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Draft Technical Support Document: Control of Emissions of Hazardous Air Pollutants from Motor Vehicles and Motor Vehicle Fuels

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

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This technical report does not necessarily represent final EPA decisions or positions. It is intended to present technical analysis of issues using data that are currently available. The purpose in the release of such reports is to facilitate the exchange of technical information and to inform the public of technical developments which may form the basis for a final EPA decision, position, or regulatory action.

Executive Summary

A range of compounds known as hazardous air pollutants are emitted from motor vehicles and fuels and are known or suspected to have serious health impacts. This document describes in greater detail the technical reasoning behind our proposed program to address emissions of hazardous air pollutants from mobile sources.

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 and, more specifically, a review of our 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. Selection was 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 identifies EPA's consensus scientific judgment on the characterization of the potential adverse health effects that may result from a lifetime exposure to various substance. This process resulted in a list of 21 compounds. Chapter 3 contains important health and environmental information for each of those MSATs.

Before we can evaluate whether additional mobile source air toxics controls are appropriate, we must evaluate the effectiveness of current controls in reducing on-highway emissions of these MSATs. Our analysis, contained in Chapter 4, shows that the programs we currently have in place, including our reformulated gasoline (RFG) program, national low emission vehicle (NLEV) program, Tier 2 motor vehicle emissions standards and gasoline sulfur control requirements (Tier 2), and our recently proposed heavy-duty engine and vehicle standards and on-highway diesel fuel sulfur control requirements (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 75 percent or more. In addition, we expect to see on-highway diesel PM emission reductions of over 90 percent. Nonroad engines and equipment also contribute substantially to levels of MSATs emissions and have not been subject to the same stringent controls as highway sources.

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 an 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.

We next consider whether there are additional air toxics controls that should be put in place at this time to further reduce on-highway MSAT inventories. Chapter 6 provides our analysis with respect to vehicle-based controls. With regard to vehicle-based controls, we conclude that the it is not appropriate at this time to propose more stringent standards than the technology forcing standards found in our recently adopted Tier 2 and recently proposed HD2007 rule standards. Chapter 7 provides our analysis for fuel-based controls. With regard to fuels-based controls, we are proposing a gasoline benzene control program that requires refiners to maintain the current levels of over-compliance with RFG and anti-dumping toxics requirements. Because the proposed standards for each refiner as the same as the 1998/9 average gasoline benzene level for that refiner, the proposed standards are expected to impose only negligible costs, if any.

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

$\mu g/m^3$	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	Comprehensive Human Activitiy 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
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
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
UATS	Integrated Urban Strategy (also called Urban Air Toxics Strategy)
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. 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 §112 of the Act, §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 our estimates of highway motor vehicle emissions ("emission inventories") of these compounds. We present baseline inventory information for 1990 and 1996 and compare 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 highway emission control programs are fully phased-in. We estimate that highway inventories of certain key MSATs will decrease by as much as 70 percent by 2020. This chapter also describes the methods we used to estimate these emission inventories.

In Chapter 5 we review existing information on ambient concentrations of toxic compounds, from both monitoring and modeling efforts. We also present our 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 or are proposed for the near future. Chapter 6 addresses vehicle-based controls and concludes that our recently finalized Tier 2/Gasoline Sulfur program and our Heavy-Duty Engine/Diesel Sulfur (2007) proposal represent the most stringent controls feasible for motor vehicles air toxics control at this time. Chapter 7 contains our analysis of fuel-based controls. In it we present our rationale for our proposed provisions relating to benzene in gasoline. We also discuss the challenges to fuel-based air toxics control.

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.

B. 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 those emissions. 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 pollution 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 Tail Pipe 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 on the order of 8 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 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[1].

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[1] 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.

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

Year	1970	1972	1975	1980	1994	1999
HC	2.2	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 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 air/fuel ration and quicker catalyst "light-off"). Another example is the Supplemental Federal Test Procedure and standards, finalized in 1996, which better captures 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 Tail Pipe Emission Controls

We have controlled emissions from heavy-duty engines and vehicles since 1984. As of 1998, new heavy-duty truck engines must meet standards of 4 g/bhp-hr NOx, 1.3 g/bph-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 proposed rule to reaffirm these standards for diesel trucks for 2004, and to adopt separate standards for gasoline trucks (64 FR 58471; 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
NOX	10.7 –>		6.0	5.0 –>				4.0	2.4[2]
PM	N/A	0.60 –>		0.25 –>		0.10 –>			
HC	1.3 –>								2.4[2]

1. Standards for Gasoline Engines are the same as for diesel, until 2004. See Table I.B-3 for gasoline engine standards proposed for 2004.
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.

Table I.B-3
Heavy-Duty GasolineVehicle Standards Proposed for 2004

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	1.0 g/bhp-hr (combined NOX and HC)	

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 proposed rule for 2004 and later heavy-duty engines and vehicles, we also proposed 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 would also extend 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 propose to extend requirements for on-board diagnostic (OBD) systems to both diesel and gasoline fueled vehicles weighing between 8,500 and 14,000 pounds (gross vehicle weight) to help identify any possible failure of components of the emission control system.

Our recent proposed Heavy-Duty Engine/Diesel Sulfur (2007) program, proposed June 2, 2000, proposes stringent exhaust emission standards for heavy-duty engines and vehicles beyond the 2004 levels starting in 2007, as well as reductions in sulfur levels from diesel fuel starting in 2006 (65 FR 35430). We describe this program in greater detail in Chapter 6 of this TSD.

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. 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 by 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 proposed rule for Heavy-Duty Engines/Diesel Sulfur control also proposes to 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 proposed to extend ORVR to heavy-duty gasoline vehicles between 8,500 and 10,000 lbs (gross vehicle weight) (64 FR 58471). 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)[1]	2004-2006	

[1] Proposed October 1999 (64 FR 58471)

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 Clean Air Act 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 when compared with 1990 baseline gasoline (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 that are known to be emitted from passenger cars, on-highway trucks, and various types of nonroad equipment. Several of these compounds 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 emission control standards for hazardous air pollutants from motor vehicles and their fuels. Except for benzene and formaldehyde which are specifically mentioned in 202(l), the Act does not specify the compounds that should be included in such a control program. Therefore, the first step in developing a mobile source air toxics control program is to identify the compounds that should be controlled.

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

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

EPA developed the list of MSATs by first compiling all available recent (i.e., less than 10 years old) studies which speciated the emissions from mobile sources and their fuels. The compilation did not include speciations of emissions from alternative fueled vehicles, currently in a very small number of vehicles, such as flexible-fueled vehicles. We then compared the list of compounds in EPA's Integrated Risk Information System (IRIS) database to the speciated lists of compounds in these studies. IRIS is a database of compounds that identifies EPA's consensus scientific judgment on the characterization of the potential adverse health effects that may result from a lifetime exposure to various substance. IRIS may also indicate that based on the current data a compound has been found not to have the potential to cause adverse health effects.

By comparing the list of compounds in IRIS to these emission speciation studies , we generated a list of 21 compounds. An evaluation of the potential for adverse health effects reflected in IRIS and in the ongoing agency scientific assessments of these compounds indicates that the potential for adverse health effects from exposure to these compounds warrants inclusion as a MSAT. EPA also compared its universe of known compounds emitted from mobile sources against other lists or sources of information on toxic substances, and did not identify any additional substance that we believe should be listed at this time. EPA believes this process is fluid, and allows for re-evaluation of the MSAT list in the future, as information is learned about additional compounds or new information is learned about the 21 compounds.

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

In the sections that follow, we will describe in more detail each step of our methodology for identifying the list of MSATs.

1. Identifying Pollutants Emitted from Mobile Source

In identifying a list of MSAT, EPA first compiled all available recent studies which speciated emissions from mobile sources and their fuels. Again, this compilation did not include speciation of emissions from alternative fueled vehicles, currently used in a very small number of vehicles. It is difficult to get a precise picture of these emissions due to the variety and number of databases in the literature. This is particularly true for hydrocarbon (HC) speciation databases. Most toxic air pollutants are hydrocarbons by their chemical nature and thus will be detected only if the HCs are chemically separated and identified (speciated). Many test programs that characterize mobile source emissions identify only total hydrocarbons (THC) without separating out the individual species of hydrocarbons and many use different test methods. The issue is further complicated by the limited availability of these databases for certain vehicle classes and engine types.

We have recent (less than ten years old) speciation profiles for emissions from light-duty gas vehicles (LDGV), heavy-duty diesel vehicles (HDDV), heavy-duty gasoline vehicles (HDGV), gasoline powered nonroad engines, and turbine engine aircraft.[2] Data for other vehicle/engine types (e.g., light-duty diesel engines and nonroad diesel engines) either does not exist or are outdated (more than 10 years old) and thus are judged not to be representative of the emissions from vehicles on the highway today. However, it is unlikely that the lack of recent data for these vehicle and engine types would result in the absence of compounds from the list, since the combustion process is similar to vehicle and engine types for which we do have data.

The forty-four speciation studies listed in Appendix I at the end of this chapter all attempt to accomplish more or less the same objective: separating and identifying the compounds that comprise the hydrocarbon and particulate matter emissions.[3] The protocol followed in each study is generally the same, though parts of the analysis may vary depending on the purpose of the study. Each study generally starts by defining the fleet, or types, of vehicles or engines on which data will be collected. This could be light-duty gas vehicles, heavy-duty diesel engines,

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[2] See Appendix I.

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

nonroad engines, or a mix of various types. The vehicles themselves can also be a mix of older and newer models, with varying mileage. The type of fuel to be used must also be specified.

Vehicles and engines are tested on a dynamometer, which is basically mechanical treadmill for the vehicle/engine. The test vehicle/engine is run through a set pattern of starts, stops, idle, and 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 emission are trapped on particulate filters and analyzed. Analysis of metals usually doesn't specify the actual individual chemical form of the metal, only reporting the total amount of the identified metal emitted. Particulate samples, in the past, have routinely looked at particles no finer than 10 μm in diameter, whereas newer research indicates that most particles are much smaller. Once the chemical, metal, and/or particle analysis of the collected material is complete, the results are routinely presented as grams (or milligrams) of chemical "x" per mile driven (g/mi).

Appendix I provides a list of the speciation studies from various types of mobile sources and their fuels. In the next section we describe which compounds emitted from mobile sources and their fuels may be considered toxic air pollutants.

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

IRIS is an EPA database of scientific information that contains the Agency consensus scientific positions on potential adverse health effects that may result from lifetime exposure to various 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.[4]

IRIS contains chemical-specific

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.

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[4] EPA IRIS Database, http://www.epa.gov/ngispgm3/iris/index.html

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

The RfD or RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime. RfDs and RfCs are based on an assumption of lifetime exposure and may not be appropriately applied to less-than-lifetime exposure situations. RfDs and RfCs may be derived for the noncarcinogenic effects of 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. This judgment is made independent of consideration of the agent's potency. Whether a quantitative assessment is performed is dependent both on the weight-of-evidence and the suitability of the available dose-response data. A quantitative assessment, which may include an oral slope factor and oral and/or inhalation unit risk estimates, may then be presented. The oral slope factor is an upper-bound estimate of the human cancer risk per mg of agent/kg body weight/day. The unit risk, which can be calculated from the slope factor or other scientifically appropriate methodologies, is also an upper-bound estimate in terms of either risk per μg/L drinking water, or risk per μg/cu.m air concentration.

Each reference dose/concentration and carcinogenicity assessment has been 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 information in IRIS must be combined with data on specific exposure situations.

3. List of Mobile Source Air Toxics

By comparing the list of pollutants for which information is stored in IRIS to the compounds identified in the emission speciation studies, we identified 21 compounds listed on IRIS that have been found in the emissions from mobile sources and their fuels. This list of MSATs is set out in Table II.B-1, below. Each of these pollutants are known, probable or possible human carcinogens (Group A, B or C) or were considered by the Agency to pose a risk of adverse noncancer health effects.[5] It should be noted that this list is not meant to be fixed, and may change as new health and emissions data become available.

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

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

[1] Although the different metal compounds differ in their toxicity the onroad 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] MTBE is listed due to its inhalation air toxics effects and not due to ingestion exposure associated with drinking water contamination.
[4] 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.

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. The purpose of the list is more as a screening tool - it identifies those compounds emitted from motor vehicles or their fuels, and where the available information about their potential for adverse health or welfare effects indicates that 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 the 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

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[5] 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.

appear to be warranted.

As described in section II.B.1, it is difficult to identify the specific form of metals being emitted in mobile source exhaust because the databases only report the total amount of metal compound identified. As a result, we have chosen to list the entire group of metal compounds if any compound of the metal is listed in IRIS as potentially causing adverse human health effects and any compound of the metal has been detected in emissions from mobile sources or their fuels.[6] For example, if we assume most chromium (Cr) emissions for mobile sources are unidentified as to the species, we would present the emissions as total chromium and not attempt to allocate these emissions because of the lack of accurate metal speciation information in most cases. When we assess the range of potential health impacts associated with exposure to chromium compounds, we consider the health effects associated with each compounds for which we have information. For chromium, the most toxic form in IRIS is Cr+6; hence the health impacts described for chromium compounds include these most serious effects even though it is highly unlikely that all mobile source emissions are Cr+6. EPA believes this listing approach is a reasonable, health-protective way to handle the uncertainty surrounding mobile source emissions of metals. We also recognize that this is not an appropriate methodology for assessing the actual health risks of the entire group of metal compounds emitted from mobile sources.

With regard to alternative-fueled vehicles, most of the compounds included in their exhaust are included on our list of MSATs (e.g., formaldehyde, acetaldehyde). It should be noted that, depending on their fuel, these vehicles may also emit unburned ethanol 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. However, there is a paucity of data on potential inhalation effects of ethanol, and the compound is not listed in IRIS. Thus it has not included on the proposed list of MSATs.

Methanol is also a promising alternative fuel for motor vehicles, and a small number of flexible fuel vehicles operate on a methanol mixture (e.g., 85% methanol). Inhalation of methanol at high concentrations (greater than 1000 ppm) has caused birth defects in rats and mice and at low levels can cause symptoms such as eye irritation, headaches, dizziness, and nausea. Methanol is highly toxic by oral exposure routes and is listed in IRIS.

The Agency will re-examine whether to include these two compounds on the list of MSATs in the final technical support document, and we are requesting comment on this issue in the preamble.

C. How Our List of MSATs Compares to Other Lists or Sources of Data on Toxics

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[6] Metals emissions are only present in mobile source exhaust and not in evaporative emissions from fuels.

To address the possibility that the IRIS files do not include all the mobile source emission chemicals with significant potential for adverse health effects, other lists of pollutants posing potential health and/or environmental impacts were consulted.

1. Comparison to Other Lists of Toxics

In addition to IRIS, there are other lists of chemicals that have been derived in response to potential human health or environmental hazards. IRIS was chosen as the primary source in developing the MSATs list because it presents Agency consensus opinions, and its current process includes both internal and external (sometimes involving EPA's Science Advisory Board) peer review steps. To address the possibility that the IRIS file does not include all the mobile source emission chemicals with potential for significant adverse health effects, four other lists of pollutants posing potential health and/or environmental impacts were consulted. The four other lists of toxic air pollutants were: the CAA section 112(b) list of hazardous air pollutants, the California EPA (CalEPA) list of toxic air contaminants (TAC), the Department of Health and Human Service Agency for Toxic Substances and Disease Registry (ATSDR) list of Minimal Risk Levels (MRLs), and the list of agents evaluated in the International Agency for Research on Cancer (IARC) monographs series on cancer.

a. Section 112(b) of the Clean Air Act

Section 112(b) of the Clean Air Act lists 188 compounds as hazardous air pollutants (HAPs).[7] Congress added this list as part of the 1990 Amendments to the Clean Air Act using the lists of hazardous substances and contaminants from other federal programs and databases.[8] Section 112(b)(2) authorizes EPA to add to this list pollutants that present, or may present, through inhalation or other routes of exposure, a threat of adverse human health effects or adverse environmental effects. To date, no additional pollutants have been added to the 112(b) list.

b. The Agency for Toxic Substances and Disease Registry (ATSDR)

ATSDR, which is part of the U.S. Centers for Disease Control, regularly publishes Health Guidelines Comparison Values (CVs) for many toxic substances. ATSDR describes CVs as media-specific concentrations to be used by health assessors in selecting environmental contaminants for further evaluation. CVs are concentrations below which it is considered unlikely that contaminants pose a health threat. Concentrations above a CV do not necessarily

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[7] The Act includes 189 hazardous air pollutants, but in 1996, EPA delisted caprolactam as a HAP [61 Fed. Reg. 30,816 (June 18, 1996)].

[8] The 112(b) list evolved from the lists of pollutants found at the time in section 313 of the Emergency Planning and Community Right-to-Know Act, 42 U.S.C. § 11023, section 104 of the Comprehensive Environmental Response, Compensation and Liability Act, 42 U.S.C. § 9604(i), and the National Air Toxics Information Clearinghouse database on July 1986.

represent a threat, and CVs are thus not intended for use as predictors of adverse health effects or for setting cleanup levels.

ATSDR's chronic duration minimum risk level (MRL) is the CV that most closely approximates EPA's RfD and RfC. An MRL is an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (other than cancer) over a specified duration of exposure. ATSDR develops MRLs for acute, intermediate, and chronic duration exposures by the inhalation and oral routes. The concept, definition, and derivation of MRLs are consistent with those of EPA's RfC and RfD. ATSDR publishes MRLs as part of its toxicological profile documents for each substance (ATSDR 1998). ATSDR toxicological profiles receive public review as well as extensive internal and external peer review.

c. California Environmental Protection Agency (CalEPA)

CalEPA has developed dose-response assessments for many HAPs that have not been evaluated by either EPA or ATSDR. These assessments contain information on carcinogenicity, and health effects other than cancer resulting from chronic and acute exposure. The non-cancer information includes available inhalation health risk guidance values developed by EPA or CalEPA, expressed as acute or chronic reference exposure levels (RELs). CalEPA defines the REL as a concentration level or dose at or below which no health effects are anticipated. Because this concept is substantially similar to EPA's non-cancer dose-response values, RELs are useful tools for substances that EPA has not assessed. CalEPA's quantitative dose-response information on carcinogenicity by inhalation exposure is expressed in terms of the unit risk, defined similarly to EPA's unit risk. CalEPA's methodology and values were subjected to an external peer review process in 1995-1996, and although some individual values were judged in need of improvement, the methodology was considered generally similar to that of the EPA. Since then Cal EPA has updated many of their assessments to further improve consistency with the EPA and reflect current knowledge.[9]

The CalEPA Toxic Air Contaminant (TAC) list includes over 200 compounds that CalEPA has determined to pose a threat to public health. The TAC list, like IRIS, receives extensive internal and external peer review, and the supporting assessments are reviewed by an independent Scientific Review Panel (SRP) before a compound is added to the TAC list.

d. International Agency for Research on Cancer (IARC)

IARC was established in 1965 by the World Health Organization, to coordinate and conduct research on the causes of human cancer and to develop scientific strategies for cancer control. IARC performs epidemiological and laboratory research, and disseminates scientific

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[9] CalEPA, 1997. Toxic Air Contaminant Identification List. California Environmental Protection Agency, Air Resources Board. September 1997.

information through meetings, publications, courses and fellowships. As part of its mission, the IARC assembles evidence that substances cause cancer in humans and issues judgments on the strength of the evidence. To this end they have created a database known as the IARC Monographs series which publishes authoritative independent assessments by international experts of the carcinogenic risks posed to humans by a variety of agents, mixtures and exposures. Since its inception in 1972, IARC has reviewed more than 800 agents.

IARC's weight-of-evidence categories are Group 1 (carcinogenic in humans), Group 2A (probably carcinogenic), Group 2B (possibly carcinogenic), Group 3 (not classifiable), and Group 4 (probably not carcinogenic). The rankings may be applied to either single chemicals or mixtures (IARC 1998). EPA often relies on IARC weight-of-evidence determinations for substances that EPA itself has not assessed.

e. Results of Comparison

Comparing these four lists against the emissions speciation studies, we identified two additional compounds not included on our list of 21 MSATs -- propionaldehyde and 2,2,4-trimethylpentane. Both the CalEPA TAC list and the CAA section 112(b) HAP list contain these compounds.

Propionaldehyde is a highly volatile compound that is emitted into the air as a result of combustion of wood, gasoline, and diesel fuel, as well as by certain plants and at sites where it is produced or used as a chemical intermediate in the manufacture of propionic acid, polyvinyl and other plastics in the synthesis of rubber chemicals and as a disinfectant and preservative. A recent study estimates that 63 percent of the propionaldehyde in the draft 1996 National Toxics Inventory (NTI) was attributable to on-highway motor vehicles.[10] There are little data available on the potential human health effects associated with propionaldehyde exposure. The few animal studies available focused on acute (short-term) inhalation exposure to very high levels and reported anesthesia (loss of consciousness) and liver damage, as well as lethal pulmonary edema (fluid accumulation in the lung).[11]

The compound 2,2,4-trimethylpentane is found in both exhaust and evaporative emissions from gasoline motor vehicles. Current inventory estimates are not available, but since it is a component of gasoline and is not expected to have significant stationary source emission, we presume that motor vehicles will dominate this inventory. Little data are available on potential human health effects. The limited animal data indicate that 2,2,4-trimethylpentane is a

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[10] Billings, R., T. Kraus, B. Hunt, J. Mangino, R. Cook, L. Driver 1998. Development and Comparison of 1990 and 1996 Mobile sources Hazardous Air Pollutants Emissions Estimates. Presented at AWMA Conference, "Emission Inventory: Living in a Global Environment" New Orleans, LA December 8, 1998.

[11] U.S. Department of Health and Human Services. 2000. Hazardous Substances Database (HSDB, online database), National Toxicology Information Program, National Library of Medicine, Bethesda, MD. of the U.S. Dept of Health.

respiratory tract irritant and central nervous system depressant.[12]

At this time, EPA is not including propionaldehyde or 2,2,4-trimethylpentane in the list of MSATs because the Agency has not drawn a conclusion on the potential adverse human health effects asiciated with exposure to these pollutants. In the preamble to the NPRM, however, EPA is requesting comment on this decision as well as any scientific information on the potential health effects of these pollutants.

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[12] U.S. Department of Health and Human Services. 2000. Hazardous Substances Database (HSDB, online database), National Toxicology Information Program, National Library of Medicine, Bethesda, MD. of the U.S. Dept of Health.

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3. Bass, E. A., and M. S. Newkirk, 1995. Reactivity Comparison of Exhaust Emissions from Heavy-Duty Engines Operating on Gasoline, Diesel, and Alternative Fuels. Southwest Research Institute, Report No. SwRI 9856, December, 1995.

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10. Eastern Research Group, 1999. Documentation for the 1996 Base year National Toxics Inventory for Commercial Marine Vessel and Locomotive Sources. Draft Report prepared for Emission Factor and Inventory Group, Office of Air Quality Planning and

Standards, U. S. EPA, November 1, 1999.

11. Eastern Research Group, 1999. Documentation for the 1996 Base year National Toxics Inventory for Aircraft Sources. Draft Report prepared for Emission Factor and Inventory Group, Office of Air Quality Planning and Standards, U. S. EPA, November 1, 1999.

12. Eastern Research Group, 1999. Documentation for the 1996 Base year National Toxics Inventory for Nonroad Sources. Draft Report prepared for Emission Factor and Inventory Group, Office of Air Quality Planning and Standards, U. S. EPA, November 1, 1999.

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Chapter 3: Health Effects of Mobile Source Air Toxics

Chapter 2 of this Draft Technical Support Document describes how we identified the list of mobile source air toxics (MSATs). This chapter provides information on the 21 MSATs, including their physical properties, uses, sources of potential exposure, and health hazards.

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.

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. 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 formaldehyde.[13] 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. This classification is based on a sufficient database of animal carcinogenicity studies.[14] 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.

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[13] 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.

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

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. 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.[15,16]

B. Acrolein

Acrolein is an aldehyde primarily used as an intermediate in the manufacture of acrylic acid. It can be formed from the breakdown of certain pollutants in outdoor air or from burning tobacco or gasoline. 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. Acetaldehyde 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 this limited animal data, the carcinogenic potential of an acrolein metabolite, evidence of mutagenicity in bacteria and structural similarity to probable or known human carcinogens.

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. No information is available on the reproductive or developmental effects of acrolein in humans.[17]

C. Arsenic Compounds

Arsenic, a naturally occurring element, is found throughout the environment. It is

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

[16] U.S. Environmental Protection Agency (EPA). *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.

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

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.

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. The EPA has classified inorganic arsenic as a Group A, human carcinogen.[18]

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.[19]

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,

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

[19] 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.

but depends on control technology and fuel composition (e.g., benzene level and Reid Vapor Pressure (RVP)). Benzene emissions from mobile sources account for approximately 76 percent of the emissions in the 1996 National Toxics Inventory.

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. Tobacco smoke contains benzene and accounts for nearly half the national exposure to benzene.

The EPA has recently reconfirmed that benzene is a known (Group A) human carcinogen by all routes of exposure[20]. 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[21] 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.[22] 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

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

[21] 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.

[22] EPA 1993a. 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.

humans and animals results in pancytopenia,[23] a condition characterized by decreased numbers of circulating erythrocytes (red blood cells), leukocytes (white blood cells), and thrombocytes (blood platelets).[24,25] 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.[26]

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. 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.

1-3-Butadiene was classified by EPA as a Group B2 (probable human) carcinogen in 1985[27]. This classification was based on evidence from two species of rodents and epidemiologic data. EPA recently prepared a draft assessment that proposes that sufficient evidence exists to propose 1,3-butadiene be a known human carcinogen[28]. However, the Environmental Health

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[23] 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).

[24] 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.

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

[26] 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.

[27] 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.

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

Committee of EPA's Scientific Advisory Board (SAB), in reviewing the draft document, issued a majority opinion that 1,3-butadiene should instead be classified as a probable human carcinogen[29]. The SAB panel recommended that EPA calculate the lifetime cancer risk estimates based on the human data from Delzell et al. 1995[30] and account for the highest exposure of "360 ppm-year" for 70 years. Further input recommended that EPA also 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.

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 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. 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

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

[30] 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.

carcinogen, resulting in an increased risk of lung cancer. Animal studies have shown chromium (VI) to cause lung tumors via inhalation exposure. The EPA has classified chromium (VI) as a Group A, known human carcinogen.[31]

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).[32]

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) particulates. Chromium III is most appropriately designated a Group D -- not classified as to its human carcinogenicity, there are inadequate data to determine the potential carcinogenicity of chromium III.

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.[33]

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,

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

[32] 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.

[33] 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.

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) (an Office of the World Health Organization).[2] The IARC classifies the most potent form of dioxin, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), as a "Group 1" carcinogen, i.e., dioxin is carcinogenic to human.[34]

Dioxins/furans have also linked low-grade exposure to dioxin to a wide array of other health problems, including changes in hormone levels as well as developmental defects in babies and children.[35]

H. Diesel Exhaust

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.

1. Cancer Effects of Diesel Exhaust

The EPA draft *Health Assessment Document for Diesel Emissions* (draft Assessment) is currently being revised based on comments received from the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board.[36] EPA's draft position is that diesel

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[34] 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.

[35] 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.

[36] U.S. 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

exhaust is a likely human lung carcinogen and that this cancer hazard exists for occupational and environmental levels of exposure.[37]

In evaluating the available research for the draft *1999 Assessment*, EPA found that individual epidemiological studies numbering about 30 show increased lung cancer risks associated with diesel emissions within the study populations of 20 to 89 percent depending on the study. Analytical results of pooling the positive study results show that on average the risks were increased by 33 to 47 percent. Questions remain about the influence of other factors (e.g., effect of smoking), the quality of the individual epidemiology studies, exposure levels, and consequently the precise magnitude of the increased risk of lung cancer. From a weight of the evidence perspective, EPA believes that the epidemiology evidence, as well as supporting data from certain animal and mode of action studies, support the Agency's proposed conclusion that exposure to diesel exhaust is likely to pose a human health hazard at occupational exposure levels, as well as to the general public exposed to typically lower environmental levels of diesel exhaust.

While available evidence supports EPA's draft position that diesel exhaust is a likely human lung carcinogen, and thus is likely to pose a cancer hazard to humans, the absence of quantitative estimates of the lung cancer unit risk for diesel exhaust limits our ability to quantify with confidence the actual magnitude of the cancer risk. In the draft 1999 *Assessment*, EPA acknowledged these limitations and provided a discussion of the possible cancer risk consistent with general occupational epidemiological findings of increased lung cancer risk and relative exposure ranges in the occupational and environmental settings.

2. Noncancer Effects of Diesel Exhaust

The noncancer effects of diesel exhaust are also of concern to the Agency. EPA believes that chronic diesel exhaust exposure, at sufficient exposure levels, increases the hazard and risk of an adverse consequence (including respiratory tract irritation/inflammation and changes in lung function). The draft *1999 Assessment* discussed an existing inhalation reference concentration (RfC) for chronic effects that EPA intends to revise in the next draft 1999 *Assessment* in response to CASAC comments. The revised RfC will be reviewed by CASAC at a future meeting.

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www.epa.gov/ncea/diesel.htm.

[37] The EPA draft designation of diesel exhaust as a likely human carcinogen is subject to further comment by CASAC in 2000. The draft designation of diesel exhaust as a likely human carcinogen under the 1996 Proposed Guidelines for Carcinogen Risk Assessment is very similar to the current 1986 Guidelines for Carcinogen Risk Assessment that designate diesel exhaust as a probable carcinogen (B1 carcinogen). The new guidelines, once finalized, will incorporate a narrative approach to assist the risk manager in the interpretation of the carcinogen's mode of action, the weight of evidence, and any risk related exposure-response or protective exposure recommendations.

3. Diesel Exhaust and Diesel Particulate Matter

While some gaseous components of diesel exhaust may play a role in the cancer hazard attributed to diesel exhaust exposure, studies suggest that the particulate component plays a substantial role in carcinogenicity and other noncancer effects. Diesel particulate matter is mainly attributable to the incomplete combustion of fuel hydrocarbons as well as engine oil and other fuel components such as sulfur. Primary diesel particles mainly consist of carbonaceous material, with a small contribution from sulfuric acid and ash (trace metals). Many of these particles exist in the atmosphere as a carbon core with a coating of organic carbon compounds, or as sulfuric acid and ash, sulfuric acid aerosols, or sulfate particles associated with organic carbon. Most (80-95%) diesel particles are in the fine ($<2.5\ \mu m$) and ultrafine ($<0.1\ \mu m$) size range.

Investigations show that diesel particles (the elemental carbon core plus the adsorbed organics) induce lung cancer at high doses, and that the particles, independent of the gaseous compounds, elicit an animal lung cancer response. The presence of non-diesel elemental carbon particles, as well as the organic-laden diesel particles, correlate with an adverse inflammatory effect in the respiratory system of animals. Additional evidence suggesting the importance of the role of particulate matter in diesel exhaust includes the observation that the extractible particle organics collectively produce cancer and adverse mutagenic toxicity in experimental test systems. Many of the individual organic compounds are mutagenic or carcinogenic in their own right.

Diesel PM contains small quantities of numerous mutagenic and carcinogenic compounds. Some assessments report up to 16 organic compounds in primary and secondary diesel exhaust with known or suspected carcinogenic activity or other toxicologically significant effects.[38] While representing a very small portion (less than one percent) of the national emissions of metals, and a small portion of diesel particulate matter (one to five percent), we note that several trace metals of toxicological significance are also emitted in diesel exhaust in small amounts including chromium, manganese, mercury and nickel. In addition, small amounts of dioxins have been measured in diesel exhaust, some of which may partition into the particle phase, though the impact of these emissions on human health is not clear.

Mobile sources account for almost all diesel particulate matter emissions and on-road sources account for approximately one-third of the mobile source diesel particulate matter emissions. Diesel PM emissions from all mobile sources account for 17% of ambient PM_{10} emissions in 1996, excluding the contribution from natural and miscellaneous sources.

In assessing the health impacts of diesel particulate matter, it is important to acknowledge that diesel exhaust particulate matter is part of ambient $PM_{2.5.}$ While diesel particulate matter contributes to ambient levels of $PM_{2.5}$, its high content of organic compounds absorbed onto a

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[38] U.S. 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.

carbon core and the concentration of ultrafine particles in diesel particulate matter distinguish it from other non-combustion sources of $PM_{2.5}$. The composition of diesel PM, which is dominated by organic matter, contrasts strongly with the typical chemical composition of ambient $PM_{2.5}$ which is dominated by sulfate for aerosols measured in the eastern U.S and by nitrate, ammonium, and organic carbon in the western U.S. Noncancer health effects associated with exposure to diesel PM overlap with some health effects reported for ambient PM including respiratory symptoms (cough, labored breathing, chest tightness, wheezing), and chronic respiratory disease (cough, phlegm, chronic bronchitis and some evidence for decreases in pulmonary function). Considerably more research has been conducted to investigate the noncancer health effects attributable to ambient $PM_{2.5}$ than those attributable to diesel particulate matter. A qualitative comparison of adverse effects of exposure to $PM_{2.5}$ and diesel exhaust particulates shows that the respiratory system is adversely affected in both cases, though $PM_{2.5}$ has a wider spectrum of adverse effects for humans. A carcinogenicity hazard for $PM_{2.5}$ has not yet been clearly shown, however.

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. 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, 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 produces such as cleaning products or paints.

Limited information is available on the carcinogenic effects of ethylbenzene in humans or 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.

Noncancer, acute (short-term) exposure to ethylbenzene in humans results in 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. 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.

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[39]. It is present in 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).

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[40]. 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.

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

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

[40] 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.

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 obstruction, and nasal discharge following chronic exposure.[41] In persons with bronchial asthma, the upper respiratory irritation caused by formaldehyde can precipitate an acute asthmatic attack, sometimes at concentrations below 5 ppm.[42] Formaldehyde exposure may also cause bronchial asthma-like symptoms in nonasthmatics.[43,44] It is unclear whether asthmatics are more sensitive than nonasthmatics to formaldehyde's effects.[45]

Immune stimulation may occur following formaldehyde exposure, although conclusive evidence is not available. Also, little is known about formaldehyde's effect on the central nervous system. Several animal inhalation studies have been conducted to assess the developmental toxicity of formaldehyde. The only exposure-related effect noted was decreased maternal body weight gain at the high-exposure level. No adverse effects on reproductive outcome of the fetuses that could be attributed to treatment were noted.

K. n-Hexane

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. 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.

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

[42] 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.

[43] 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.

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

[45] EPA 1991a. 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.

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.

Noncancer, 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.

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 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 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.

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. 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.

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.

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 effects 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 weight and slowed postnatal neurobehavioral development noted.

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 µg/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.[46] Manganese compounds from mobile source 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. The EPA has classified manganese as a Group D, not classifiable as to carcinogenicity in humans.

Health effects in humans have been associated with both deficiencies and excess intakes of manganese. Chronic (long-term) exposure to low levels of manganese in the diet is considered to be nutritionally essential in humans, with a recommended daily allowance of 2 to 5 mg/d. 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. Impotence and loss of libido have been noted in male workers afflicted with manganism attributed to high-level inhalation exposures to

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)

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[46] 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

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.[47] 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 docker 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 the environment from the methylation of the inorganic mercurial ion.

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. Recent analytical methods typically used to measure mercury from mobile sources are not sensitive enough to measure these trace level emissions. Thus, if data were developed using more sensitive methods, we may well have detectable mobile source mercury emissions. Estimates of other mobile source mercury emissions rely on an outdated database that speciates PM emissions from mobile sources.[48] For

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[47] 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).

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

one category of vehicle, light duty diesel vehicles, no recently developed emissions factors were available, so we used factors developed in the mid-1980s by South Coast Air Quality Management District. 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. 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.

Human studies are inconclusive regarding the carcinogenicity of elemental mercury. The EPA has classified elemental mercury as a Group D, not classifiable as to human carcinogenicity.

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.

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 emission from mobile source account for approximately 86 percent of the total MTBE inventory in the 1996 National Toxics Inventory.

Human exposure to MTBE may occur via inhalation, dermal or by oral contact. For the purpose of the MSATs listing in Chapter 2, only the health effects due to inhalation exposure are considered not ingestion exposure due to drinking water contamination or dermal absorption. Acute (short-term) effects on humans receiving MTBE injections into the gallbladder during its use as a medical treatment to dissolve cholesterol gallstones included nausea, vomiting, and sleepiness have been observed; in one case renal failure was reported. No information is available on the chronic (long-term) health effects of MTBE in humans, or on reproductive or developmental effects. Information is also not available on the carcinogenic effects of MTBE in humans. Three animal cancer bioassys have been performed with MTBE.[49,50,51] The inhalation studies resulted in increased liver tumors in mice and increased kidney tumors in rats. EPA has not classified MTBE with respect to potential carcinogenicity. Rodent studies have also revealed increased liver, kidney, spleen, and adrenal weights, decreased brain weight, body weight, and body weight gain; swollen periocular tissue; and ataxia following chronic inhalation exposure, as well as reproductive and developmental effects.

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, 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.

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, a possible human carcinogen.

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

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[49] Burleigh-Flayer.HD. Chun JS and Kintigh WJ. 1992. Methyl tertiary butyl ether: vapor inhalation oncogenicity study in CD-1 mice. Export, PA: Bushy Run Research Center; OPTS-42098.

[50] Chun JS, Burleigh-Flayer, HD and Kintigh WJ. 1992. Methyl tertiary butyl ether: vapor inhalation oncogenicity study in Fischer 344 rats. Export, PA: Bushy Run Research Center; Report 91N0013B.

[51] 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.

from rodent studies, supported by other subchronic and acute studies, identify nasal and respiratory lesions as critical effects from chronic inhalation exposure to naphthalene.

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 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. 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. Animal studies of soluble nickel compounds (i.e., nickel carbonyl) have reported lung tumors. EPA has classified nickel carbonyl as a Group B2, probable human carcinogen, and given its high instability, nickel carbonyl exposure is extremely rare.

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.

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. 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). 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.

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. 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.[52]

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. 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.

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. The EPA is currently reviewing the potential of styrene to cause cancer, current data is unavailable to classify styrene as a human carcinogen.

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

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

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.

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 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.[53]

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. EPA has classified toluene as a Group D, not classifiable as to human carcinogenicity.

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

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[53] 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

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.

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 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.

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 emission 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.

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). EPA has classified mixed xylenes as a Group D, not classifiable as to human carcinogenicity.

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. Insufficient data are available on the developmental or reproductive effects of mixed xylenes in humans. Animal studies have reported developmental effects, such as an increased incidence of skeletal variations in fetuses, and fetal resorptions via inhalation.

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 planned 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 baseline inventory data for all of the MSATs except napthalene, we do not have inventory projections for all of them. Therefore, we are examining the projected impacts of our current and proposed 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[54] and we present these in this section. However, 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 track 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 they 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 notice.

As we describe in the following discussion, there have been and will continue to be significant reductions in MSATs as a result of implemented, promulgated and proposed regulations. By 2020, we project on-highway emissions of gaseous toxics such as benzene, formaldehyde, 1,3-butadiene, and acetaldehyde, to decrease by 75 percent or more from 1990 levels as a result of our mobile source control programs up to and including our Tier 2 control program and our recently proposed heavy-duty engine and vehicle standards and on-highway diesel fuel sulfur control requirements (HD2007 rule). Under these current and proposed controls we expect on-highway diesel PM emissions to be reduced by more than 90 percent by 2020, as compared with 1990 levels.

This chapter consists of three parts. First, we describe current inventories of MSAT emissions. Next, we describe the methodologies we used to develop our emissions inventories, and we present our projections of how our on-highway emission control programs will reduce

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[54] In this rulemaking the emissions inventory for diesel exhaust is looked at in terms of diesel PM, as that is what we have measured to date. Thus, even though we are proposing to list diesel exhaust as an MSAT, all emissions inventory and trends numbers are stated in terms of diesel PM.

MSAT emissions in the future. Finally, we discuss limitations and uncertainties in our analyses of MSAT emissions.

A. Baseline Inventories

In order to assess the progress we have made in controlling MSATs from our mobile source emission control programs, we need to have a baseline from which to measure our progress. We present this baseline information in this section.

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,"[55] (hereafter referred to as the 1999 EPA Motor Vehicle Air Toxics Study, or the 1999 Study). We addressed these five MSATs and diesel PM because we had detailed information on the emission impacts of emission control technologies, fuel properties, and other parameters for these compounds.

The 1999 EPA Motor Vehicle Air Toxics Study provides 1990 and 1996 estimates of toxic emissions for these compounds. The 1990 baseline represents estimated toxics emissions before any of the programs added by the 1990 Clean Air Act Amendment were implemented. The 1996 estimates reflect toxics emissions with some of the new Clean Air Act programs in place, such as Phase 1 of the RFG program. We present emission estimates for these years in Table IV.A-1. Note that since completion of the Study, we have updated our estimates of diesel PM emissions; these updated estimates are presented in Table IV.A-1. It should be noted these estimates only for on-highway vehicles. We describe how these estimates were developed in Section IV.B, below.

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[55] 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.

Table IV.A-1
Annual Emission Summary for the Total U.S. for Selected Toxics On-Highway Vehicles Only (short tons[56] per year)

Compound	1990	1996
1,3-butadiene	36,000	22,000
Acetaldehyde	41,000	27,000
Benzene	257,000	165,000
Formaldehyde	139,000	80,000
Diesel PM[57]	235,000	180,000
MTBE	55,000	65,000

2. The 1996 National Toxics Inventory

The 1996 National Toxics Inventory (NTI) prepared in connection with the Agency's National Air Toxic Assessment (NATA) activities, also contains emission estimates for 1,3-butadiene, acetaldehyde, benzene, formaldehyde and MTBE. The 1996 NTI emission estimates for these compounds differ slightly from those generated in the 1999 Study, due largely to revisions made to the NTI based on state comments. Since diesel exhaust emissions are not included on the list of 112(b) hazardous pollutants that is the focus of the 1996 NTI, diesel PM estimates have not been compiled there.

The 1996 NTI also contains 1996 emissions estimates for several other MSATs, and includes data for nonroad[58] as well as on-highway sources. We present these data in Table IV.A-

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[56] In this rule we report emissions in terms of short tons as opposed to metric tons. One short ton is 2,000 pounds. To convert to metric tons, multiply short tons by 0.9072. Note that all emissions and percentages in this and subsequent tables are rounded.

[57] The 1996 diesel PM estimate is based on the Tier 2 rulemaking inventories, updated to reflect the Updated Tier 2 Emissions Inventory for light-duty diesel emissions and the proposed 2007 heavy-duty engine rule for heavy-duty diesel emissions, and for 1990, we used estimates from EPA's Trends Report for that year, as described below.

[58] The nonroad inventory in the 1996 NTI includes emissions data for aircraft, commercial marine vessel, locomotives, and other nonroad engines. Note that under the Clean Air Act definition, nonroad does not include aircraft. For convenience, in this document the term "nonroad" will generally include aircraft. It should be noted that the NONROAD model, on which the estimates for nonroad engines are based, is still draft, and the emissions estimates based on this model are subject to change.

2. We also indicate the on-highway and nonroad percentages of the national inventories for these MSATs, where the total national inventories are the sum of the emission from on-highway and nonroad mobile sources, major and area stationary sources, and other sources such as forest fires). Between the 1999 EPA Motor Vehicle Air Toxics Study and the 1996 NTI we have inventory data for all of the 21 MSATs except naphthalene.[59]

Table IV.A-2
1996 On-Highway and Nonroad Emission Inventories of Proposed MSATs
1996 NTI (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*[60]	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%

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

[60] Mass given in tons of TEQ (toxic equivalency quotient). The EPA Office of Research and Development (ORD) has recently developed an inventory for dioxin and dioxin-like compounds using different methods than those used in the 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.

Napthalene	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%

* Indicates also on the list of urban HAPs for the Integrated Urban Air Toxics Strategy (64 Federal Register 38706, July 19,1999).

The above inventory data reflect certain interesting characteristics of mobile source air toxics emissions. First, mobile sources account for the majority of the national inventory of three of the gaseous MSATs that are included on the urban HAP list.[61] 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 used to boost the octane of aviation gasoline.[62] 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 (ethylbenzene, MTBE, n–hexane, styrene, toluene, and xylene), mobile source contributions are high because of the presence of these compounds in gasoline.

In addition, mobile sources account for almost all of diesel PM emissions. As shown in Table IV.A-1, above, 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.

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

[62] 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.

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

This section summarizes our projections of the impacts of our control programs on emissions of MSATs in future years, and describes how we derived these projections. To provide a framework for understanding our results, we first present an overview of our various inventory methodologies. We then present the emissions projection estimates for the five gaseous toxics addressed in the 1999 EPA Motor Vehicle Air Toxics Study. Next, we discuss our projected 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. We used these inventories directly to estimate emissions for all four years of interest for the Pre-Tier 2 and Tier 2 control scenarios (described below). To calculate emission inventories for these toxics under the “Heavy-Duty 2007 Control Scenario” (described below), we relied on the 1999 Study and on data from a spreadsheet model developed in support of the proposed 2007 heavy-duty engine rule.[63] For our final toxics rulemaking, we expect to use the emissions inventories developed for the final 2007 heavy-duty engine rule to more accurately estimate this rule's effect on emissions of the five gaseous air toxics addressed in the 1999 Study. These inventories will reflect our most recent information on emissions factors and local emissions modeling inputs.

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 based our VOC inventories on the emissions inventories constructed for the Tier 2 rulemaking, updated to reflect more recent information. More specifically, we updated the Tier 2 inventories to account for changes reflected in our Updated Tier 2 Emissions Inventory spreadsheet.[64] We replaced the heavy-duty VOC inventories with inventory estimates from the national emissions inventory spreadsheet developed and used for our proposed 2007 heavy-duty engine rule. These heavy-duty engine emission estimates are based on national average fleet mixes, temperatures, VMT distributions by roadway type, and speed by roadway type. For our final toxics rulemaking, we expect to use the VOC emissions inventories developed for the final 2007 heavy-duty engine rule, which will incorporate our most recent information on emissions factors and local emissions modeling inputs.

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.[65] We could not use the Tier 2 rulemaking inventories, since they do not extend to 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 from the Tier 2 rule, since EPA's 1990 Trends estimates have not been updated to reflect the data and analyses that will be used in MOBILE6.

Diesel PM emissions for 1996 and later years were based on several sources. Light-duty diesel PM emissions were taken from the inventory used for the Tier 2 air quality analyses, updated to reflect the Updated Tier 2 Emissions Inventory for light-duty diesel emissions. To estimate heavy-duty diesel PM emissions, we used the national emissions inventory spreadsheet developed and used for our proposed 2007 heavy-duty engine rule; this spreadsheet incorporates recent findings on heavy-duty diesel engine PM emissions that were not reflected in the 1999 Air Toxics Study, accounts for the increasing prevalence of diesel engines in certain engine classes,

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[63] This spreadsheet model can be found in EPA Air Docket A-99-06, Item II-B-31.

[64] Details of this approach can be found in a memorandum by Harvey Michaels to Docket A-2000-12 titled "Adjustment to the Tier 2 Air Quality Inventory for the Mobile Source Air Toxics Proposed Rule".

[65] 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.

and is capable of modeling the effects of the proposed 2007 heavy-duty engine rule.

The spreadsheet model is not capable of producing heavy-duty diesel PM estimates for 1990, so we chose to use heavy-duty diesel inventory estimates from EPA's Trends Report[66] for that year. 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. The 1990 diesel PM inventories developed for this proposal and for the 1999 Study are roughly comparable; the former is 235,000 tons while the latter is 202,000 tons. The 1999 Study results do not explicitly account for county-specific inputs, unlike Trends and the Tier 2 inventory; 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.

For the final toxics rule, we expect to revise our diesel PM inventory based on the analyses being conducted for the final 2007 heavy-duty engine rule. These analyses will include our most current information on light- and heavy-duty diesel engine emissions factors and VMT, and will also reflect county-by-county information on VMT distribution by vehicle class, roadway type, and speed.

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 (http://www.epa.gov/otaq/toxics.htm).[67] 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.[68] 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

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[66] 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.

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

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

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.[69]

The remainder of this subsection provides additional information on how we developed the 1999 Study, and presents emissions inventory results from that study under three control scenarios. 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 inventory based on a spreadsheet model developed in support of the 2007 heavy-duty engine proposed rule.

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

In the 1999 Study we estimated emissions of benzene, formaldehyde, acetaldehyde, and 1,3-butadiene 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 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 does not incorporate impacts of evaporative emission standards in the Tier 2 rule. 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.

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

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[69] 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.

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 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.[70]

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

Chicago, IL	Atlanta, GA	Florida
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.[71] Data from

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[70] 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.

[71] 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.

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.[72] 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 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.

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

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:

$$\mathrm{TOX}_{\mathrm{Flt,\ Fuel\ A,\ FTP}} = \mathrm{A} + \mathrm{B}*\mathrm{TOG}_{\mathrm{Baseline\ \ fuel,\ FTP}} \qquad (1)$$

A and B are determined as follows:

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

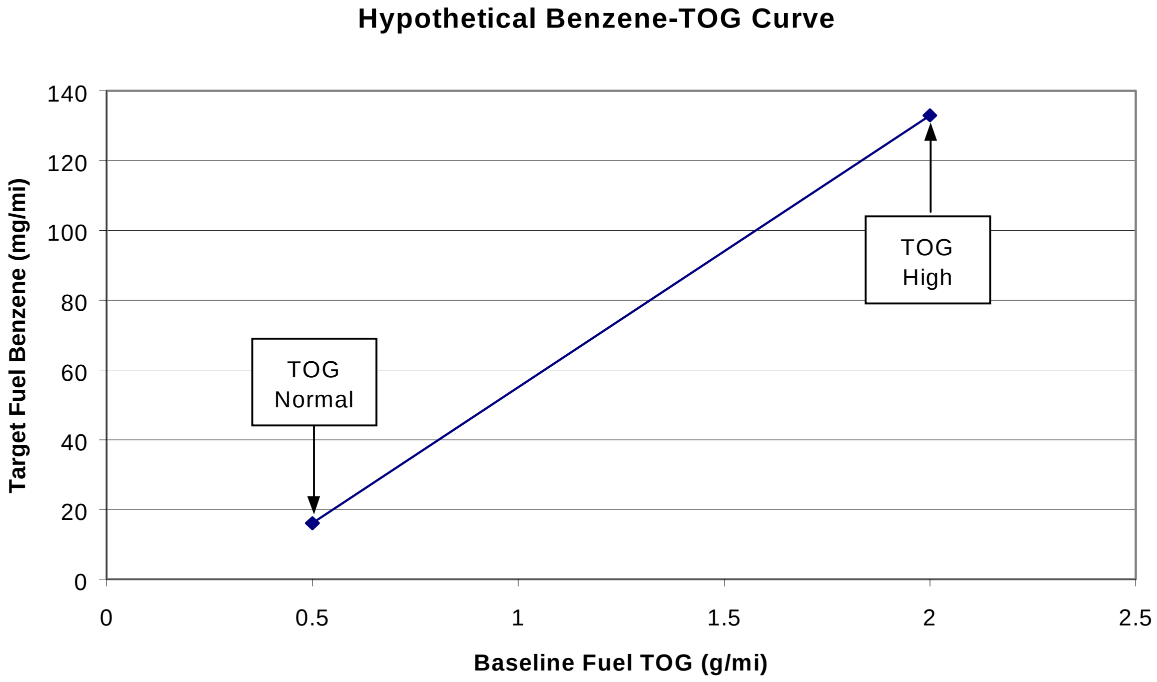
$$\mathrm{B} = (\mathrm{TOX\text{-}H} - \mathrm{TOX\text{-}N})/(\mathrm{TOG\text{-}H} - \mathrm{TOG\text{-}N}) \quad (3)$$

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
TOX-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 noncatalyst 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.[73] 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.[74] 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 Heavy-Duty 2007 Control Scenario

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[73] 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.

[74] 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 proposed 2007 heavy-duty engine rule to further reduce gaseous toxics emissions below the levels that were projected for the Tier 2 scenario. To estimate toxics inventories under the Heavy-Duty 2007 control scenario (described below), we used the light-duty toxic emissions estimates for the Tier 2 scenario from the 1999 Study, and added to these the heavy-duty toxic emissions estimates from the 1999 Study (for the Tier 2 control scenario) adjusted by the ratio of non-methane hydrocarbons (NMHC) emissions under the "control" case compared to NMHC emissions under the "base" case from the 2007 Heavy-Duty Inventory Spreadsheet developed for the proposed heavy-duty 2007 rule.[75] Because benzene and MTBE have an exhaust and an evaporative component, we used the ratio of total NMHC for these compounds; for formaldehyde, acetaldehyde, and 1,3-butadiene, we used the ratio of exhaust NMHC.

We used this combination of sources because the 1999 Study accounts for local conditions, but does not reflect the emissions impacts of the proposed 2007 heavy-duty engine rule, while the HD2007 rule spreadsheet accounts for the effects of the rule, but it does not account for local conditions. So we used the spreadsheet to calculate the percentage reduction due to the rule, and then applied that percentage to the 1999 Study inventory, thereby accounting for the effects of both the rule and local conditions.

b. Projected Emissions Inventories of Selected Gaseous Toxics

In this section we present the emission estimates for the five gaseous MSATs addressed in 1999 EPA Motor Vehicle Air Toxics Study under three control scenarios (benzene, acetaldehyde, formaldehyde, 1,3 butadiene, and MTBE).

The first control scenario is the "Pre-Tier 2" control scenario, which reflects fuels and emission rates assuming all on-highway emission control programs through EPA's national low-emission vehicle (NLEV) program, the reformulated gasoline (RFG) program, and the 2004 heavy-duty diesel engine standards. This scenario does not include implementation of the Tier 2/Sulfur control program.[76] The second control scenario is the "Tier 2" scenario, which includes all the controls in the first scenario, with the addition of Tier 2 controls. The third control scenario is the "Heavy-Duty 2007" scenario, which reflects all the controls in the second scenario with the addition of our recently proposed 2007 heavy-duty engine controls.

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[75] In this analysis, we adjusted the heavy-duty gasoline vehicles (HDGVs) inventory from the spreadsheet model to reflect a 50-state inventory based on California's share of total VMT; in our analysis, the ratio of 50-state VMT to 49-state VMT (excluding California) was 1.133. In addition, we modeled NMHC emissions from HDGVs in California at the same level as NMHC emissions from such vehicles in the rest of the U.S. California has its own standards that apply to HDGVs, but available certification data on Federally-certified HDGVs suggest that these vehicles would also meet the California standards. As a result, we did not attempt to generate unique emission factor estimates for California-certified HDGVs.

[76] The data in this scenario are the "baseline scenario" data from the 1999 Study.

Pre-Tier 2 Control Scenario

Tables IV.B-3 and IV.B-4 present on-highway emission estimates from the 1999 EPA Motor Vehicle Air Toxics Study under the Pre-Tier 2 control scenario. The results of this analysis show that on-highway emissions of the five gaseous MSATs examined are expected to decline by as much as 70 percent by 2020 under this scenario, as compared with 1990 levels. Most of this emission decrease is expected to occur between 1990 and 2007. Between 2007 and 2020, more moderate decreases are observed as VMT growth starts to overtake the fleet-average emission reductions achieved through fleet turnover.

Table IV.B-3
Annual Emissions Summary for Selected Toxics for the Total U.S.
On-Highway Vehicles Only
Pre-Tier 2 Control Scenario*
(thousand short tons per year)

Compound	1990	1996	2007	2020
Benzene	257	165	95	86
Acetaldehyde	41	27	15	15
Formaldehyde	139	80	37	37
1,3-Butadiene	36	22	13	13
MTBE	55	65	24	18

* Note: The Pre-Tier 2 control scenario reflects fuels and emission rates assuming all on-highway emission control programs through EPA’s national low-emission vehicle (NLEV) program, reformulated gasoline (RFG) program, and the 2004 heavy-duty diesel engine standards.

Table IV.B-4
Annual Emissions Summary for Selected Toxics for the Total U.S.
On-Highway Vehicles Only
Pre-Tier 2 Control Scenario*

	Cumulative Percent Reduction from 1990		
Compound	1996	2007	2020
Benzene	36%	63%	67%
Acetaldehyde	33%	62%	63%
Formaldehyde	42%	73%	73%
1,3-Butadiene	39%	65%	65%
MTBE	-18%	57%	67%

* Note: The Pre-Tier 2 control scenario reflects fuels and emission rates assuming all on-highway emission control programs through EPA's national low-emission vehicle (NLEV) program, reformulated gasoline (RFG) program, and the 2004 heavy-duty diesel engine standards.

Tier 2 Control Scenario

Tables IV.B-5 and IV.B-6 present the on-highway inventories of these five gaseous toxics under the Tier 2 control scenario, which assumes all the controls from the Pre-Tier 2 control scenario, plus Tier 2 controls.[77] While the Tier 2 rule is primarily designed for ozone and PM control, it will also result in important toxics reductions.

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[77] These tables reflect control scenario #7 from the 1999 Study, which reflects Tier 2 controls and also heavy-duty gasoline vehicle controls proposed for 2004.

Table IV.B-5
Annual Emissions Summary for Selected Toxics for the Total U.S.
On-Highway Vehicles Only
Tier 2 Control Scenario
(thousand short tons per year)

Compound	1990	1996	2007	2020
Benzene	257	165	86	67
Acetaldehyde	41	27	15	13
Formaldehyde	139	80	36	33
1,3-Butadiene	36	22	11	10
MTBE	55	65	25	18

Table IV.B-6
Annual Emissions Summary for Selected Toxics for the Total U.S.
On-Highway Vehicles Only
Tier 2 Control Scenario

	Cumulative Percent Reduction from 1990		
Compound	1996	2007	2020
Benzene	36%	67%	74%
Acetaldehyde	33%	64%	68%
Formaldehyde	42%	74%	76%
1,3-Butadiene	39%	69%	72%
MTBE	-18%	54%	67%

Heavy-Duty 2007 Control Scenario

Tables IV.B-7 and IV.B-8 illustrate the on-highway emission estimates we project under the Heavy-Duty 2007 control scenario, which assumes all the controls from the Tier 2 control scenarios, plus the recently proposed heavy-duty 2007 controls.

Table IV.B-7
Annual Emissions Summary for Selected Toxics for the Total U.S.
On-Highway Vehicles Only
Heavy-Duty 2007 Control Scenario
(thousand short tons per year)

Compound	1990 Emissions	1996 Emissions	2007 Emissions	2020 Emissions
Benzene	257	165	86	64
Acetaldehyde	41	27	14	7
Formaldehyde	139	80	35	17
1,3-Butadiene	36	22	11	9
MTBE	55	65	25	18

Table IV.B-8
Annual Emissions Summary for Selected Toxics for the Total U.S.
On-Highway Vehicles Only
Heavy-Duty 2007 Control Scenario

	Cumulative Percent Reduction from 1990		
Compound	1996	2007	2020
Benzene	36%	67%	75%
Acetaldehyde	33%	65%	82%
Formaldehyde	42%	75%	87%
1,3-Butadiene	39%	69%	75%
MTBE	-18%	54%	67%

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 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 the emissions inventories constructed for the Tier 2 rulemaking, updated to reflect more recent information. Light-duty VOC emissions were calculated for the Tier 2/Sulfur Air Quality Inventory by applying adjustment factors to county MOBILE5 runs. These adjustment factors were developed for all combinations of I/M (I/M and no I/M), fuel types (RFG, conventional gasoline, geographic phase-in area), and vehicle type. This procedure uses MOBILE5 to account for local conditions and the adjustment factors to incorporate improved emission factors. The Tier 2/Sulfur Air Quality Inventory was generated for 1996, 2007, and 2030.

We subsequently further improved our understanding of light-duty vehicle and truck emissions. These improvements are reflected in the Tier 2/Sulfur Updated Inventory, a nationwide inventory that does not fully account for local inputs. Results from this updated inventory were also presented in the Tier 2 final rule.

For this proposal, we updated the Tier 2/Sulfur Air Quality Inventory to account for additional improvements in our understanding of light-duty vehicle and truck emissions and to better account for county-specific conditions. These updates were applied by multiplying emissions by county and vehicle type in the Tier 2/Sulfur Air Quality Inventory by the ratio of the improved emission factors to those used to generate the Tier 2/Sulfur Air Quality Inventory. Details of this method can be found in a memorandum by Harvey Michaels to Docket A-2000-12 titled, "Adjustments to the Tier 2 Air Quality Inventory for the Mobile Source Air Toxics Proposed Rule."

We also replaced the Tier 2 heavy-duty inventory estimates with inventory estimates from the national emissions inventory spreadsheet developed and used for our proposed 2007 heavy-duty engine rule.[78] These heavy-duty engine emission estimates are based on national average fleet mixes, temperatures, VMT distributions by roadway type, and speed by roadway type. We did 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. As is the case for the gaseous toxics Heavy-Duty 2007 control scenario described above, we expect to use the heavy-duty engine emissions inventories developed for the

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[78] This spreadsheet model can be found in EPA Air Docket A-99-06, Item II-B-31. In this analysis, we adjusted the heavy-duty gasoline vehicles (HDGVs) inventory from the spreadsheet model to reflect a 50-state inventory based on California's share of total VMT; in our analysis, the ratio of 50-state VMT to 49-state VMT (excluding California) was 1.133.

final 2007 heavy-duty engine rule for our final toxics rulemaking. These inventories will integrate our most recent information on emissions factors with county-specific modeling inputs.

For 1990, we could not use the Tier 2 rulemaking inventories, since they do not extend to 1990. We also 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 from the Tier 2 rule, because EPA's 1990 Trends estimates have not been updated to reflect the data and analyses that will be used in MOBILE6. For 1990, we modified the modeling methods applied in the 1999 Air Toxics 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.[79]

b. Projected VOC Emissions Inventory

The results of this analysis, presented in Table IV.B-9, show that on-highway VOC inventories are projected to decrease by over 70 percent between 1990 and 2020 under the Tier 2 control scenario. We assume that other gaseous toxics will decrease by approximately 70 percent as well. Most of the emission decrease is expected to occur before 2007.

Table IV.B-9
Annual VOC Emissions Summary for the Total U.S.
On-Highway Vehicles Only
Tier 2 Control Scenario

	1990	1996	2007	2020
Thousand short tons per year	7,585	4,819	2,673	2,061
Cumulative Annual Reductions from 1990 (thousand short tons)	N.A.	2,766	4,912	5,525
Cumulative Percent Reductions from 1990	N.A	36%	65%	73%

Table IV.B-10 reflects our on-highway inventory estimates for VOC under the Heavy-Duty 2007 control scenario, and indicates that VOC inventories from on-highway vehicles are projected to decrease 75 percent between 1990 and 2020 under this scenario. We assume that other gaseous toxics would decrease by approximately 75 percent as well.

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[79] 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.

Table IV.B-10
Annual VOC Emissions Summary for the Total U.S.
On-Highway Vehicles Only
Heavy-Duty 2007 Control Scenario

	1990	1996	2007	2020
Thousand short tons per year	7,585	4,819	2,662	1,838
Cumulative Annual Reductions from 1990 (thousand short tons)	N.A.	2,765	4,924	5,748
Cumulative Percent Reductions from 1990	N.A.	36%	65%	76%

4. Diesel PM Inventory

This section describes how we derived diesel PM estimates for this rule, and then presents those estimates.

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.[80] These estimates account for county-specific inputs in a more reliable way than our 1999 Study.[81]

For 1996 and later years, light-duty diesel PM emissions were taken from our Tier 2 Air Quality Analysis Inventory. These estimates were 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. We have determined that our Tier 2 Air Quality Analysis Inventory is a more appropriate source for national light-duty diesel PM emission estimates than the 1999 Study since it better accounts for county-specific conditions.

To estimate heavy-duty exhaust diesel PM emissions for 1996 and later years, we used

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[80] 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.

[81] The Air Toxics 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.

the national emissions inventory spreadsheet developed and used for our proposed 2007 heavy-duty engine rule.[82] This spreadsheet incorporates recent findings on heavy-duty diesel engine PM emissions that were not reflected in the 1999 Motor Vehicle Air Toxics Study, accounts for the increasing prevalence of diesel engines in certain engine classes, and is capable of modeling the effects of the proposed 2007 heavy-duty engine rule. We did not include crankcase PM emissions in our estimates of diesel PM, since they are not part of diesel exhaust. The spreadsheet model is not capable of producing heavy-duty diesel PM estimates for 1990.

For the final toxics rule, we expect to revise our diesel PM inventory based on the analyses being conducted for the final 2007 heavy-duty engine rule. These analyses will include our most current information on light- and heavy-duty diesel engine emissions factors and VMT, and will also reflect county-by-county information on VMT distribution by vehicle class, roadway type, and speed.

b. Projected Diesel PM Emissions Inventory

Our diesel PM inventory estimates for the Tier 2 control scenario are presented below in Table IV.B-11. Diesel PM emissions are expected to decline by over 60 percent under the Tier 2 control scenario, as compared with 1990 levels. These emissions are expected to decline even more, by almost 95 percent as compared with 1990 levels, under the Heavy-Duty 2007 control scenario, as shown in Table IV.B-12.

Table IV.B-11
Annual Diesel PM Emissions Summary for the Total U.S.
On-Highway Vehicles Only
Tier 2 Control Scenario

	1990	1996	2007	2020
Thousands of short tons per year	235	180	93	88
Cumulative Annual Reduction from 1990 (thousand short tons)	N.A	55	142	147
Cumulative Percent Reduction from 1990	N.A.	23%	60%	63%

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[82] This spreadsheet model can be found in EPA Air Docket A-99-06, Item II-B-31.

Table IV.B.12
Annual Diesel PM Emissions Summary for the Total U.S.
On-Highway Vehicles Only
Heavy-Duty 2007 Control Scenario

	1990	1996	2007	2020
Thousands of short tons per year	235	180	82	15
Cumulative Annual Reduction from 1990 (thousand short tons)	N.A.	55	153	220
Cumulative Percent Reduction from 1990	N.A.	23%	65%	94%

C. Limitations and Uncertainties in the Analyses

While the 1999 EPA Motor Vehicle Air Toxics Study and our modeling analyses for VOC and diesel PM emissions for this rule are the most recent information we have to project MSAT emissions inventories for future years, we are aware of limitations and uncertainties in these sources. In this section, we describe these limitations and uncertainties.

1. 1999 EPA Motor Vehicle Air Toxics Study

There are a number of limitations and uncertainties associated with the emissions modeling in the analyses in the 1999 EPA Motor Vehicle Air Toxics Study. First, the VMT distribution by vehicle type we used in the 1999 Study differ from what we plan to use in MOBILE6, and what we used to develop criteria pollutant inventory estimates in our recent 2007 heavy-duty proposed rule. Table IV.C-1 below presents the two sets of VMT fractions. The VMT fractions that are planned for MOBILE6 will result in a greater proportion of total fleet VMT allocated to heavy-duty vehicles and a correspondingly smaller proportion of fleet VMT allocated to light-duty gasoline vehicles. Table IV.C-2 presents toxic emission factors for 1996, 2007, and 2020, under the Tier 2 control scenario. These data suggest that the VMT shift from gasoline- to diesel-powered vehicles should have little impact on fleet average emissions of benzene and 1,3-butadiene, but will cause fleet average emissions of acetaldehyde and formaldehyde to increase by about 10 percent. It should be noted however, that diesels emit lower amounts of VOC than gasoline engines; since VOCs react in the atmosphere to form aldehydes, the reduction in VOC emissions should result in reduced levels of secondary aldehydes.

Table IV.C-1
VMT Fractions used in the 1999 EPA Motor Vehicle Air Toxics Study Compared with proposed fractions for MOBILE6

VMT Fractions									
Year	Source	LDGV	LDGT1	LDGT2	HDGV	LDDV	LDDT	HDDV	MC
1996	Toxics Assessment	0.556	0.269	0.089	0.021	0.003	0.002	0.055	0.005
1996	Planned MOBILE6	0.565	0.233	0.080	0.035	0.003	0.002	0.077	0.005
2007	Toxics Assessment	0.395	0.383	0.127	0.023	0.000	0.002	0.065	0.005
2007	Planned MOBILE6	0.388	0.361	0.124	0.036	0.000	0.001	0.085	0.005
2020	Toxics Assessment	0.300	0.448	0.146	0.025	0.000	0.005	0.071	0.005
2020	Planned MOBILE6	0.279	0.440	0.152	0.036	0.000	0.001	0.088	0.004

Notes: LDGV = light-duty gasoline vehicles, LDGT1 = light-duty gasoline truck 1 (up to 6,000 pounds gross vehicle weight, LDGT2 = light-duty gasoline truck 2 (6,001 to 8,500 pounds gross vehicle weight), HDGV = heavy-duty gasoline vehicle, LDDV = light-duty diesel vehicle, LDDT = light-duty diesel truck, HDDV = heavy-duty diesel vehicle, MC = motorcycle.

Table IV.C-2
U.S. Annual average toxic emission factors (mg/mi) from the 1999 EPA Motor Vehicle Air Toxics Study

Emission Factors (mg/mi)									
Year	Pollutant	LDGV	LDGT1	LDGT2	HDGV	LDDV	LDDT	HDDV	MC
1996	Benzene	53.94	65.13	90.40	161.28	16.67	25.81	17.52	88.81
	Acetaldehyde	6.05	7.87	12.37	32.74	10.25	15.83	48.06	18.84
	Formaldehyde	16.26	22.59	37.53	154.83	32.18	49.66	130.49	68.60
	1,3-Butadiene	6.32	7.77	13.13	29.85	7.50	11.58	10.18	26.27
2007*	Benzene	20.46	25.15	39.28	54.82	4.45	7.78	9.38	74.41
	Acetaldehyde	1.96	2.51	4.18	9.10	2.74	4.78	25.73	17.99
	Formaldehyde	4.03	5.35	9.08	32.54	8.58	15.02	69.85	59.87
	1,3-Butadiene	2.31	3.07	4.68	5.29	2.00	3.50	5.45	23.30
2020*	Benzene	13.07	15.18	20.75	24.98	2.22	2.37	8.25	74.48
	Acetaldehyde	1.18	1.37	1.91	3.33	1.36	1.45	22.61	18.01
	Formaldehyde	2.47	2.90	3.90	10.39	4.28	4.57	61.39	59.73
	1,3-Butadiene	1.69	2.04	2.79	1.43	1.00	1.06	4.79	23.37
* 2007 and 2020 estimates reflect the Tier 2 control scenario									

Notes: LDGV = light-duty gasoline vehicles, LDGT1 = light-duty gasoline truck 1 (up to 6,000 pounds gross vehicle weight, LDGT2 = light-duty gasoline truck 2 (6,001 to 8,500 pounds gross vehicle weight), HDGV = heavy-duty gasoline vehicle, LDDV = light-duty diesel vehicle, LDDT = light-duty diesel truck, HDDV = heavy-duty diesel vehicle, MC = motorcycle.

Second, in estimating toxic emissions in tons per year, VMT estimates from the Emission Trends Database were used. These data represent EPA's best estimate of VMT, but in some instances, States may have more accurate VMT data.

Third, our emission modeling also assumed an average vehicle speed of 19.6 miles per hour. Actual vehicle speeds will tend to vary considerably within and between areas. Since the relationship between emissions and speed distribution is complex, actual toxics emissions are likely to differ from the toxics emissions estimates based on a 19.6 mph vehicle speed.

Fourth, there are significant uncertainties in projecting emissions for future years under various control scenarios. For instance, the future year VMT mix among vehicle classes was based on sales projections. Actual sales may differ from these projections. Our projections of fuel properties in future years are also subject to considerable uncertainty.

Fifth, our emissions estimates are sensitive to assumptions about compliance margins, in-use deterioration, and toxics fractions. We explored some of these parameters in the 1999 EPA

Motor Vehicle Air Toxics Study.

2. VOC Inventory Modeling

Our 1990 VOC emissions estimate was derived using the 1999 Study methodology, hence it has the same uncertainties and limitations as those described above.

Our light-duty vehicle VOC emissions estimates for 1996 and later years have several notable sources of uncertainty. First, it treats California the same as the rest of the country, which results in an overestimate of VOC emissions in 1996 and 2007. Second, our light-duty VOC estimates do not account for the changes in VMT distribution between light-duty and heavy-duty described in the previous section, which results in an approximately three percent overestimate in light-duty vehicle VOC emissions. Finally, our light-duty vehicle VOC estimates do not account for county-specific inputs directly, nor are they based on explicit calculations of projected emissions in 2020. Instead, we attempted to adjust the county-specific inventories generated for the Tier 2 Air Quality Analysis to account for more recent information about light-duty vehicle emissions for 1996, 2007, and 2030; we then used an interpolation method to generate inventory estimates for 2020. Both the adjustment approach and the interpolation method introduce uncertainty in our results.

Our heavy-duty VOC estimates for 1996 and later years are based on the spreadsheet developed for the 2007 heavy-duty engine proposed rule. This spreadsheet is designed to generate national emissions estimates that do not fully account for local inputs. It is based on national average fleet mixes, temperatures, VMT distributions by roadway type, and speed by roadway type. It also does not account for the differences between Federal and California emission standards for heavy-duty gasoline vehicles or between Federal and California diesel fuel sulfur standards. While we believe these differences are small, we expect to account for them more explicitly and completely in our final rule.

3. Diesel PM Inventory Modeling

Our 1990 diesel PM inventory is taken from EPA’s Emission Inventory Trends Report. This estimate may not fully account for the shift from gasoline to diesel engines in medium-heavy-duty vehicles because it is based on 1987 data regarding diesel engine use. It should be noted that the 1999 EPA Motor Vehicle Air Toxics Study analysis has the same limitation while also not reflecting local inputs as fully.

For 1996 and later years, we based our light-duty vehicle diesel PM estimates on the Tier 2 Air Quality Analysis Inventory. This inventory does not account for the changes in VMT distribution between light-duty and heavy-duty vehicels, which results in an approximately three percent overestimate in light-duty vehicle diesel PM emissions. Furthermore, our light-duty vehicle diesel PM emissions estimates are based on the PART5 model, which assumes that PM emissions do not increase over time due to engine wear or use. However, PM emissions from diesel engines may well increase over time as a result of deterioration. We are currently evaluating diesel PM emission estimates for PART6.

Our heavy-duty vehicle diesel PM estimates for 1996 and later years are based on the spreadsheet developed for the 2007 heavy-duty engine proposed rule. This spreadsheet is designed to generate national emission estimates and incorporates recent findings on heavy-duty vehicle diesel PM emissions that were not reflected in the 1999 EPA Motor Vehicle Air Toxics Study, accounts for the increasing prevalence of diesel engines in certain engine classes, and is capable of modeling the effects of the proposed 2007 heavy-duty engine rule. It is based on national average fleet mixes, temperatures, VMT distributions by roadway type, and speed by roadway type. It also does not account for the differences between Federal and California diesel fuel sulfur standards. While we believe these differences are small, we expect to account for them more explicitly and completely in our final rule.

And while our spreadsheet model does incorporate some degree of PM emissions deterioration over time, real-world diesel PM emissions from diesel engines may increase over time to a considerably greater extent. Also, there could be more idling and hard acceleration in the real world than in the current heavy-duty vehicle test cycle used to determine compliance with engine emission standards; this could also result in an underestimate of real world PM emissions. One reviewer[83] suggested that PART5 may underestimate heavy-duty diesel vehicle emissions by up to 50 percent. EPA is currently evaluating diesel PM emission estimates for PART6.

For the final toxics rule, we expect to revise our diesel PM inventory based on the analyses being conducted for the 2007 heavy-duty engine rule. These analyses will include our most current information on light- and heavy-duty diesel engine emissions factors and VMT, and will also reflect county-by-county information on VMT distribution by vehicle class, roadway type, and speed.

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[83] DeLucchi, M. A. 1999. Analysis of Particulate Matter Emission Factors in the PART5 Model. Submitted to EPA.

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 for gaseous air toxics are compared to estimates of the on-highway vehicle contribution to modeled ambient concentrations. As exposure estimates via a different approach are not yet available for comparison, the NATA national scale assessment modeled ambient concentration estimates (which will be used to prepare exposure estimates later summer) are used to evaluate the reasonableness of the exposure estimates. We will also discuss what we know about inhalation exposures in various micro-environments. As discussed in Chapter 4, we have the most reliable inventory data for these five compounds, and hence the most accurate modeled mobile source estimates of ambient concentrations and exposure.

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

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

Monitor data for air toxics are somewhat limited, but are 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.

1. Ambient Monitoring

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.

a. EPA Monitoring Data

This section summarizes monitored air toxics concentration data from the EPA

Aerometric Information Retrieval System (AIRS), Air Quality System.[84] 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 great deal of variability in the numbers. In addition, differences in criteria used to select the sites of monitors may also result in discrepancies in the significance of different measurements. For example, many monitors are placed at sites where readings are expected to be high. Conversely, other monitors might be sited away from areas of highest concentration (for instance to measure concentrations in residential areas). These differences make it difficult to interpret these results. 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.

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.[1] 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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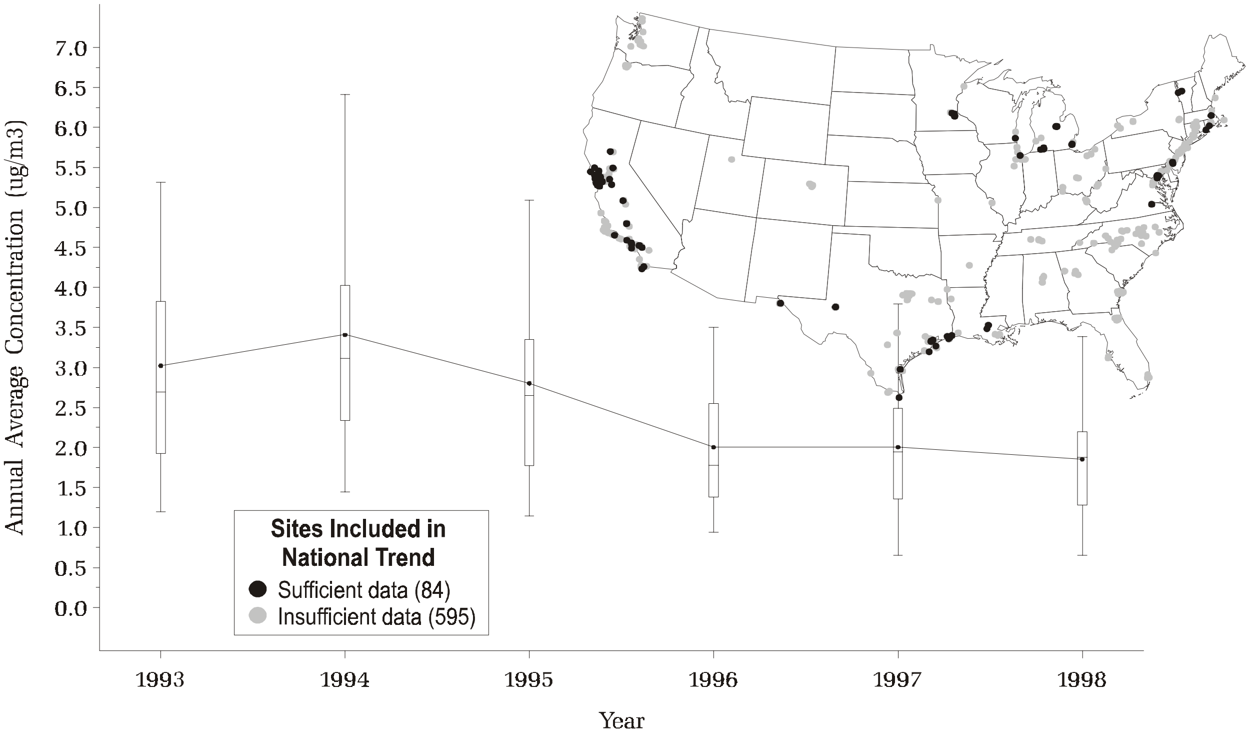
[84] The AIRS Air Quality System contains measurements of ambient concentrations of air pollutants and associated meteorological data. The data is collected by thousands of monitoring stations operated by EPA, national, state and local agencies. EPA uses this data to assess the overall status of the 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. (µg/m^3)	Standard deviation	95th percentile conc. (µg/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.[2, 3] 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 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.[4] [5] [6] [7] 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 Emissions*[8] 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 monitoring diesel PM 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.

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

Location	Year of Sampling	Diesel PM$_{10}$ & PM$_{2.5}$ μg/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[9]	1982, annual	11.6	36%	
Phoenix area, AZ[10]	Winter,1989-90	4-22*	†	
Phoenix, AZ[11]	1994-95, annual	0-5.3 (2.4)	0-27%	
California, 15 Air Basins[12]	1988-92, annual	0.2-3.6*	†	
Manhattan, NY[13]	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[14]	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[15]	1995, annual	0.2-0.5 (0.3)	1-5%	
Washington, DC[16]	1992-1995, annual	1.3-1.8 (1.6)	6-10%	
South Coast Air Basin[1]	1995-1996, annual	2.4-4.7‡	†	

*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.

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.[17] 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.

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.

Thus, 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.

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 (µg/m^3)

Compound	Conc. (µg/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. Preliminary National Air Toxics Assessment Results for Mobile Sources

As part of its National Air Toxics Assessment (NATA) activities, EPA is currently conducting 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 UATS, plus diesel PM. The NATA national scale assessment will report average ambient concentrations nationally and at the county level, as well as average concentrations for counties at or above the 95th percentile. 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 will also apportion the contribution to ambient concentrations between major, area, nonroad mobile, and on-highway sources. Draft results will be released later this year, in conjunction with a review by the Scientific Advisory Board. Estimates of the mobile source contributions have been developed in advance of the estimates for stationary sources.[18] Table V.A-5 presents draft mean and median nationwide gaseous toxic ambient concentrations attributable to on-highway and nonroad mobile sources.[85] 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. Concentrations for other mobile source toxics estimated using ASPEN can be found in Appendix 1. Diesel PM estimates are being revised to account for inventory

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

changes and will be included in the release for EPA Science Advisory Board review.

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. (µg/m^3)		Nonroad Contribution to Ambient Conc. (µg/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

c. Diesel PM Estimates

Two dispersion model studies reporting diesel PM have been conducted in Southern California. Results are summarized in Table V.A-6. Secondary formation of particulate matter accounted for 27% to 67% of the total particulate matter associated with diesel engines.[19] [20] 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-6
Annual average diesel particulate matter concentrations predicted from dispersion modeling

Location	Year of Sampling	Diesel PM$_{2.5}$ μg/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[21]	1982, annual	3.8**	16%
Claremont, CA[22]	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[23]	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), which is currently under development. 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).

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".[24]

Other than these two efforts, the only available estimates of inhalation exposure to motor vehicle related air toxics were developed using the Hazardous Air Pollutant Exposure Model for Mobile Sources, version 3 (HAPEM-MS3).[25, 26, 27] 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.

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.

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.[28] Based on peer review comments, a number of improvements were made to the model, 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 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 using paired ambient and personal exposure monitor measurements from CO studies in Denver and Washington.[29, 30] In our modeling, we set the additive term to zero, to eliminate non-ambient sources of CO, such as gas stoves. 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.

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.[31, 32] This scaling 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:

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

where:

$\text{TOX}_{\text{Exposure}(\mu g/m3)}$ = exposure to on-highway vehicle toxic emissions
$\text{CO}_{\text{Exposure}(\mu g/m3)}$ = exposure to on-highway vehicle CO emissions
$\text{CO}_{\text{EF}(g/mi)}$ = CO emission factor
$\text{TOX}_{\text{EF}(g/mi)}$ = 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.[33] 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, diesel PM emission estimates for 1996, 2007, and 2020 developed for the 2007 heavy-duty engine proposed rule are higher than estimates in the EPA 1999 Study. 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.[34] 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. Planned Improvements to HAPEM

As mentioned previously, a new version of HAPEM is being developed for use in the National Air Toxics Assessment. This new version will have a number of major modifications and improvements. The model has been revised to accept monitored or modeled toxics concentrations directly as input, rather than using CO as a surrogate for toxics. Thus the mobile source acronym (-MS) was dropped. The model also now incorporates a new time-activity database derived from the CHAD (Comprehensive Human Activity Database). Draft results of modeling with HAPEM4 for 1996 should be available later in 2000.

2. Comparison of Exposure Modeling Results to Modeled Ambient Concentrations

In this section, we compared HAPEM-MS3 exposure modeling results to modeled ambient concentrations from the Cumulative Exposure Project and NATA national scale assessment.[86] 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 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

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

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.

Until the NATA HAPEM4 results are available, however, it is difficult to make a strong inference on whether NATA exposures are likely to be very close to HAPEM-MS3 exposures. This will depend on the micro-environmental factors selected and the activity data from the CHAD. Nonetheless, 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 (µg/m^3)	CEP Ambient Conc. (µg/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 µg/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 (μg/m^3)	NATA On-Highway Vehicle Mean Ambient Concentration (μg/m^3)
Benzene	0.68	0.55
1,3-Butadiene	0.07	0.05
Formaldehyde	0.34	0.38
Acetaldehyde	0.12*	0.40

*Unadjusted estimate based on direct emissions – adjusted level which includes secondary formation is 0.36 μg/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 twice 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.

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 are 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.

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

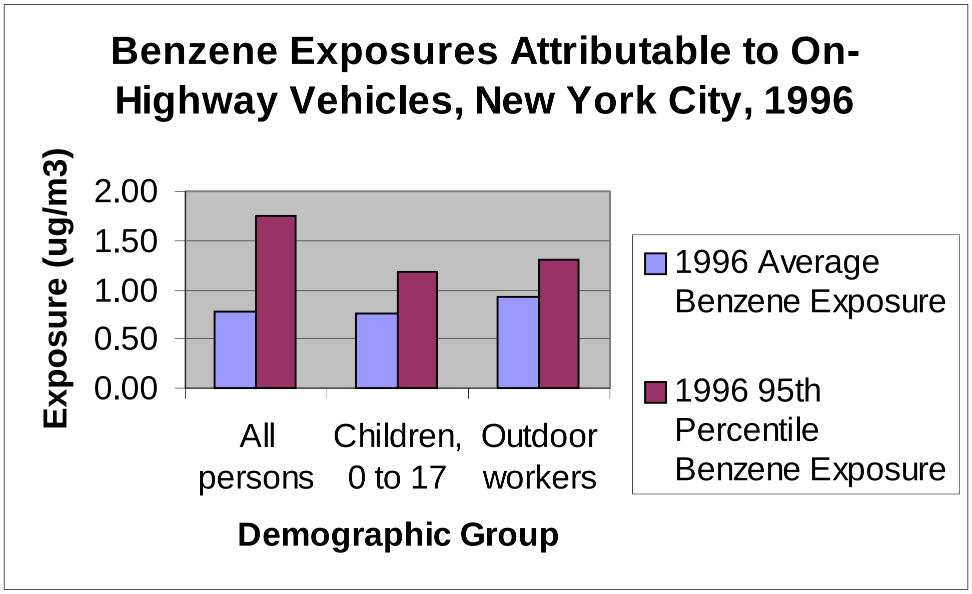


Table V.B-4
Highway vehicle portion of nationwide average inhalation exposures to benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and diesel PM for three demographic groups in 1996, based on HAPEM-MS3

Pollutant	Overall Population Exposure (µg/m^3)	Outdoor Worker Exposure (µg/m^3)	Children's Exposure (µg/m^3)
Benzene	0.68	0.79	0.65
1,3-Butadiene	0.07	0.08	0.07
Formaldehyde	0.34	0.39	0.32
Acetaldehyde	0.36	0.39	0.33
Diesel PM	0.73	0.85	0.70

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 µg/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. Minnesota 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	0.84
Chicago	0.48
Denver	0.71
Houston	0.53
Minneapolis	1.11
New York	0.79
Philadelphia	0.51
Phoenix	1.26
Spokane	0.91
St. Louis	0.47
Urban Area Average	0.74
Rural Area Average	0.43

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	0.87	0.62	0.53	0.71
Acetaldehyde	0.47	0.30	0.25	0.36
Formaldehyde	0.45	0.30	0.26	0.34
1,3-Butadiene	0.12	0.06	0.03	0.07
Diesel PM	0.85	0.71	0.63	0.73

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. 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, and the 2004 heavy-duty standards. With current 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 55 percent. Standards recently proposed for heavy-duty engines in 2007 will result in additional exposure reductions, as shown in Figure V.B-2. With these standards, diesel PM exposure will be reduced by 93% from 1990 levels.

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.

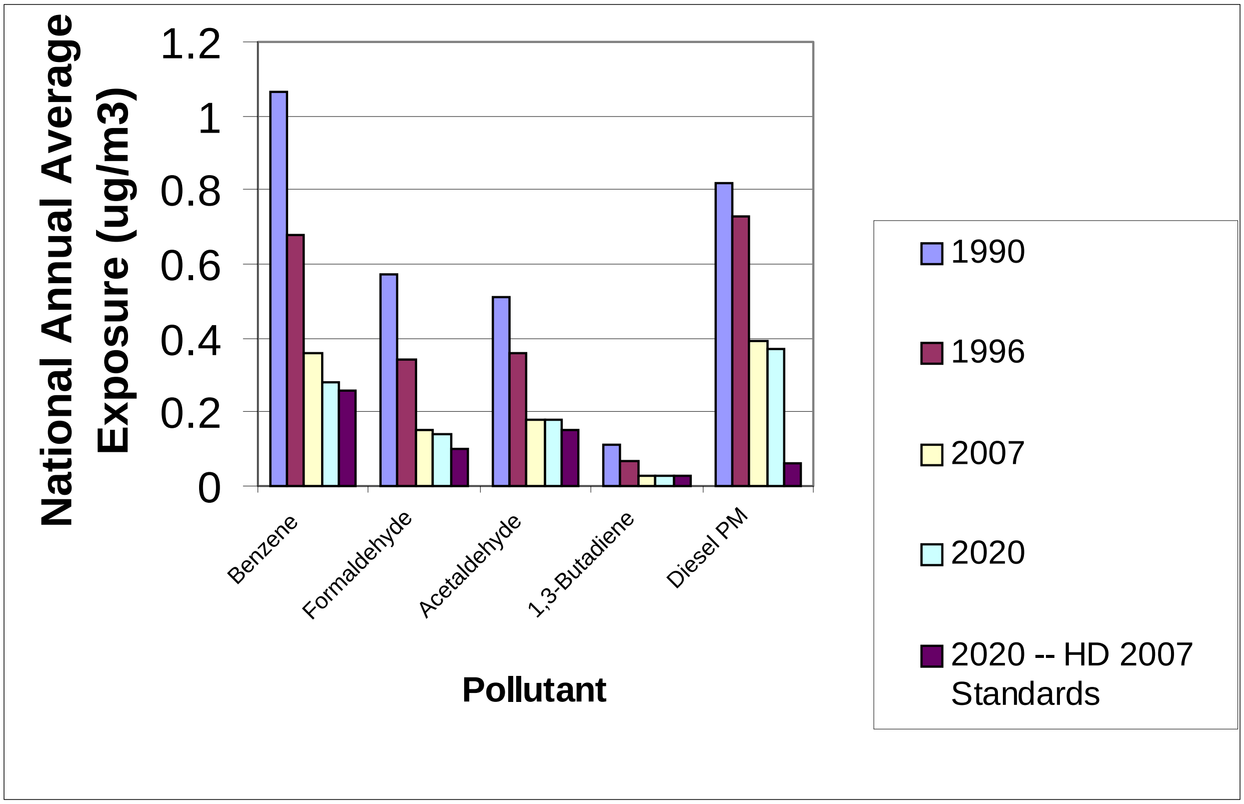
1) ***Representativeness of, and Variability in, Measured Ambient Carbon Monoxide Levels.*** Toxics exposure estimates are based on measured ambient CO concentrations averaged over 10 selected urban areas. Important limitations of these data relate to variations in CO levels both within and among urban 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 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.

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)

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.[35]

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 sources within micro-environments is large, estimates of these 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. 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 μg/m^3), near roadways with diesel traffic (diesel PM concentrations are calculated to be 0.7-7.5 μg/m^3 higher than background), and in schools (0.9-5.5 μg/m^3).

Recently, elemental carbon measurements were reported for enclosed vehicles driving on Los Angeles roadways.[36] Applying a ratio of diesel PM mass to elemental carbon mass, diesel PM concentrations in the vehicle ranged from approximately 2.8 μg/m^3 to 36.6 μg/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.[37] 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 μg/m^3 to 4.0 μg/m^3 excess diesel PM above background concentrations with a maximum 24 hour measurement of 7.5 μg/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.[38] 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 μg/m^3 to 6 μg/m^3, with an average between 3 and 4 μg/m^3. These results correspond to diesel PM concentrations ranging from 0.9 μg/m^3 to 5.5 μg/m^3 with a mean of approximately 2.7-3.7 μg/m^3. Total PM2.5 concentrations inside the schools averaged 23.0 μg/m^3 while PM2.5 outside was only slightly higher (24.8 μg/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,[39] Commuter's Exposure to Volatile Organic Compounds, Ozone, Carbon Monoxide, and Nitrogen Dioxide,[40] In-Vehicle Air Toxics Characterization Study in the South Coast Air Basin,[41] Air Toxics Micro-environment Exposure and Monitoring Study,[42] a 1998 California EPA study of in-vehicle concentrations (California EPA, 1998), a 2000 study of commuter exposures in Detroit, MI,[43] and a 1993 NIOSH study of concentrations at service stations.[44]

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 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 nonindustrial park were sampled for in-vehicle concentrations of 14 toxic air pollutants, carbon monoxide, and lead.

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.

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.[45] 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.[46] 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).[47] 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 $\mu g/m^3$.

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

Pollutant	Highway Contribution to Ambient Conc. (µg/m^3)		Nonroad Contribution to Ambient Conc. (µg/m^3)	
	Mean	Median	Mean	Median
1,3-Butadiene	0.05	0.04	0.02	0.01
Acetaldehyde	0.40	0.32	0.27	0.12
Acrolein	0.05	0.04	0.04	0.02
Arsenic Compounds	6.5E-07	4.2E-07	7.7E-06	9.1E-08
Benzene	0.55	0.45	0.24	0.16
Chromium Compounds	4.2E-05	2.8E-05	1.4E-04	4.5E-05
Dioxins/Furans	2.9E-10	not estimated	2.0E-10	not estimated
Ethylbenzene	0.32	0.23	0.11	0.07
Formaldehyde	0.38	0.29	0.48	0.20
Lead Compounds	5.9E-05	3.9E-05	3.4E-03	4.1E-04
Manganese Compounds	1.7E-05	1.1E-05	1.4E-04	5.1E-05
MTBE	0.44	0.04[1]	0.29	0.00[1]
n-Hexane	0.24	0.18	0.08	0.05
Nickel Compounds	3.3E-05	2.2E-05	3.2E-04	3.3E-05
POM (as sum of 7-PAH)	1.2E-04	8.8E-05	2.6E-05	1.7E-05
Styrene	0.04	0.03	0.005	0.003
Toluene	2.18	1.55	0.43	0.29
Xylenes	1.20	0.85	0.40	0.26

[1] Nationwide median values are much lower than mean values because a limited number of areas in the country use MTBE in non-premium gasoline as part of the Federal Reformulated Gasoline Program, California Reformulated Gasoline Program, or Winter Oxygenated Gasoline Program.

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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 proposed determination that it is appropriate that additional motor vehicle-based controls (beyond those already adopted or proposed) not be proposed at this time under §202(l)(2). This is based on considerations of the technical feasibility and cost of proposing 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 and their control it is helpful to categorize them into three groups: gaseous organic toxics, 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 is discussed below and in Chapter 4, these VOC controls have contributed to large reductions in gaseous mobile source air toxics (MSATs). Similarly, other EPA controls have had major impacts on the non-gaseous MSATs as well.

1. Gaseous Organic Toxics

Fourteen of the 21 proposed MSATs are gaseous organics. These 14 gaseous toxics can be further categorized depending on whether they are fuel components or combustion products. Those that are gasoline fuel components are 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
1,3-Butadiene		YES
Formaldehyde		YES
Acrolein		YES
Dioxin/Furans		YES
POM	YES	YES
Styrene		YES
Benzene	YES	YES
MTBE	YES	YES
Ethylbenzene	YES	YES
n-Hexane	YES	YES
Naphthalene	YES	YES
Toluene	YES	YES
Xylene	YES	YES

Vehicle-based controls are effective in reducing all 14 of the gaseous air toxics. The toxics that are fuel components 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 that control of exhaust VOC emissions in general. The Agency is not aware of any exhaust emission controls that 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.[87] 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[88]
Acetaldehyde	0.5 %
1,3-Butadiene	0.5 %
Formaldehyde	1 %
Benzene	4 %
Toluene	10 %
Other Gaseous MSATs	9 %

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 is 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 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

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[87] 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.

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

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.

As noted above, manufacturers are 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).[89] 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. However, there are technological, implementation, and cost issues that still need to be addressed.

Thermally insulated catalysts maintain sufficiently high catalyst temperatures by surrounding the catalyst with an insulating vacuum. Prototypes of this technology have demonstrated the ability to store heat for more than 12 hours.[90] 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.[91] These systems require a modified catalyst, as well as an upgraded battery and charging

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

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

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

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 lead to much more 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[92]
Acetaldehyde	0 %
1,3-Butadiene	0 %
Formaldehyde	0 %
Benzene	2 %
Toluene	4 %
Other MSATs	5 %

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.

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,

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

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 which 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 Rulmaking.

2. Diesel Exhaust

Diesel PM consists of 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 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 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.[93, 94]

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

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[93] 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.

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

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 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. Yet when tested on the US Heavy-Duty Federal Test Procedure (HD FTP), they demonstrated PM reductions in excess of 90 percent.[95] 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.[96] 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

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

[96] 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.

Mobile source toxics included 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.

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 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 that 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 are projected to be feasible without using HC traps or electrically heated catalysts. In that rulemaking, EPA determined that those technologies were not likely to be cost-effective.

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 proposed to significantly reduce VOC emissions from heavy-duty vehicles in two separate proposals (64 FR 58472, October 29, 1999; and 65 Federal Register 35430, June 2, 2000**)**. In the first proposal, we proposed new standards for 2004 and later heavy-duty gasoline vehicles that are projected to reduce exhaust VOC emissions by two-thirds or more. To comply with these proposed 2004 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. We also proposed to require all complete vehicles under 10,000 pounds GVWR to comply with the ORVR requirements.

The other proposal 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 will 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. When combined, these new heavy-duty standards are projected to result in a 83,000-ton reduction in PM emissions and a 230,000-ton reduction in VOC emissions in 2020.

C. Potential for Further Reductions

Given the technology-forcing nature of the recently finalized Tier 2 emission standards for light-duty vehicles and the recently proposed 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 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 propose more stringent standards based on these technologies.[97] 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, there remains little that a manufacturer would be able to do to reduce emissions further. They are already required to design their Tier 2 vehicles to have essentially zero 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 only 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. Thus, we believe that these remaining sources of in-use evaporative emissions cannot be readily addressed by the manufacturer with current technology.

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 manufacturer's control. An EPA standard for metals set at a level that is feasible for manufacturers would only enforce existing engine designs. It would not be likely to achieve any real reductions. 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.

The primary vehicle-based opportunity to reduce toxic emissions is in the area of in-use operation. 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.[98] 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

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[97] Analysis of Cold-Start Emission Controls, Memo to Docket.

[98] 58 FR 9467, February 19, 1993.

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.

Chapter 7: Fuel Controls

This Chapter contains background information and analyses supporting our proposed benzene control program. We first summarize benzene levels in gasoline produced by the U.S. oil industry, the benzene reduction technologies expected to be used by the industry to maintain or reduce gasoline benzene levels, and the means through which we expect the industry to comply with our proposed anti-backsliding requirement. We then describe the costs and emission benefits of our proposed program, and end with a short discussion of the more stringent controls for fuel benzene content that we have chosen not to propose in this rulemaking.

A. Industry and Product Characterization

1. Description of entities subject to the proposed benzene standards

Our proposed anti-backsliding program will apply to every domestic refinery which produces gasoline and every importer or foreign refiner of gasoline to the U.S. We are not proposing any new standards applicable to parties who buy, sell, and/or trade gasoline downstream of refineries. This Section will provide a summary of current gasoline operations for refiners and importers.

a. Refiners

Approximately 146 domestic refineries collectively produced approximately 2.9 billion barrels of gasoline in 1998, or approximately 7.9 million barrels per calendar day on average[99] (however, not all refineries produce gasoline). Refineries are often identified by the Petroleum Administration for Defense District (PADD) in which they reside. PADDs are shown in Figure 7.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 1 is produced by PADD 3 refineries.

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

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

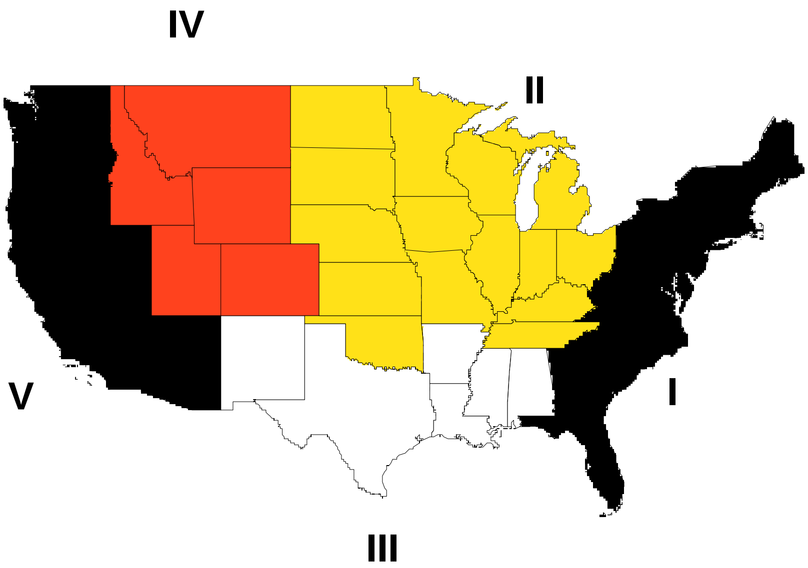

Table VII.A-1
Refinery Count and 1998 Gasoline Volumes by PADD

PADD	Number of operating refineries as of 1/1/2000	Gasoline production (million barrels)[100]	Gasoline consumption (million barrels)[101]
I	14	354	1092
II	30	673	886
III	59	1270	418
IV	15	94	96
V	28	490	521

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

[101] *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.[102] 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

2. Gasoline benzene level variations

This Section presents the average benzene levels for refineries across the U.S. Brief explanations are given for why the benzene levels vary from one refinery to another.

a. Refinery impacts

The 1998 average benzene levels for gasoline are shown in Table VII.A-3 on a PADD-by-PADD basis.

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

Table VII.A-3
PADD-Average Refinery Benzene Levels for Gasoline in 1998[103]

	CG benzene concentration	RFG benzene concentration
PADD I	0.87 vol%	0.56 vol%
PADD II	1.37	0.91
PADD III	0.91	0.65
PADD IV	1.59	0.92
PADD V Excl. Ca[a]	2.15	n/a

[a] Excluding gasoline consumed in California

The values shown in Table VII.A-3 represent the average benzene levels for all refineries in each PADD. However, fuel benzene levels can also vary significantly from refinery to refinery. There are a number of reasons for these variations, including:

1) Type(s) of crude being processed
2) Refinery size and configuration
3) Other products produced
4) Proximity to petrochemical markets for extracted benzene
5) Available capital funds to install benzene reduction equipment
6) Whether a refinery produces CG, RFG, or both

b. Reformulated Gasoline Production Impacts

The Clean Air Act Amendments of 1990 mandated that the benzene content of reformulated gasoline (RFG) not exceed 1.0 volume percent. EPA’s RFG rules include this requirement, and also provide refiners and importers the option of meeting this requirement on an annual average basis. Under the averaging program for RFG, a refinery or importer is in compliance with the requirements of the program if the benzene content of their gasoline annually averages 0.95 volume percent or less. This averaging standard also includes a per-gallon standard of 1.3 volume percent to ensure that benzene levels in RFG are not dramatically different from one area to another. The more stringent averaging standard was intended to recapture the margin of safety that gasoline suppliers were expected to build into their fuels to comply with the per-gallon standards. Thus, because of the reformulated gasoline program, RFG benzene levels, on average, would be expected to be less than 1.0 volume percent. In fact, RFG benzene levels in 1998 averaged 0.65 volume percent, significantly less than required by the RFG program. EPA believes that this "overcompliance" with respect to the RFG benzene requirement is due to a number of factors, including:

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[103] Based on 1998 batch reports collected by EPA under the RFG and anti-dumping programs

1) Benzene extraction for the petrochemical industry
2) Reduction in overall aromatics due to use of oxygenates for octane
3) Dilution with oxygenates

Absent regulatory changes affecting toxic emissions and/or oxygenates, or reduction in the petrochemical demand for benzene, EPA expects that this average level of overcompliance would likely continue. Note that the overcompliance is only a national average, and not all gasoline overcomplies with the current fuel benzene requirements. For instance, in 1998 approximately seven percent of RFG contained benzene at levels higher than the 0.95 volume percent averaging standard (this does not necessarily represent a breech of the standard, since individual batches can be above the standard so long as the annual average is at or below the standard).

The anti-dumping program applicable to conventional gasoline (CG) was instituted to ensure that refiners could not move the dirtiest components of gasoline from RFG to CG. It requires that CG produced after 1994 be no dirtier than it was in 1990 on a refinery-by-refinery basis in terms of exhaust toxics and NOx emissions. The anti-dumping program does not include any direct controls on the benzene content of CG. However, in 1998 the CG benzene content averaged 1.1 volume percent while the average 1990 fuel benzene content of those refiners or importers with individual baselines is approximately 1.3 volume percent. Thus, there is significant overcompliance on CG benzene content. We believe that this overcompliance is due to many of the same factors given above for reformulated gasoline as most refiners in overcompliance for RFG benzene are also in overcompliance for CG benzene.

B. Benzene Emissions and Inventory

As described in more detail in the preamble, we have determined that an anti-backsliding program focusing on gasoline benzene control is the most appropriate program to propose at this time. In this section we present a brief discussion of the benzene inventories by motor vehicle category, as well as the emissions. On the basis of these inventory and emission estimates, it is clear that gasoline benzene control provides an optimum means for lowering toxics emissions through fuel changes.

According to a study conducted by Sierra Research, Inc.[104], the vast majority of benzene emissions from on-highway vehicles is produced by gasoline-powered vehicles. See Table VII.B-1. In 2007, benzene emissions from diesel-powered vehicles are expected to be only one-fortieth that coming from gasoline-powered vehicles. This substantial difference is due in part to the fact that the fraction of total hydrocarbon emissions which is benzene is significantly lower for diesel vehicles than it is for gasoline vehicles.

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[104] "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-1
On-highway Baseline Benzene Inventories from Sierra Report (50-state Tons)

	Gasoline	Diesel
1990	252,136	4625
1996	162,117	2905
2007[a]	83,907	2152
2020[a]	64,894	2609

[a] Assumes that Tier 2 vehicle standards and 30 ppm gasoline sulfur standards have gone into effect. Implications of the proposed standards for MY2007 heavy-duty engines have not been included.

From this table it is clear that current ambient benzene concentrations are more a function of gasoline than of diesel. However, benzene and other MSATs are produced by diesel vehicles, and so do represent a potential alternative avenue for control. At this time we do not have sufficient data to correlate fuel properties with individual toxic compounds from diesel vehicles. As a result, it is unclear how refiners would change diesel fuel to lower toxics emissions.

Benzene emissions from gasoline-powered vehicles are an ideal focus of any toxics control program. Of the toxic compounds emitted by gasoline-powered vehicles, benzene clearly constitutes the largest single fraction on a mass basis. This fact is illustrated both by the baseline emissions used in the RFG program as well as the toxics inventories estimated by Sierra research. These values are summarized in Table VII.B-2.

Table VII.B-2
Relative Importance of Toxics Emissions[a] from Gasoline-powered Vehicles

	Summer baseline emissions for Phase II RFG program		Sierra Research 50-State inventories for gasoline vehicles in 2007	
	mg/mi	% of total	Tons	% of total
Benzene	59.04	69 %	83,907	68 %
Acetaldehyde	4.44	5 %	8,849	7 %
Formaldehyde	9.70	11 %	20,161	16 %
1,3-Butadiene	9.38	11 %	10,049	8 %
POM	3.04	4 %	n/a	n/a

[a] Other toxic compounds may also be present in vehicle emissions. However, those compounds are emitted at much lower levels than those listed in this table.

C. Technological Feasibility of the Proposed Program

1. Requirements for refiners and importers

The proposed program would require that, beginning in 2002 the benzene content of gasoline produced at a domestic refinery or imported cannot exceed the average benzene content of the gasoline produced at that refinery or importer from January 1, 1998 through December 31, 1999. In effect, the proposal maintains current gasoline performance with respect to benzene content, on an individual refinery or importer basis. Because we are not requiring benzene controls more stringent than current performance, compliance should not require extra effort by refiners, barring unexpected circumstances.

We are proposing that compliance with the proposed benzene requirement be met separately for RFG and CG. EPA believes this is the most appropriate methodology for benzene accounting, as it would ensure that RFG and CG areas are in general receiving the same quality of gasoline, with respect to benzene, as they did during the 1998-1999 timeframe. We do not expect significant changes between 1998-1999 and 2000-2001 (immediately prior to the program start) in terms of gasoline benzene levels despite the fact that the Phase II RFG program began on January 1, 2000. Phase II RFG will require more stringent emission performance standards that could include lower concentrations of butanes and pentanes and might tend to lower octane levels. However, we believe that these potential changes will have only negligible effects on fuel benzene levels, and that as a result the 1998-1999 baselines will remain representative of current benzene levels.

2. Requirements for those without complete 1998-1999 benzene data

Certain regulated parties did not produce or import gasoline into the U.S. during some or all of 1998-1999 , and thus do not have an appropriate benzene baseline as required under the proposal. EPA is proposing the following criteria for determining the baseline benzene for a regulated entity for use in our proposed benzene control program:

> 1) For parties which produced or imported for more than one year during 1998-1999 . EPA is proposing that refineries and importers must establish an individual benzene baseline if it produced gasoline for at least 12 consecutive months during 1998-1999 . All relevant and valid data from 1998-1999 must be included in the baseline determination.
>
> 2) For parties which produced or imported for less than one year during 1998-1999 . EPA is proposing that refiners or importers who produced or imported gasoline for less than 12 consecutive months during 1998-1999 would be assigned the 1998-1999 industry averages for CG as their benzene baseline. The industry average baseline will be determined and announced by EPA as part of this rulemaking.

3. Refinery technologies for controlling gasoline benzene

Under today's proposal, a refinery must produce gasoline with an annual average fuel benzene level no greater than the refinery's 1998-1999 average. This Section presents a summary of how refineries control the benzene content of their gasoline. These technologies can be used to maintain benzene levels at the 1998-1999 baselines, or could be used to lower fuel benzene levels further in the event the we promulgate more stringent benzene standards in the future. Other means of controlling gasoline benzene levels may be available in 2004, and will be considered during our evaluation of the appropriateness of additional future benzene controls.

a. General description

Gasoline is a mixture of blended streams from various units in the refinery. The refinery unit which contributes the most benzene to gasoline is the reformer. The product from the reformer, reformate, is a highly aromatic, high octane gasoline-blending stream. Reformate constitutes approximately 30 percent of gasoline volume and typically contains 3 to 8 percent benzene. Thus, the reformer contributes 70 to 85 percent of the benzene in the gasoline pool[105] . As a result, most methods for controlling gasoline benzene either affect reformer operation or address reformate benzene, as discussed below.

Some refiners may be able to make adjustments to their reformer in order to maintain

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[105] Higgins, Terry, *Background Paper on Benzene Control in Gasoline,* National Petroleum Refiners Association. March 23, 2000.

current benzene levels in their gasoline pool. Refinery adjustments to the reformer may include changing out the catalyst, less throughput, and/or running the reformer less severely. Two types of reformers are most prevalent in refineries: semi-regenerative and continuous regeneration. Continuous regeneration reformers operate at less pressure than semi-regenerative reformers, which means less dealkylation of heavy aromatics to benzene. Therefore, another reformer option would be to change from a semi-regenerative reformer to a continuous regeneration reformer because a continuous regeneration reformer generally produces less benzene than a semi-regenerative reformer. Another option available to a refinery in order to maintain current benzene levels in their gasoline would be to blend oxygenates in their gasoline. Since oxygenates do not contain benzene, it may be used as a diluent to maintain or reduce benzene levels in gasoline. Also, oxygenates are high octane blending components which enable a refinery to run their reformer less severely and use less feed to reformer.

The primary methods of reducing the benzene content of gasoline are pre-fractionation and post-fractionation. Pre- and post- refer to the timing of fractionation, or stream separation, relative to the reformer, that is, either before or after the reformer, respectively. There are several processes of pre-fractionation and post-fractionation, which are explained below. Refiners may use one method or a combination of methods to achieve their desired gasoline benzene content, depending on refinery configuration and crude oil mix.

There are two pre-fractionation options with different processes. The first option involves the use of a naphtha splitter which splits the naphtha stream coming from the crude tower into a light naphtha stream and a heavy naphtha stream. The light naphtha stream, which are C-5 and C-6 compounds, may be blended directly to gasoline if the light naphtha benzene content is low and octane is not needed. The naphtha splitter removes and diverts part of this stream which contains some of the chemical components that would have formed benzene in the reformer (at some refiners this split is made in the crude tower). The heavy naphtha stream, which are C-7 and heavier compounds, are sent to the reformer. Since this process removes some of these "benzene precursors" from the feed to the reformer, the benzene content of the reformate, and thus of gasoline, is reduced. The second option of pre-fractionation involves the use of the naphtha splitter technology described previously, and in addition, uses an isomerization unit which rearranges the structure of the benzene precursors and converts the light naphtha stream into a higher octane stream.

Benzene reduction strategies which occur after the reformer is called post-fractionation. Post-fractionation is the fractionation of the reformate stream after the reformer. There are three post-fractionation options that a refinery may choose, but each post-fractionation option has different processes.

The first option of post-fractionation is isomerization of the benzene rich portion of the reformate stream. In this process, benzene is saturated to cyclohexane using hydrogen. This strategy recovers the octane of the stream by isomerizing the C5/C6 paraffins to higher octane iso-paraffins. One variation of this type of technology involves a combination of both pre-fractionation light naphtha stream and post-fractionation of the reformate product, where both resulting splits are routed to the C5/C6 isomerization unit. This option would be chosen by a refinery if they needed more higher octane blending components. Benzene saturation in an isomerization unit normally requires a separate reactor specifically designed for saturating benzene.

The second option of post-fractionation is hydrogen saturation of the benzene rich stream from the reformate product. In the case of benzene, the process of hydrogen saturation converts benzene to cyclohexane. This process can require a significant amount of hydrogen, and hydrogen is an expensive commodity in a refinery. Further, cyclohexane has a lower octane than benzene. If hydrogen saturation is used as a stand alone unit, then it will most likely have an impact on the octane of the refiner's gasoline. However, the octane may be increased by further processing the saturation product in an isomerization unit. This additional step may further increase the cost of benzene reduction. As a result of these issues, a refinery may choose not to pursue this route unless it already has hydrogen saturation capacity or if hydrogen is readily available.

Finally, extraction is used to separate and collect chemical grade benzene for the petrochemical market. It requires the use of a reformate splitter to separate out a benzene-rich stream from the reformate. This benzene-rich stream is then sent to an aromatic extraction complex which extracts the benzene or converts it into other petrochemicals, such as xylenes. The capital cost of a benzene extraction unit is quite high, approximately $12.5 million for a 10,400 barrel per day unit[106]. Thus a refinery wants to ensure that such a unit will be run to the fullest extent possible so as to maximize the return on investment. Additionally, benzene has a very high freezing point (i.e., around 40 degrees F) which requires it to be shipped in heated barges or heated railway cars. This method of transportation costs approximately three times more than transportation costs of other petrochemicals. Further, stationary source benzene emissions from a refinery are heavily regulated, so refiners must invest significant capital at the refinery to control stationary source benzene emissions. Therefore, in order to make benzene extraction economically feasible for a refinery, the refinery must be located near a petrochemical market. Most petrochemical markets are on the East or Gulf coasts. Approximately twenty-five refiners (outside of California) have benzene extraction units.

b. Impacts of refinery size and location

In general, larger refineries and multi-refinery refiners have more options for reducing benzene than smaller refiners. In general, larger refineries and multi-refinery refiners have more technological options for reducing benzene. For example, of the estimated 26 refineries with benzene extraction units, 20 have crude capacities greater than 140,000 barrels per calendar day,

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[106] Meyers, Robert A, *Handbook of Petroleum Refining Processes*, second edition, McGraw-Hill, Boston (1997).

and all but one belong to multi-refinery refiners[107].

On average, refiners in PADDs I, II and III have more complex refineries with more options for stream processing, than refiners in PADDs IV and V outside of California. Additionally, most of the petrochemical benzene markets are in PADDs I, II and III, thus providing a readily accessible market for chemical benzene. Thus, in general, refiners in PADDs I, II and III would be expected to have less difficulty dealing with any production issues which would affect their compliance with the proposed program.

D. Costs and Benefits of the Proposed Program

We have referred to our proposed program as an "anti-backsliding" program because it is designed to ensure that fuel benzene levels rise no higher than they were in 1998-1999 . Refineries will not be required to reduce their benzene levels below the average levels they achieved in 1998-1999 . In addition, we believe that, because our proposed program applies annual average standards to each refinery, each refinery will be able to meet their 1998-1999 baseline without the need for a compliance margin to account for blending tolerances, measurement uncertainty, and contamination. As a result, we do not expect that our proposed standards will result in reductions in fuel benzene levels in comparison to 1998-1999 levels, and refiners should therefore incur negligible costs, if any, for compliance with these proposed standards.

No reduction in fuel benzene levels also means that our proposed standards will produce no emission benefits in comparison to 1998-1999 levels. However, benzene emissions associated with fuel benzene content could go down over time for another reason. Currently, much of the overcompliance on gasoline benzene levels arise from the demand for chemical benzene which rose approximately 30 percent between 1990 and 1998[108]. This benzene was used in the production of ethylbenzene, styrene, cumene, and other compounds. The trends for chemical benzene demand show continued increases through 1998, and future forecasts from both Dewitt & Company, Inc. and Honeywell Hi-Spec Solutions include continued increases for the next several years. This forecast is shown in Figure 7.D-1.

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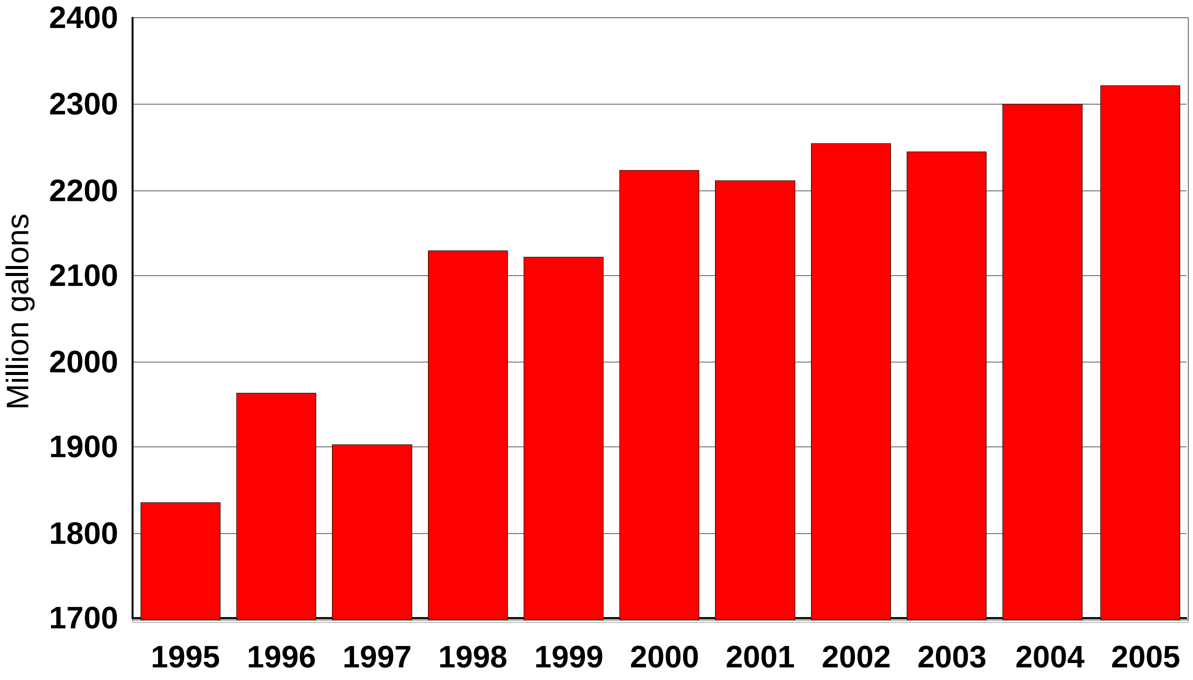
[107] Oil & Gas Journal, December 20, 1999

[108] Based on an analysis from Honeywell Hi-Spec Solutions, based on 1999 NPRA Petrochemical Survey for benzene. Contact: Robert Harvin, Honeywell Hi-Spec Solutions, 713-953-3400.

Figure VII.D-1
Chemical Benzene Demand in the U.S. (Honeywell Hi-Spec Solutions forecast)



Based on this forecast, then, it is possible that existing extraction capacity could be expanded in the coming years to take advantage of the expected growth in the chemical benzene market, which would lower benzene levels in gasoline even further than their 1998-1999 levels.

This proposed anti-backsliding program does not include a credit trading program. However, EPA is seeking comment on the need for and viability of a credit trading program such as outlined below. While the agency believes it has provided sufficient flexibility with the proposed deficit carryover program, we are seeking comment on this credit trading approach as an alternative, or additional, means of providing compliance flexibility.

The current Reformulated Gasoline Rules provide a credit program that allows the transfer of benzene credits by refiners, importers, and blenders (see 40 CFR 80.67). In this program, benzene credits can be generated from a baseline average of 0.95 vol% benzene. This program will remain in place. Refiners that currently rely on this program, if any, will continue to be able to use it in meeting the basic RFG requirements in 40 C.F.R. subpart D.

This credit generation and transfer approach could also be incorporated in the proposed anti-backsliding benzene standard. Refiners could generate credits by reducing the average benzene in their product below the anti-backsliding baseline. Under such a trading program, compliance could be achieved through a transfer of benzene credits provided that (1) the credits are generated in the same averaging period as they are used; (2) the credit transfer takes place not later than 15 working days following the end of the averaging period in which the benzene credits were generated; (3) the credits were properly created; and (4) the credits are transferred directly from the refiner, importer, or blender that created the credits to the refiner, importer, blender that used the credits to achieve compliance (i.e., no brokering of credits). In addition, based on the fact that RFG and CG would have separate baselines, EPA believes it would be inappropriate to allow credit trading between the RFG and conventional gasoline pools.

E. More Stringent Control Programs

There are a wide variety of programs more stringent than the anti-backsliding program we are proposing today which may yield additional toxics emissions reductions beyond the reductions we expect under existing programs. These include further fuel benzene controls, controls on other fuel properties which affect toxics emissions, or emission performance standards directed at one or multiple toxic compounds. Because we do not have sufficient information to adequately evaluate the costs and benefits of such additional programs at this time, we are not proposing any such an additional program in today's action. Instead, we will conduct further evaluations of the costs and benefits of more stringent toxics controls through fuels in our Technical Analysis Plan over the next few years.

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.

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 urban air pollution, 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.[109] 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,[110] and CO from nonroad engines and equipment contribute significantly to ozone and CO concentrations in more than one nonattainment area.[111] Thus, we initiated regulatory programs for several categories of nonroad engines as required by section 213(a)(3) of the Clean Air Act.

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

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[109] "Nonroad Engine and Vehicle Study - Report and Appendices," EPA-21A-201, November 1991 (available in Air Docket A-96-40).

[110] HC stands for hydrocarbon. HC and VOC are very similar and are generally used interchangeably.

[111] 59 FR 31306, June 17, 1994.

that may be reasonably anticipated to endanger public health or welfare.[112] 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 engines, and HC and CO reductions from gasoline 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 have 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), 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

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[112] 59 FR 31306, June 17, 1994.

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.[113] 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.[114]

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.[115] 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, which will include proposal and adoption of appropriate Tier 3 standards for PM.

2. Small Land-Based SI Engines

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[113] 59 FR 31306, June 17, 1994.

[114] 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.

[115] 63 FR 56968, October 23, 1998.

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. [116] 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.[117] 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 to achieve approximately 350,000 tons of HC + NOx emission reductions, and the program for handheld engines to achieve approximately 230,00 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.

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 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

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[116] 60 FR 34582, July 3, 1995.

[117] 64 FR 15208, March 30, 1999 and 65 FR 24267, April 25, 2000.

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 HC 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.[118] 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 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.[119] 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.

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

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[118] 61 FR 52088, October 4, 1996.

[119] 64 FR 73300, December 29, 1999.

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.

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.[120] 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. 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.

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, 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. However, only about one third of these VOC and diesel PM reductions will be realized by 2010.

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[120] 63 FR 18978, April 16, 1998.

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.[121] 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.

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.

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

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[121] International Civil Aviation Organization (ICAO) Annex 16, Volume II, Environmental Protection, Aircraft Engine Emissions.

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 have dramatically reduced the sulfur levels in on-highway diesel fuel, and are currently considering further reductions in on-highway diesel fuel sulfur levels. 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 are considering the control of sulfur in nonroad diesel fuel as part of our Tier 3 technology review. This would 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 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 engine 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. As part of our Technical Analysis Plan, described in the preamble to the proposed regulation, we will also assess the need for and appropriateness of controls for nonroad sources of MSATs.

1. Nonroad MSAT Baseline Inventories

We previously presented the 1996 baseline inventories for several key nonroad MSATs in Table IV.A-2. 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 which we have completed. As previously discussed, we are developing the first national regulations applicable to recreational vehicles and recreational marine sterndrive and inboard engines. We are also developing additional Tier 3 emission regulations applicable to land-based nonroad CI engines. These regulations, when completed, will result in additional VOC (and, thus, gaseous MSAT) reductions. This rulemaking action will also include proposal and adoption of appropriate Tier 3 standards for PM.

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, 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.

a. MSATs

Table VIII..B-1 shows our estimates of four nonroad gaseous MSAT emissions. These estimates were based on the 1996 inventories contained in the 1996 NTI study. The 1990 estimates were derived by applying nationwide VOC totals and toxic fractions from the draft NONROAD model to the 1996 NTI numbers.[122] The 2007 and 2020 estimates were derived from the draft NONROAD model, also 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

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[122] 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. The version of the NONROAD model that was used in this analysis is the one which we also used in support of our recently proposed 2007 heavy-duty vehicle and diesel fuel control proposal (65 FR 35429, June 2, 2000).

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.

Table VIII.B-1
Annual Toxics Emissions Summary for Selected Air Pollutants for the Total U.S. Nonroad Mobile Sources from 1990 to 2020
(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
Summary of Percent Emission Reductions in 2007 and 2020 for Selected Air Pollutants for the Total U.S. from 1990 or 1996
Nonroad Mobile 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 do not have detailed emissions data from nonroad mobile sources for the other gaseous MSATs. 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, 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 proposed 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.[123] 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 shows that our existing nonroad emission control programs will also result in significant gaseous MSAT reductions (assuming, as previously discussed, that gaseous MSATs emissions track VOC reductions).

Table VIII.B-3
Annual VOC Emissions Summary for the Total U.S.
Nonroad Mobile 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. 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

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[123] "National Air Pollutant Emission Trends, 1900-1996," EPA-454/R-97-011, December, 1997.

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.

Table VIII.B-4
Annual Diesel PM Emissions Summary for the Total U.S.
Nonroad Mobile 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 proposed rule, contains a strategy to obtain and evaluate this data so we can evaluate the feasibility of, and need for, additional nonroad engine controls in the future.