_i	=	Toxics value of gasoline batch i produced or imported during January 1, 1998 through December 31, 2000. Units are mg/mi for CG and percent reduction from statutory baseline for RFG.
n	=	Total number of batches of gasoline produced or imported during January 1, 1998 through December 31, 2000.
i	=	Individual batch of gasoline produced or imported during January 1, 1998 through December 31, 1999.
M	=	Compliance margin, described in Section VII.E below

To determine compliance, a refinery or importer will calculate its annual average toxics performance according to equation (1) for either RFG or CG, and will compare the result to the compliance baseline calculated according to equation (2). Any incremental volumes will, in the course of the comparison, be held only to the existing RFG or CG toxics standards, while all production volumes up to the refinery's baseline volume will be held to that refinery's 1998 - 2000 annual average toxics performance.

E. Compliance Margins

Whenever gasoline is required by regulation to meet a standard for either individual fuel properties or an emission performance standard such as those administered via the Complex Model, the refiner plans its operations in such as way as to provide reasonable assurance that the standard will never be breeched. In practical terms, the refiner aims for some level below the standard so that it can assure compliance even though fuel properties often vary due to imprecise test methods, blending tolerances, commingling and other factors. The difference between the standard and the level that the refiner actually targets to assure compliance is termed the compliance margin.

In today's action we are finalizing an anti-backsliding program which is intended to indefinitely preserve the benefits of the overcompliance exhibited by gasoline in the 1998 - 2000 timeframe. Our program is not intended to produce additional emission benefits. However, if we set the anti-backsliding requirements for toxics emissions at exactly the annual average levels exhibited in 1998 - 2000, refiners would actually target toxics emissions below those 1998 - 2000

levels and would therefore be generating additional emission benefits, potentially at some non-negligible cost. To avoid this, we have determined that it is appropriate to add a compliance margin to the toxics performance requirement applicable to RFG and CG at every refinery.

The overcompliance with the RFG and anti-dumping toxics standards that occurred in the 1998 - 2000 timeframe far exceeded what might be expected from typical compliance margins. Refiners were clearly producing gasoline with lower than necessary toxics emissions for reasons other than simply applying a compliance margin to their applicable standard, as described more fully in Section VII.A.3 above. Therefore, we could not directly assess from historical data the magnitude of an appropriate compliance margin to apply to the toxics performance requirements that will be applicable to individual refineries under our anti-backsliding program. Instead, it was necessary for us to conduct a statistical analysis of the distribution of toxic performance values to estimate the value of an appropriate compliance margin.

Under the RFG and anti-dumping programs, refiners are required to report to EPA the fuel properties and associated toxics emission levels, as estimated by the Complex Model, for every batch of gasoline they produce. To date our most complete and accurate set of batch reports is from 1998. Beginning with this set of over 40,000 batch reports, we separated the data on the basis of whether a refinery produced only CG, only RFG, or produced both CG and RFG. We then randomly selected ten refineries in each of these three categories of refineries and collected all of their applicable 1998 batch reports to produce a selection of refineries which varied in terms of annual gasoline volume, geographic location, and whether it produced CG only or CG and RFG. For each selected refinery we then calculated the compliance margin that would result if the refinery in question intended to meet an anti-backsliding standard equal to the average toxics performance for its 1998 gasoline production. To do this, we used a one-tailed t-test at the 95 percent confidence level. This approach allowed us to determine the buffer that would need to be added to a refinery's 1998 average toxics performance to assure that its future average toxics performance fell below the anti-backsliding requirement. In mathematical terms this calculation was:

$$\text{Compliance margin} = t \cdot \text{STDS} \div \sqrt{\text{OBSERVATIONS}}$$

Where

t	=	Critical value of t for a 95% confidence level
STDS	=	Sample standard deviation
OBSERVATIONS	=	Number of observations (batch reports)

The results of this calculation are given in Table VII.E-1. The CG data represented a total of 1141 batch reports, while the RFG data represented 686 batch reports. The calculated compliance margins could not be correlated with the annual average toxics levels for 1998 - 2000. Therefore, we have calculated the average compliance margins for the refineries we randomly selected for the analysis. The average compliance margins of 2.5 mg/mi for CG and

0.7 percent for RFG will be added to the anti-backsliding baselines approved by the EPA for every refinery and importer in the U.S.

Table VII.E-1
Compliance Margins for Selected Refineries

Refinery	Exhaust toxics for CG (mg/mi)	Total toxics for RFG (% change from statutory baseline)
A	1.76	0.91
B	1.37	n/a
C	2.75	n/a
D	1.04	n/a
E	4.57	n/a
F	n/a	0.79
G	5.53	n/a
H	2.10	0.49
I	0.74	n/a
J	n/a	0.61
Average	2.5	0.7

F. Exclusion of California gasoline

We are not requiring gasoline intended for use in California to be included in a refiner's or importer's compliance determination under the toxics anti-backsliding rule. This action is consistent with other Agency actions on similar fuel issues - California gasoline is exempt from the recently promulgated federal gasoline sulfur requirements [65 FR 6698, February 10, 2000], and while subject to the RFG and anti-dumping provisions, California refineries have been exempted from several of the enforcement and compliance mechanisms of those programs.

Most of the gasoline used in California is produced by California refineries which are subject to the California Cleaner Burning Gasoline (CBG) requirements. The current set of Phase 2 CBG requirements began in 1996 and run through 2002; beginning in 2003, the California Phase 3 gasoline requirements take effect. The sulfur, benzene, aromatics and olefins standards for Phase 2 and Phase 3 are shown in Table VII.F-1. The Flat Limits in Table VII.F-1 are equivalent to per-gallon caps.

Table VII.F-1
Select California Phase 2 and Phase 3 Gasoline Standards

Property	Flat Limit		Averaging Limit		Cap Limit	
	Phase 2	Phase 3	Phase 2	Phase 3	Phase 2	Phase 3
Sulfur (ppm)	40	20	30	15	80	60/30
Benzene (vol%)	1.0	0.8	0.8	0.7	1.2	1.1
Aromatics (vol%)	25.0	25.0	22.0	22.0	30.0	35.0
Olefins (vol%)	6.0	6.0	4.0	4.0	10.0	10.0

In 1998, under the 0.8 vol% Phase 2 benzene standard, California refineries averaged 0.57 vol%. Because of this overcompliance, and the upcoming more stringent Phase 3 standards on all the other fuel parameters, it is likely that toxics emissions under Phase 3 will not be greater than toxics emissions under Phase 2. Thus, we do not expect California refineries, on average, to backslide relative to their 1998-2000 average toxic emission level. Additionally, given the compliance margin we are including in today's rule, it is highly unlikely that any backsliding would exceed the combination of the actual 1998-2000 baseline plus the compliance margin.

Chapter 8: Nonroad Mobile Source Air Toxics

In this chapter we first describe our current nonroad engine emission control programs and then present our estimates of the impacts of these programs on future air toxics inventories. We are looking at nonroad MSATs emissions separately from motor vehicle emissions of MSATs primarily because our understanding of nonroad MSAT emissions is much more limited than that of motor vehicle MSAT emissions. Therefore, this chapter ends with a discussion of the significant uncertainty and data gaps that exist with respect to toxics emissions from nonroad engines. We will need to fill these gaps in our data before we can assess the need for, and appropriateness of, programs intended to further reduce nonroad MSATs. Under our Technical Analysis Plan, described in Section VII of the preamble for this rule, we will seek to gather this information by 2003. We intend to evaluate potential strategies relating to hazardous air pollutants from nonroad engines and vehicles.

A. Overview of Current Nonroad Engine Emission Control Programs

The 1990 Clean Air Act Amendments specifically directed us to study the contribution of nonroad engines to air pollution which may reasonably be anticipated to endanger public health or welfare, and regulate them if warranted. "Nonroad" is a term that covers a diverse collection of engines, equipment, and vehicles. Also referred to as "off-road" or "off-highway," the nonroad category includes outdoor power equipment, recreational equipment, farm equipment, construction equipment, lawn and garden equipment, and marine vessels. Though dealt with separately in the Clean Air Act, locomotives and aircraft can also be considered categories of nonroad engines. Except for aircraft, we did not regulate emissions from nonroad engines prior to the mid-1990s.

In 1991, we released a study documenting emission levels across a broad spectrum of nonroad equipment that were higher than expected.[200] The study showed that emissions from nonroad engines are a significant source of oxides of nitrogen (NOx), volatile organic compound (VOC), and particulate matter (PM) emissions. In some areas of the country, emissions from nonroad engines represent a third of the total mobile source NOx and VOC inventory and over two-thirds of the mobile source PM inventory. Based on the results of this study, referred to as NEVES, we determined that emissions of NOx, HC,[201] and CO from nonroad engines and equipment contribute significantly to ozone and CO concentrations in more than one nonattainment area.[202] Thus, we initiated regulatory programs for several categories of nonroad engines as required by section 213(a)(3) of the Clean Air Act.

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[200] "Nonroad Engine and Vehicle Study - Report and Appendices," EPA-21A-201, November 1991 (available in Air Docket A-96-40).

[201] HC stands for hydrocarbon. HC and VOC are very similar and are generally used interchangeably.

[202] 59 FR 31306, June 17, 1994.

In addition to the determination of significance for NOx, HC, and CO emissions just discussed, we made a determination, under section 213(a)(4) of the Clean Air Act, that smoke and PM emissions from nonroad engines and equipment significantly contribute to air pollution that may be reasonably anticipated to endanger public health or welfare.[203] Under this determination we are authorized to establish smoke and PM emission standards for nonroad engines and equipment. While we have established smoke and PM regulations for many categories of nonroad engines and equipment, our efforts to date have been more focused on achieving NOx reductions from diesel nonroad engines, and HC and CO reductions from gasoline nonroad engines.

The broad category of nonroad equipment encompasses a large variety of equipment types, from hand-held lawn and garden equipment to locomotives and large marine vessels. The engines used in nonroad equipment also vary dramatically, from very small two-stroke spark-ignited (SI) gasoline engines to very large two-, and four-stroke diesel engines. Many of these engines are designed and manufactured specifically for their nonroad applications. Others are adaptations of on-highway engine designs, or even other nonroad engines. For example, most land-based nonroad diesel engines are based on on-highway engines, with modifications as necessary for nonroad application. Likewise, most small and medium size diesel marine engines are modified land-based nonroad engines.

Even though many nonroad engines are derived from on-highway engines, the technologies applied to on-highway engines to reduce emissions are often not readily transferrable to nonroad engines, or are transferrable to different nonroad applications in different degrees. The physical limitations of nonroad equipment, as well as different operating environments and duty cycles, sometimes limit the application of on-highway emission reduction technologies. For example, charge air cooling is widely used as a NOx reduction technique for large diesel truck engines. With the aftercooler mounted on the front of the truck, the ram air available as a truck travels down the road can afford a large degree of cooling. However, with land-based nonroad equipment the available cooling tends to be significantly lower, both because of the physical limitations of the equipment in terms of mounting the aftercooler, as well as the typically slower speeds at which such equipment tends to operate. Conversely, there is a large amount of cooling available in marine applications through the use of the surrounding water to cool the charge air.

Due to the variety of nonroad engine and equipment types and sizes, combustion processes, uses, and potential for emissions reductions, we placed nonroad engines into several categories for regulatory purposes. These categories include land-based diesel engines (e.g., farm and construction equipment), small land-based spark-ignition (SI) engines (e.g., lawn and garden equipment), large land-based SI engines (e.g., forklifts, airport ground service equipment), marine engines (including diesel and SI, propulsion and auxiliary, commercial and recreational),

[203] 59 FR 31306, June 17, 1994.

locomotives, aircraft, and recreational vehicles (e.g., large land-based SI engines used in off-road motorcycles, all-terrain vehicles and snowmobiles). Summaries of our current or anticipated programs for these nonroad categories follow. The information presented for these programs is, in many cases, taken directly from the preambles and supporting documents of the final rules. Following the discussion of the specific nonroad control programs, we present a general overview of the nonroad fuels issue.

1. Land-Based Nonroad Diesel Engines

Nonroad diesel (also referred to as compression-ignition) engines dominate the large nonroad engine market and comprise approximately 25 percent of the current mobile source NOx emissions inventory and 40 percent of the current mobile source PM emissions inventory. Examples of applications falling into this category include agricultural equipment such as tractors, construction equipment such as backhoes, material handling equipment such as heavy forklifts, and utility equipment such as generators and pumps.

Under our regulations, diesel engines greater than 50 horsepower (hp) must comply with Tier 1 emissions standards that are being phased in between 1996 and 2000, depending on the size of the engine.[204] Under the Tier 1 standards, we project that NOx emissions from new diesel nonroad equipment will be reduced by over 30 percent from uncontrolled levels. The Tier 1 standards do not apply to engines used in underground mining equipment, locomotives, and marine vessels.[205]

In August 1998, we adopted more stringent emission standards for NOx, HC, and PM for new nonroad diesel engines, to be phased in over several years beginning in 1999.[206] Engines used in underground mining equipment, locomotives, and marine engines over 50 hp are not included. This comprehensive new program includes the first set of standards for nonroad diesel engines less than 50 hp. Standards for these small engines will be phased in from 1999 to 2000. The rule also phases in more stringent Tier 2 standards for all engine sizes from 2001 to 2006, and yet more stringent Tier 3 standards for engines over 50 hp from 2006 to 2008. Finally, the new program includes a voluntary program to encourage the production of advanced, very-low emitting engines. Under these new standards, we project that emissions from new nonroad diesel equipment will be further reduced by 60 percent for NOx and 40 percent for PM compared to the emission levels of engines meeting the Tier 1 standards. We are currently working on the Nonroad Tier 3 technology review for 2001. We intend to consider the control of sulfur in nonroad diesel fuel as part of our Tier 3 technology review. This could potentially allow more

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[204] 59 FR 31306, June 17, 1994.

[205] The Mine Safety and Health Administration is responsible for setting requirements for underground mining equipment. Locomotives and marine vessels are covered by separate EPA programs.

[206] 63 FR 56968, October 23, 1998.

effective diesel PM control technologies such as catalysts to be applied to nonroad engines and vehicles.

2. Small Land-Based SI Engines

Small spark-ignition (SI) engines (e.g., engines operating on gasoline, natural gas, propane, or methanol) at or below 25 hp comprise about 9 percent of the mobile source VOC inventory. These small engines are used primarily in lawn and garden equipment, such as lawn-mowers, string trimmers, edgers, chain saws, commercial turf equipment, and lawn and garden tractors.

Under Phase 1 of our nonroad small SI engine regulations, new small SI engines must comply with emission standards for HC, CO, and NOx beginning in 1997. [207] The Phase 1 standards apply to all SI engines at or below 25 hp, except for those used in aircraft, marine vessels, and recreational equipment. We expect that these Phase 1 standards will result in a 32 percent reduction in HC emissions from small SI engines (approximately 340,000 tons from uncontrolled levels).

We finalized Phase 2 nonroad small SI engine regulations in March 1999 for nonhandheld engines, and in March 2000 for handheld engines.[208] The Phase 2 programs include more stringent emission levels and new provisions to ensure low in-use emissions. We expect the Phase 2 program for nonhandheld engines, when fully phased in, to achieve approximately 350,000 tons of HC + NOx emission reductions, and the program for handheld engines to achieve approximately 450,00 tons of reduction in HC + NOx emissions. These reductions represent reductions in HC+NOX beyond the Phase 1 levels of 60 percent for nonhandheld engines and 70 percent for handheld engines.

3. Large Land-Based Spark-Ignition Engines

We do not currently have emission standards in place for spark-ignition engines above 25 hp used in commercial applications. These engines are used in a variety of industrial equipment, including forklifts, airport ground-service equipment, generators, and compressors. We are currently developing an emission control program for these engines (65 FR 76797, December 7, 2000).

4. Marine Engines

Like land-based nonroad engines, marine engines serve a wide variety of applications. The smallest marine engines, virtually all of which use gasoline, are used in recreational

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[207] 60 FR 34582, July 3, 1995.

[208] 64 FR 15208, March 30, 1999 and 65 FR 24267, April 25, 2000.

outboards and personal watercraft. Small gasoline or diesel marine engines provide auxiliary power on many vessels. Larger marine engines provide propulsion for both recreational and commercial applications. Recreational sterndrive and inboard engines tend to be gasoline, though diesel engines are making inroads into that market. Commercial engines, virtually all diesel, power vessels such as tugs, ferries, and crew/supply boats. These engines also provide auxiliary power on larger vessels. The largest marine diesel engines, sometimes exceeding 60,000 hp, propel ocean-going vessels. We group engines under three control programs reflecting their application and, to some extent, the fuel they use.

a. Gasoline Outboards and Personal Watercraft Marine Engines

Gasoline outboards and personal watercraft contribute about 5 percent of the national mobile source VOC inventory. However, in areas with large boat populations, the contribution of these recreational marine engines may exceed 10 percent of the regional mobile source VOC inventory. These engines typically employ 2-stroke technology, which has changed very little over the last 50 years. Regulations to control exhaust emissions from new outboards and personal watercraft went into effect beginning with the 1998 model year.[209] The emission controls for these engines involve increasingly stringent standards over the course of a nine-year phase-in period beginning in model year 1998. By the end of the phase-in, each manufacturer must meet an emission standard, on a corporate-average basis, that represents a 75-percent reduction (on the order of 500,000 tons) in HC compared to unregulated levels. The gradually decreasing emission standard allows manufacturers to determine the best approach to achieving the targeted reductions over time. Manufacturers are able to phase in the types of control technologies in the most sensible way, while minimizing the cost impact to the consumer.

b. Commercial Diesel Marine Engines

Commercial diesel marine engines contribute about 8 percent of the national mobile source NOx inventory, and about 1 percent of the national mobile source PM inventory. In areas with large commercial ports or near busy shipping lanes, the contribution of diesel marine engines to the local mobile source NOx and PM inventory may be much higher. We published regulations for the control of exhaust emissions from new marine diesel engines in December 1999.[210] The emission limits, which vary depending on the size of the engine, are similar to emission limits for corresponding land-based nonroad or locomotive engines. These limits apply beginning with engines manufactured in 2004, and will result in 13-percent VOC and 26-percent diesel PM reductions from uncontrolled levels. The emission limits for very large commercial marine diesel engines are the same as those contained in Annex VI of the International Convention on the Prevention of Pollution from Ships (MARPOL). Consistent with MARPOL Annex VI, these limits will apply to engines installed on ships constructed on or after January 1, 2000.

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[209] 61 FR 52088, October 4, 1996.

[210] 64 FR 73300, December 29, 1999.

c. Recreational Sterndrive and Inboard Engines

Recreational sterndrive and inboard engines can be either gasoline or diesel engines. While their contribution to national mobile VOC and NOx levels is smaller than the other two marine engine categories, their emissions are expected to increase due to the growing number of recreational vessels. We did not finalize emission limits for gasoline sterndrive and inboard engines as part of the 1996 marine rule. Likewise, we did not propose limits for recreational diesel engines in the commercial diesel engine rule. We do not currently have emission regulations in place for this category of marine engine, but have begun developing them (65 FR 76797, December 7, 2000).

5. Locomotives

Locomotives are estimated to contribute about 9 percent of the nationwide mobile source NOx emissions inventory. These engines are generally larger and last longer than any land-based nonroad diesel engines. In April 1998, we published emission standards for NOx, HC, CO, PM, and smoke for locomotives.[211] The new standards are ultimately expected to reduce NOx emissions by two-thirds, while HC and PM emissions from these engines will be decreased by 50 percent.

A unique feature of the locomotive program is that it includes emission standards for remanufactured engines, including all those that were originally built since 1973.[212] Regulation of the remanufacturing process is critical because locomotives are generally remanufactured 5 to 10 times during their total service lives, which is typically 40 years or more.

Three separate sets of emission standards have been adopted, with applicability of the standards dependent on the date a locomotive is first manufactured. The first set of standards (Tier 0) applies to locomotives and locomotive engines originally manufactured from 1973 through 2001, any time they are manufactured or remanufactured. The second set of standards (Tier 1) applies to locomotives and locomotive engines originally manufactured from 2002 through 2004. These locomotives and locomotive engines will be required to meet the Tier 1 standards at the time of original manufacture and at each subsequent remanufacture. The final set of standards (Tier 2) applies to locomotives and locomotive engines originally manufactured in 2005 and later. Tier 2 locomotives and locomotive engines will be required to meet the applicable standards at the time of original manufacture and at each subsequent remanufacture. Electric locomotives, historic steam-powered locomotives, and locomotives originally manufactured before 1973 do not contribute significantly to the emissions problem and, thus, are not subject to the locomotive regulations.

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[211] 63 FR 18978, April 16, 1998.

[212] Locomotives are typically overhauled to "as new" condition every four to eight years in a process known as remanufacturing.

While the Tier 0 and Tier 1 regulations are primarily intended to reduce NOx emissions, the Tier 2 regulations will result in 50 percent reductions in VOC and diesel PM from unregulated levels, as well as additional NOx reductions beyond the Tier 0 and Tier 1 regulations. As a result, almost half of the NOx reductions we ultimately expect will be achieved by 2005. In contrast, the VOC and diesel PM reductions are achieved more slowly, due to the very slow fleet turnover. By 2040 we expect VOC reductions of about 18,000 tons per year and diesel PM reductions of about 12,000 tons per year. About one third of these VOC and diesel PM reductions will be realized by 2010.

6. Aircraft

Aircraft emissions comprise less than 2 percent of the mobile source NOx emissions inventory, but they are significant contributors to the NOx inventory in some cities. In addition, commercial aircraft emissions are a fast growing segment of the transportation emissions inventory. Aircraft emissions are potentially important contributors to global climate change and may also contribute to the depletion of the stratospheric ozone layer.

Emission standards for gas turbine engines that power civil aircraft have been in place for about 20 years. Such engines are used in virtually all commercial aircraft, including both passenger and freight airlines. The standards do not apply to military or general aviation aircraft. Controls on engine smoke and prohibitions on fuel venting were instituted in 1974 and have been revised several times since then. Beginning in 1984, limits were placed on the amount of unburned HC gas turbine engines can emit per landing and takeoff cycle.

In April 1997, we adopted the existing International Civil Aviation Organization (ICAO) NOx and CO emission standards for gas turbine engines.[213] ICAO, a specialized agency of the United Nations, is the most appropriate forum for first establishing commercial aircraft engine emission standards due to the international nature of the aviation industry.

None of the actions just discussed have resulted in significant emissions reductions, but rather have largely served to prevent increases in aircraft emissions. We are also exploring other ways to reduce the environmental effects associated with air travel throughout the nation. We are working with the Federal Aviation Administration (FAA) to encourage continuing progress in reducing emissions from airport ground service equipment and aircraft auxiliary power units. We sponsored compilation of technical data and emission inventory methods, which the FAA will use to develop an Advisory Circular for airlines and airport authorities interested in reducing emissions from these sources.

Some municipalities are taking an interest in toxic air pollution from airports and are

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[213] International Civil Aviation Organization (ICAO) Annex 16, Volume II, Environmental Protection, Aircraft Engine Emissions.

undertaking their own studies. An example is a study being undertaken by the City of Park Ridge, Illinois concerning O'Hare International Airport.[214]

7. Recreational Vehicles

We do not have standards in place for large land-based SI engines used in recreational vehicles, such as off-road motorcycles, all-terrain vehicles, and snowmobiles. However, we are currently developing emission regulations for recreational vehicles. As part of this regulatory process, we intend to consider toxics emissions from these vehicles (65 FR 76797, December 7, 2000).

8. Fuels

In addition to the above engine technology-based emission control programs, fuel controls will also reduce emissions of air toxics from nonroad engines. For example, gasoline formulation (the removal of lead, limits on gasoline volatility and reformulated gasoline) will reduce nonroad MSATs because most gasoline-fueled nonroad vehicles are fueled with the same gasoline used in on-highway motor vehicles. An exception to this is lead in aviation gasoline. Aviation gasoline is a high octane fuel used in a relatively small number of aircraft (those with piston engines). Such aircraft are generally used for personal transportation, sightseeing, crop dusting, and similar activities.

As just discussed, most of our fuel controls aimed at gasoline cover both on-highway and nonroad vehicle fuel. The same is not true for diesel fuel. We have regulations in place which will dramatically reduce the sulfur levels in on-highway diesel fuel. These controls do not apply to nonroad diesel fuel, and prior to these controls there was no distinction between on-highway and nonroad diesel fuel. We intend to consider the control of sulfur in nonroad diesel fuel as part of our Tier 3 technology review. This could potentially allow more effective diesel PM control technologies such as catalysts to be applied to nonroad engines and vehicles.

B. Impacts of Nonroad Control Programs on Air Toxics

As a whole, our nonroad programs significantly reduce the impact of nonroad equipment on the nation's air quality. As with motor vehicle controls, while we've focused our controls on achieving reductions in criteria pollutants (NOx, HC, and PM), our control programs have also been effective in reducing emissions of air toxics.

As is the case with motor vehicle emissions, we expect nonroad emissions of gaseous toxics to decrease over the next 20 years under our current control programs. By 2020, we

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[214] "Preliminary Study and Analysis of Toxic Air Pollutant Emissions from O'Hare International Airport and the Resulting Health Risks Created by these Toxic Emissions in Surrounding Residential Communities," City of Park Ridge, IL, August, 2000.

estimate that benzene emissions will decrease by 31 percent (over 31,000 tons) and formaldehyde emissions will decrease by 49 percent (over 38,000 tons), as compared with 1990 levels. However, nonroad emissions of diesel PM are not decreasing dramatically. We estimate that by 2020, nonroad engines will emit more that 310,000 tons per year of diesel PM emissions, as compared with 346,000 tons in 1996, a 10-percent decrease. Our land-based nonroad Tier 3 technology review will examine nonroad engine diesel PM emissions. Although we are not required to address nonroad MSATs under §202(l) of the Act, as part of our Technical Analysis Plan, described in the preamble to the regulation, we also intend to evaluate potential strategies relating to hazardous air pollutants from nonroad engines and vehicles.

1. Nonroad MSAT Baseline Inventories

We previously presented the 1996 baseline inventories for several key nonroad MSATs in Table IV.A-1. This nonroad MSAT data was taken from the 1996 National Toxics Inventory (NTI). In general, the data shows that nonroad vehicles tend to be significant contributors of those MSATs for which motor vehicles are also significant contributors. Nonroad vehicles contribute as much as 39 percent of the national inventory of some MSATs, such as acetaldehyde and MTBE, and contribute significantly to the national inventories of several others, including 1,3-butadiene, acrolein, benzene, formaldehyde, lead compounds, n-hexane, toluene and xylene.

2. Emission Reductions from Current Programs

The programs summarized in Section A of this chapter are expected to result in reductions of national inventories of the MSATs. This section summarizes our estimates of nonroad MSAT inventories into the future, based on the nonroad emission control programs we currently have in place. The discussion in this section consists of three parts. First, we discuss the inventories of four MSATs: benzene, formaldehyde, acetaldehyde and 1,3-butadiene. Second, we discuss nonroad VOC emissions inventories as a surrogate for the other nonroad gaseous MSATs. Finally, we discuss the trend of nonroad diesel PM emissions. We focused on these pollutants for nonroad mobile sources primarily to allow comparisons with the on-highway analyses presented in earlier chapters. The inventories presented here are based only on regulations that we have completed. As previously discussed, we are developing the first national regulations applicable to recreational vehicles and recreational marine sterndrive and inboard engines (65 FR 76797, December 7, 2000). We are also conducting a technology review through regulation for land-based nonroad diesel engines. As part of this review, we intend to consider whether additional VOC (and, thus, gaseous MSAT) reductions would be appropriate. We intend to consider the control of sulfur in nonroad diesel fuel as part of our Tier 3 technology review. This could potentially allow more effective diesel PM control technologies such as catalysts to be applied to nonroad engines and vehicles.

We are not reporting inventory trends for the metals on our list of MSATs (arsenic compounds, chromium compounds, mercury compounds, nickel compounds, manganese compounds, and lead compounds) or for dioxin/furans. Metals in mobile source exhaust can

come from fuel, fuel additives, engine oil, engine oil additives, or engine wear. Formation of dioxin and furans requires a source of chlorine. Thus, while metal emissions and dioxins/furans emissions are associated with particles and it is possible that these compounds track PM emissions to some extent, there are a number of other factors that contribute to emission levels and we do not have good data on these relationships.

a. Benzene, Acetaldehyde, Formaldehyde, and 1,3-Butadiene

Table VIII.B-1 shows our estimates of the nonroad emissions of these four gaseous MSATs . These estimates were based on the 1996 inventories contained in the 1996 NTI study. The 1990 estimates were derived by applying a ratio of nationwide 1990 to 1996 VOC inventories from the draft NONROAD model to the 1996 NTI numbers.[215] Toxic fractions represent the fraction of total VOC that a given MSAT makes up. The toxic fractions were derived from speciated emissions data on different engines and come from a variety of studies which are discussed in Chapter 2. By knowing the total VOC inventory and the toxic fraction for a given MSAT, we can estimate the inventory of that specific MSAT indirectly. The 2007 and 2020 estimates were also derived from the draft NONROAD model, with the toxic fractions applied to the VOC results. The draft NONROAD model does not include locomotives, commercial diesel marine engines, or aircraft. We do not have enough information to estimate the inventories of the four gaseous MSATs for these three nonroad vehicle categories. Thus, they are not included in the 1990, 2007 and 2020 estimates. For consistency's sake, we have excluded these categories from the 1996 NTI numbers as well. Thus, the 1996 estimates shown here differ slightly from those shown in Table IV.A-2. However, these three nonroad categories only represent about three percent of the total nonroad VOC. Using VOC as a surrogate for gaseous toxics, as discussed in the next section, we conclude that the exclusion of locomotives, commercial diesel marine engines, and aircraft from our estimates of gaseous MSATs does not have a significant impact on those estimates.

[215] The draft NONROAD model is a model we are developing which is used to project emissions inventories from nonroad mobile sources. Because this is a draft model and subject to future revisions, the inventories derived from the draft NONROAD model and presented here are subject to change.

Table VIII.B-1
Annual Emissions for Benzene, Acetaldehyde, Formaldehyde, and 1,3-Butadiene from Nonroad Sources
(thousand short tons per year)

Compound	1990 Emissions	1996 Emissions	2007 Emissions	2020 Emissions
Benzene	100.2	98.7	75.4	69
Acetaldehyde	37.7	40.8	26.3	20
Formaldehyde	79.2	86.4	53.8	40.7
1,3-Butadiene	9.4	9.9	8.8	7.8

Table VIII.B-2 summarizes the percent reductions in 2007 and 2020 from 1990 and 1996 levels represented by the inventories in Table VIII.B-1. This table shows that the reductions expected from our existing nonroad control programs are significant, although not as substantial as the reductions of these pollutants for on-highway motor vehicles presented in Chapter 4.

Table VIII.B-2
Emission Reductions for Benzene, Acetaldehyde, Formaldehyde, and 1,3-Butadiene from Nonroad Sources

	Reduction in 2007		Reduction in 2020	
Compound	From 1990	From 1996	From 1990	From 1996
Benzene	25%	24%	31%	30%
Acetaldehyde	30%	36%	47%	51%
Formaldehyde	32%	38%	49%	53%
1,3-Butadiene	7%	11%	18%	21%

b. VOCs

With the exception of the four MSATs shown in Table VIII.B-1, we cannot estimate emissions from nonroad mobile sources for the other gaseous MSATs because we do not have toxic fraction information for the other gaseous MSAT emissions. Therefore, to estimate projected inventory impacts from our current nonroad mobile source emission control programs, we use VOC inventories. We believe this is appropriate because the gaseous MSATs are constituents of total VOC emissions. By using VOC emissions as a surrogate, we are assuming that MSAT emissions track VOC reductions. In reality, however, as can be seen from Table VIII.B-2, some gaseous MSATs may not decrease at the same rate as VOCs overall. Without

having more detailed emission data for each of the MSATs, however, we are unable to offer any insights on how those rates may differ. This is one of the issues we intend to address as part of the Technical Analysis Plan described in the preamble to the regulation.

Our VOC emission inventories were developed using the draft NONROAD model. Because the draft NONROAD model does not include locomotives, commercial marine diesel engines, or aircraft we supplemented the draft NONROAD model inventories with locomotive and diesel marine inventories developed in support of our regulations for those categories, and with aircraft emission inventories from the National Air Pollutant Emissions Trends, 1900-1996 report.[216] The results of this analysis shows that VOC inventories are projected to decrease approximately 44 percent between 1996 and 2020 due to existing nonroad mobile source emission control programs. This analysis, however, shows that our existing nonroad emission control programs will nonetheless result in significant gaseous MSAT reductions (assuming, as previously discussed, that gaseous MSATs emissions track VOC reductions).

Table VIII.B-3
Annual VOC Emissions
From Nonroad Sources

Year	1996	2007	2020
Million short tons per year	3.6	2.2	2.0
Cumulative Percent Reduction	***	39%	44%

c. Diesel PM

We estimated the nonroad diesel PM inventories using the draft NONROAD model. We are using diesel PM as a surrogate for diesel PM and diesel exhaust organic gases. As noted above, because the draft NONROAD model does not include locomotives, commercial marine diesel engines, or aircraft we supplemented the draft NONROAD model inventories with locomotive and diesel marine inventories developed in support of our regulations for those categories, and with aircraft emission inventories from the National Air Pollutant Emissions Trends, 1900-1996 report. Table VIII.B-4 shows our estimates of nonroad diesel PM emissions inventories. As can be seen, we expect nonroad diesel PM emissions to begin to drop with the implementation of some of our nonroad regulations. However, in the absence of additional controls, we expect that nonroad diesel PM emission inventories will begin to increase due to expected growth in the populations of nonroad vehicles and equipment.

[216] "National Air Pollutant Emission Trends, 1900-1996," EPA-454/R-97-011, December, 1997.

Table VIII.B-4
Diesel PM Emissions
From Nonroad Sources

Year	1996	2007	2020
Thousand short tons per year	345,800	282,800	310,800
Cumulative percent reduction from 1996	***	18%	10%

C. Data Gaps and Uncertainties

There are significant gaps in data on MSAT emissions from nonroad engines. These data gaps contribute to a less developed understanding of nonroad MSAT inventories compared to our understanding of on-highway vehicle MSAT emissions. The largest single data gap is in the area of emission factors. While we have basic emission factors for VOC and PM for most of the nonroad categories, we have very little VOC speciation data for the given categories that would allow us to use VOC as a surrogate to estimate emissions of specific MSATs. Given the large variety of nonroad engine sizes, types and uses, as well as the likelihood that this variety will result in some differences in VOC composition, it is important that we obtain or develop speciated VOC data specific to each nonroad category in order to more accurately project nonroad MSAT inventories. These gaps, too, must be filled in order to accurately assess the need for, and the most appropriate direction of, any future MSAT control program targeted specifically at nonroad mobile sources. Our Technical Analysis Plan, described in the preamble to our rule, contains a strategy to obtain and evaluate this data so we can evaluate potential strategies relating to hazardous air pollutants from nonroad engines and vehicles.