MOTOR VEHICLE-RELATED AIR TOXICS STUDY

April 1993

Technical Support Branch
Emission Planning and Strategies Division
Office of Mobile Sources
Office of Air and Radiation
U.S. Environmental Protection Agency

Acknowledgements

The primary EPA contributors to this study were Pamela Brodowicz, Penny Carey, Richard Cook, and Joseph Somers. Gertrude Venlet and Betty Measley provided clerical support. An EPA work group was formed to provide review, comments, and necessary input from other parts of EPA. Members of this work group are Dwight R. Atkinson, Joseph J. Bufalini, James W. Caldwell, Ila L. Cote, Stanley B. Durkee, Robert B. Faoro, Robert Fegley, John T. Hannon, Oscar Hernandez, Alan H. Huber, Gary L. Kimmel, Kenneth T. Knapp, Thomas F. Lahre, Mike Lidgard, Chris Lindhjem, Thomas R. McCurdy, William E. Pepelko, Steven A. Shedd, and Chon R. Shoaf. The contributions of these individuals are gratefully acknowledged.

This study incorporates material and information from four work assignments, three initiated specifically to provide input for this study. Systems Applications International prepared two reports titled "Atmospheric Transformation of Air Toxics: Benzene, 1,3-Butadiene, and Formaldehyde," under the direction of Mary P. Ligocki, and "Atmospheric Transformation of Air Toxics: Acetaldehyde and Polycyclic Organic Matter," prepared for EPA's Office of Policy Planning and Evaluation by Mary P. Ligocki and Gary Whitten. Also, Clement International Corporation prepared a report titled "Motor Vehicle Air Toxics Health Information," under the direction of Sharon Siegel. Finally, Ted Johnson, Roy Paul, and Jim Capel of International Technology Air Quality Services prepared a report titled "Application of the Hazardous Air Pollutant Exposure Model (HAPEM) to Mobile Source Pollutants."

Other individuals who contributed significantly to this study, providing technical expertise and guidance, include Jeff Alson, David Brzezinski, Gary Dolce, Alva Edwards, Carl Fulper, Karl Hellman, Greg Janssen, Karen Levy, Phil Lorang, Steve Mayotte, Carl Mazza, Will Smith, Todd Ramsden, Rick Rykowski, and Mark Wolcott of EPA, Robert Kelty and Tom McDonnell of Computer Sciences Corporation, and Edward Sienicki of Navistar Corporation. Comments on preliminary drafts of various portions of the study were provided by Larry Claxton, Larry Cupitt, Linda Birnbaum, Mike Davis, and Vanessa Vu of EPA, and K. D. Drachand and Joe DeVita of the California Air Resources Board. Additional EPA comments on the public review draft were provided by Aparna M. Koppikar, Cheryl Siegel Scott, and Hugh McKinnon. The following organizations provided comments on the public review draft: American Automobile Manufacturers Association, American Petroleum Institute, Association of International Automobile Manufacturers, Arco Chemical Company, California Air Resources Board, California Environmental Protection Agency, Chemical Manufacturers Association (CMA), Engine Manufacturers Association, Ford Motor Company, General Motors Corporation, Health Effects Institute, Konheim and Ketcham, Northeast States for Coordinated Air Use Management, and Zephyr Consulting.

The authors of this study wish to express their sincere appreciation for the efforts of all participants.

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

TABLE OF CONTENTS

(Note: page numbers may be slightly off in electronic PDF version)

EXECUTIVE SUMMARY

Section 202(l)(1) of the Clean Air Act (CAA), as amended (Section 206 of the Clean Air Act Amendments (CAAA) of 1990 added paragraph (l) to Section 202 of the CAA), directs EPA to complete a study by May 15, 1992 of the need for, and feasibility of, controlling emissions of toxic air pollutants which are unregulated under the Act and associated with motor vehicles and motor vehicle fuels. In addition, the study is to consider the means and measures for such controls. The required study is to focus on those categories of emissions that pose the greatest risk to human health or about which significant uncertainties remain, including emissions of benzene, formaldehyde, and 1,3-butadiene. This study has been prepared in response to Section 202(l)(1).

Motor vehicle emissions are extremely complex. Hundreds of compounds have been identified. For this study, specific pollutants or pollutant categories which are discussed include benzene, formaldehyde, 1,3-butadiene, acetaldehyde, diesel particulate matter, gasoline particulate matter, and gasoline vapors, all of which have been considered in previous analyses of air toxics, as well as selected metals and motor vehicle-related pollutants identified in Section 112(b) of the Clean Air Act.

The focus of the study is on carcinogenic risk. The discussion of non-carcinogenic effects is less quantitative due to the lack of sufficient health data. Nevertheless, noncarcinogenic effects should not be viewed as less important. Noncancer effects associated with exposures to the pollutants discussed in this study await assessment.

There are a number of major limitations and uncertainties which need to be considered carefully when reviewing the results of this study. In the interest of readability, the contents of this study are discussed first, then the limitations and uncertainties presented.

There are chapters devoted to each individual pollutant or pollutant category. Topics covered for each pollutant/pollutant category include chemical and physical properties, formation and control technology, emissions (including other emission sources), atmospheric reactivity and residence times, exposure estimation, EPA's carcinogenicity assessment, other views of carcinogenicity assessment, recent and ongoing research, carcinogenic risk, and non-cancer health effects. There is also a chapter which describes EPA's Integrated Air Cancer Project, aimed at identifying the major carcinogenic chemicals emitted into the air, and the sources of these chemicals. A chapter is also included which describes qualitative changes in toxic pollutant levels with the use of alternative clean fuels such as methanol, ethanol, compressed natural gas, and liquid propane gas. Another brief chapter discusses toxic emissions from nonroad mobile sources. In addition, a chapter discusses the costs of various

existing regulatory programs and a qualitative discussion of the toxics benefits of these programs.

This study attempts to summarize what is known about motor vehicle-related air toxics and to present all significant scientific opinion on each issue. Based on information presented in this study and other relevant information, EPA is to promulgate (and from time to time revise) regulations by May 15, 1995 that contain reasonable requirements to control hazardous air pollutants from motor vehicles and motor vehicle fuels. The regulations, at a minimum, apply to emissions of benzene and formaldehyde. This study does not address whether to promulgate standards or what standards should be promulgated, since those issues will be addressed in the rulemaking activity.

Briefly, cancer risk estimates were obtained in the following manner. First, emission factors in units of gram/mile were estimated as a function of vehicle technology and fuel composition. These emission factors were then used in a model to calculate annual average exposures. The annual nationwide exposures were compared to the range of ambient data, and where necessary, adjustments were applied such that modeled data matched the upper end of the ambient range. Then, the adjusted exposures were multiplied by the population of interest and the EPA unit risk factor to calculate lifetime cancer incidence or, for benzene and diesel particulate matter, cancer deaths. The unit risk factor is the excess individual lifetime risk due to continuous lifetime exposure to one unit (in this case, $\mu g/m^3$) of carcinogen concentration. To calculate annual cancer incidence (or deaths), the lifetime cancer incidence (or deaths) was divided by 70, the average years per lifetime.

Cancer risk estimates for benzene, diesel particulate matter, formaldehyde, 1,3-butadiene, and acetaldehyde are provided for the following years: 1990, 1995, 2000, and 2010. The following scenarios are examined:

1) a base control scenario, which takes into account implementation of the motor vehicle-related Clean Air Act requirements,

2) a scenario involving expanded use of reformulated gasoline, and

3) a scenario involving expanded adoption of California motor vehicle emission standards.

The expanded control scenarios are not intended to be predictive, but instead are intended to encompass a wide range of possibilities. Base control scenarios for the years examined take into account implementation of the motor vehicle-related CAA requirements, but assume no expanded adoption of CAA programs or California standards. The expanded use of reformulated fuel scenario is considered for the years 1995, 2000, and 2010. In this scenario, all ozone nonattainment areas opt into the federal

reformulated gasoline program. The expanded adoption of California standards scenario is considered for the years 2000 and 2010. California emission standards are similar to federal motor vehicle-related standards in 1995; thus, this scenario is not considered for that year. However, California motor vehicle emission standards become increasingly more stringent with time, so that in 2000 and 2010, they are markedly lower than federal standards. In this scenario, Northeast states and states with ozone nonattainment areas categorized as extreme, severe, or serious adopt California emission standards. This scenario also assumes expanded use of reformulated gasoline, as described in the previous scenario. Federal Tier II standards were not evaluated in this study.

Table ES-1 summarizes the emission factors, annual average exposure estimates, nationwide cancer incidence (or deaths), and nationwide annual individual risks for all scenarios/years. The limitations and uncertainties listed in the footnotes to this table and discussed at the end of the executive summary should be considered when reviewing these numbers. For the base control scenarios, the cancer incidences or deaths decrease from 1990 to 1995 and from 1995 to 2000. From 2000 to 2010, the cancer incidences or deaths increase for 1,3-butadiene, formaldehyde, and acetaldehyde. For these toxics, even though the fleet average emission factors in gram/mile continue to decrease from 2000 to 2010, the projected increase in vehicle miles travelled (and population to a lesser extent) more than offsets this decrease. For benzene, cancer deaths remain unchanged from 2000 to 2010, whereas for diesel particulate, cancer deaths decrease. It should be noted that, due to uncertainties associated with the additivity of cancer risk associated with the toxics, total cancer risk for all toxics for a given scenario/year are not presented in Table ES-1.

The expanded use of reformulated gasoline and expanded adoption of California motor vehicle emission standards scenarios result in lower cancer deaths or incidences for benzene and 1,3-butadiene relative to their base control scenarios. Cancer incidences due to formaldehyde increase slightly, but are more than offset by the benzene and 1,3-butadiene decreases.

Oxygenated fuels provide overall health benefits because they significantly reduce winter CO in areas which exceed CO ambient air quality standards. Increased use of oxygenated fuels may result in small increases in ambient aldehyde levels and may increase intermittent exposures to concentrations higher than ambient levels. However, the use of oxygenated fuels also results in

Table ES-1. Summary of Estimates of Emission Factors, Annual Average Exposure, Nationwide Annual Cancer Deaths or Incidences, and Nationwide Annual Individual Risk for All Scenarios.[a]

	1990	1995		2000			2010		
Pollutant	**Base Control**	**Base Control**	**Expanded Reform. Gasoline Use**	**Base Control**	**Expanded Reform. Gasoline Use**	**Expanded Adoption Calif. Stds.**	**Base Control**	**Expanded Reform. Gasoline Use**	**Expanded Adoption Calif. Stds.**
BENZENE	**Estimated Cancer Deaths with Estimates of Exposure Calculated in this Study[c,d]**								
Estimated cancer deaths are based on the EPA 1985 unit risk of 8.3×10^{-6} per µg/m³, determined using human data.									
EF (g/mi)[b]	**0.0882**	**0.0472**	**0.0413**	**0.0351**	**0.0301**	**0.0305**	**0.0285**	**0.0248**	**0.0228**
Exposure[c] (µg/m³)	**2.36**	**1.40**	**1.20**	**1.10**	**0.98**	**0.98**	**1.05**	**0.93**	**0.84**
Cancer Deaths[d]	**70**	**43**	**37**	**35**	**31**	**31**	**35**	**31**	**28**
Average of Individual Risk[e]	**2.8×10^{-7}**	**1.7×10^{-7}**	**1.4×10^{-7}**	**1.3×10^{-7}**	**1.2×10^{-7}**	**1.2×10^{-7}**	**1.2×10^{-7}**	**1.1×10^{-7}**	**9.9×10^{-8}**
	Estimated Cancer Deaths with Alternative Estimates of Exposure								
Range of Exposure[f] (µg/m³)	1.37-3.98	0.81-2.36	0.70-2.02	0.64-1.86	0.57-1.65	0.57-1.65	0.61-1.77	0.54-1.57	0.49-1.42
Range of Cancer Deaths	41-118	25-72	22-62	21-59	18-52	18-52	18-53	18-52	16-47
	Estimated Cancer Deaths with Clement Associates, 1988 Unit Risk (4.3×10^{-8} per µg/m³) or CARB, 1984 Unit Risk (5.2×10^{-5} per µg/m³).[g] These are not directly comparable to the official EPA unit risk estimates.								
Exposure (µg/m³)	2.36	1.40	1.20	1.10	0.98	0.98	1.05	0.93	0.84
Cancer Deaths (Clement, 1988)	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
Cancer Deaths (CARB, 1984)	438	269	232	219	194	194	219	194	175

Please refer to footnotes on page ES-9.

Table ES-1 Continued.

	1990	1995		2000			2010		
Pollutant	**Base Control**	**Base Control**	**Expanded Reform. Gasoline Use**	**Base Control**	**Expanded Reform. Gasoline Use**	**Expanded Adoption Calif. Stds.**	**Base Control**	**Expanded Reform. Gasoline Use**	**Expanded Adoption Calif. Stds.**
FORMALDEHYDE	**Estimated Cancer Incidences with Estimates of Exposure Calculated in this Study[c,d]**								
Estimated cancer incidences are based on the EPA 1987 upper bound unit risk of 1.3×10^{-5} per µg/m³, determined using animal data.									
EF (g/mi)[b]	**0.0412**	**0.0234**	**0.0251**	**0.0162**	**0.0166**	**0.0168**	**0.0140**	**0.0143**	**0.0138**
Exposure[c] (µg/m³)	**0.95**	**0.58**	**0.62**	**0.42**	**0.44**	**0.44**	**0.42**	**0.46**	**0.42**
Cancer Cases[d]	**44**	**28**	**30**	**21**	**22**	**22**	**22**	**24**	**22**
Average of Individual Risk[e]	**1.8×10^{-7}**	**1.1×10^{-7}**	**1.2×10^{-7}**	**7.8×10^{-8}**	**8.2×10^{-8}**	**8.2×10^{-8}**	**7.8×10^{-8}**	**8.5×10^{-8}**	**7.8×10^{-8}**
	Estimated Cancer Incidences with Alternative Estimates of Exposure								
Range of Exposure[f] (µg/m³)	0.95-2.87	0.58-1.75	0.62-1.87	0.42-1.27	0.44-1.33	0.44-1.33	0.42-1.27	0.46-1.39	0.42-1.27
Range of Cancer Cases	44-133	28-85	30-91	21-63	22-67	22-67	22-67	24-73	22-67
	Estimated Cancer Incidences with EPA, 1991 Draft Upper Bound Unit Risk (6.0×10^{-7} per µg/m³) or EPA, 1987 Upper Bound Unit Risk[g] (1.3×10^{-5} per µg/m³). The draft EPA, 1991 estimate is not an official EPA estimate.								
Exposure (µg/m³)	0.95	0.58	0.62	0.42	0.44	0.44	0.42	0.46	0.42
Cancer Cases[h] (EPA, 1991)	2	1	1	1	1	1	1	1	1
Cancer Cases (EPA, 1987)	44	28	30	21	22	22	22	24	22

Please refer to footnotes on page ES-9.

Table ES-1 Continued.

	1990	1995		2000			2010		
Pollutant	**Base Control**	**Base Control**	**Expanded Reform. Gasoline Use**	**Base Control**	**Expanded Reform. Gasoline Use**	**Expanded Adoption Calif. Stds.**	**Base Control**	**Expanded Reform. Gasoline Use**	**Expanded Adoption Calif. Stds.**
1,3-BUTADIENE	**Estimated Cancer Incidences with Estimates of Exposure Calculated in this Study[c,d]**								
Estimated cancer incidences are based on the EPA 1985 upper bound unit risk of 2.8×10^{-4} per µg/m³, determined using animal data.									
EF (g/mi)[b]	**0.0156**	**0.0094**	**0.0093**	**0.0071**	**0.0069**	**0.0069**	**0.0067**	**0.0064**	**0.0062**
Exposure[c] (µg/m³)	**0.30**	**0.20**	**0.20**	**0.16**	**0.16**	**0.16**	**0.18**	**0.17**	**0.16**
Cancer Cases[d]	**304**	**209**	**207**	**176**	**171**	**172**	**204**	**194**	**186**
Average of Individual Risk[e]	**1.2×10^{-6}**	**8.1×10^{-7}**	**8.0×10^{-7}**	**6.6×10^{-7}**	**6.4×10^{-7}**	**6.4×10^{-7}**	**7.2×10^{-7}**	**6.9×10^{-7}**	**6.6×10^{-7}**
	Estimated Cancer Incidences with Alternative Estimates of Exposure								
Range of Exposure[f] (µg/m³)	0.07-0.56	0.05-0.37	0.05-0.37	0.04-0.30	0.04-0.30	0.04-0.30	0.04-0.34	0.04-0.32	0.04-0.30
Range of Cancer Cases	70-560	48-385	48-381	41-324	39-315	40-317	47-376	45-357	43-343
	Estimated Cancer Incidences with Hattis and Watson, 1987 Upper Bound Unit Risk (1.1×10^{-7} per µg/m³) or ICF, 1986 Upper Bound Unit Risk (3.4×10^{-3} per µg/m³). These are not directly comparable to the official EPA unit risk estimate.[g]								
Exposure (µg/m³)	0.30	0.20	0.20	0.16	0.16	0.16	0.18	0.17	0.16
Cancer Cases (Hattis and Watson 1987)	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
Cancer Cases (ICF, 1986)	3691	2538	2514	2137	2076	2089	2477	2356	2259

Please refer to footnotes on page ES-9.

Table ES-1 Continued.

	1990	1995		2000			2010		
Pollutant	**Base Control**	**Base Control**	**Expanded Reform. Gasoline Use**	**Base Control**	**Expanded Reform. Gasoline Use**	**Expanded Adoption Calif. Stds.**	**Base Control**	**Expanded Reform. Gasoline Use**	**Expanded Adoption Calif. Stds.**
ACETALDEHYDE	**Estimated Cancer Incidences with Estimates of Exposure Calculated in this Study.[c,d]**								
Estimated cancer incidences are based on the EPA 1987 upper bound unit risk of 2.2×10^{-6} per µg/m³, determined using animal data.									
EF (g/mi)[b]	**0.0119**	**0.0071**	**0.0071**	**0.0051**	**0.0051**	**0.0052**	**0.0045**	**0.0044**	**0.0041**
Exposure[c] (µg/m³)	**0.67**	**0.44**	**0.44**	**0.33**	**0.33**	**0.33**	**0.34**	**0.34**	**0.31**
Cancer Cases[d]	**5.3**	**3.6**	**3.6**	**2.8**	**2.8**	**2.8**	**3.0**	**3.0**	**2.8**
Average of Individual Risk[e]	**2.0×10^{-8}**	**1.4×10^{-8}**	**1.4×10^{-8}**	**1.0×10^{-8}**	**1.0×10^{-8}**	**1.0×10^{-8}**	**1.1×10^{-8}**	**1.1×10^{-8}**	**9.9×10^{-9}**
	Estimated Cancer Incidences with Alternative Estimates of Exposure								
Range of Exposure[f] (µg/m³)	0.67-1.71	0.44-1.12	0.44-1.12	0.33-0.84	0.33-0.84	0.33-0.84	0.34-0.87	0.34-0.87	0.31-0.79
Range of Cancer Cases	5.3-13.4	3.6-9.1	3.6-9.1	2.8-7.1	2.8-7.1	2.8-7.1	3.0-7.6	3.0-7.6	2.8-7.1
	Estimated Cancer Incidences with EPA 1987 Upper Bound Unit Risk (2.2×10^{-6} per µg/m³) or CARB, 1992 Upper Bound Unit Risk (2.7×10^{-6} per µg/m³). The CARB, 1992 estimate is not directly comparable to the official EPA estimate.[g]								
Exposure (µg/m³)	0.67	0.44	0.44	0.33	0.33	0.33	0.34	0.34	0.31
Cancer Cases (EPA, 1987)	5.3	3.6	3.6	2.8	2.8	2.8	3.0	3.0	2.8
Cancer Cases (CARB, 1992)	6.5	4.4	4.4	3.4	3.4	3.4	3.7	3.7	3.4

Footnotes can be found on page ES-9.

Table ES-1 Continued.

	1990	1995		2000			2010		
Pollutant	**Base Control**	**Base Control**	**Expanded Reform. Gasoline Use**	**Base Control**	**Expanded Reform. Gasoline Use**	**Expanded Adoption Calif. Stds.**	**Base Control**	**Expanded Reform. Gasoline Use**	**Expanded Adoption Calif. Stds.**
DIESEL PARTICULATE MATTER	**Estimated Cancer Deaths with Estimates of Exposure Calculated in this Study[c,d]**								
Estimated cancer deaths are based on the EPA 1991 draft upper bound unit risk of 1.7×10^{-5} per µg/m³, determined using animal data. This unit risk has not been peer reviewed and is subject to change.									
EF (g/mi)[b]	**0.0669**	**0.0356**	**-**	**0.0188**	**-**	**-**	**0.0105**	**-**	**-**
Exposure[c] (µg/m³)	**1.80**	**1.05**	**-**	**0.60**	**-**	**-**	**0.39**	**-**	**-**
Cancer Deaths[d]	**109**	**66**	**66**	**39**	**39**	**39**	**27**	**27**	**27**
Average of Individual Risk[e]	**4.4×10^{-7}**	**2.5×10^{-7}**	**2.5×10^{-7}**	**1.4×10^{-7}**	**1.4×10^{-7}**	**1.4×10^{-7}**	**9.6×10^{-8}**	**9.6×10^{-8}**	**9.6×10^{-8}**
	Estimated Cancer Deaths with Albert and Chen, 1986 Upper Bound Unit Risk (1.2×10^{-5} per µg/m³) or Harris, 1983 Upper Bound Unit Risk (4.1×10^{-3} per µg/m³) These are not directly comparable to the draft EPA unit risk estimate.[g]								
Exposure (µg/m³)	1.80	1.05	-	0.60	-	-	0.39	-	-
Cancer Deaths (Albert and Chen, 1986)	77	47	47	28	28	28	19	19	19
Cancer Deaths (Harris, 1983)	26,346	15,967	15,967	9409	9409	9409	6443	6443	6443

Footnotes can be found on the following page.

Footnotes to Table ES-1.

[a]There are many inherent uncertainties in the emission estimates, exposure, and dose-response information that need to be considered when reviewing these results. These uncertainties are discussed at the end of the executive summary and in the individual chapters. Point estimates are presented due to the difficulty in reporting a range that would accurately bound the estimates. The true risk could be as low as zero or even fall above the point estimates in this table.

[b]A modified version of the MOBILE4.1 emission model, designated MOBTOX, was used to develop the nationwide emission factors. The emission factors are roughly 25-40% lower than those that would be obtained using the current version, MOBILE5a. The resulting annual average exposure estimates should not change appreciably, however, since the conversion from g/mile to $\mu g/m^3$ is based on CO as a surrogate. The CO emission factors with MOBILE5a relative to MOBILE4.1 increase roughly in proportion to the toxic emission factors.

[c]Exposures given are nationwide annual average estimates. The HAPEM-MS model was used to calculate exposures. Then for each pollutant, the HAPEM-MS derived exposures for 1990 were compared with the range of available ambient monitoring data (with adjustments applied to account for such factors as lower exposure from time spent indoors). Where the HAPEM-MS exposures fell outside the range of ambient monitoring data, an adjustment, based on comparing the modeled versus ambient data, was applied to the modeled data to match the upper end of the range. This adjustment was then applied to the HAPEM-MS derived exposures for all years. For 1,3-butadiene, the range of ambient data varied by over a factor of four; consequently, estimates of cancer incidence given here are roughly four times higher than those that would be calculated using the lower bound.

[d]The cancer risk estimates are based on plausible upper bound estimates of unit risk (in accordance with procedures referenced in the Risk Assessment Guidelines of 1986), except for benzene. This is because an established procedure does not yet exist for making "most likely" or "best" estimates of risk. The unit risk for benzene is based on human data. The cancer risk estimates are meant to be used in a relative sense to compare risks among pollutants and scenarios, and to assess trends. They are not meant to represent actual risk.

[e]Estimated annual individual risk is the cancer risk divided by the U.S. population for the year of interest. Since results are presented as national annual averages, changes in cancer incidences or deaths presented for the expanded control scenarios do not necessarily represent changes that would occur in specific areas where the strategies are implemented, such as the Northeast.

[f]The range of nationwide annual average exposures is obtained using the results of urban ambient monitoring studies. The lower end of the range is the lowest annual average study result, with an adjustment of 0.89 based on HAPEM-MS to account for nationwide exposure (i.e., incorporating estimated rural exposure), an adjustment applied to account for the motor vehicle fraction, and an adjustment of 0.622 to account for integrated exposure (i.e., time spent indoors at home, indoors at work, outdoors, and in motor vehicles). The upper end of the range is the highest annual average study result, with the nationwide and integrated exposure adjustments, but without the motor vehicle fraction adjustment. The motor vehicle adjustment is removed for the upper end since the relative contributions of motor vehicle and non-motor vehicle sources are not clear, especially for the nonroad contribution. The contribution of motor vehicles is likely to vary significantly from location to location and for pollutant to pollutant.

[g]Alternative unit risks were derived using different sets of data, models, assumptions and other parameters. Thus, they are not directly comparable.

[h]In the 1991 draft EPA formaldehyde risk assessment, EPA's Office of Pollution Prevention and Toxics presented several estimates of risk, the lowest of which is based on DPX formation in monkeys and is used in this table. Each estimate embodies a different set of uncertainties. Comments by the Science Advisory Board to OPPT strongly recommended that a rigorous discussion of these uncertainties and how they impact on the confidence for making human risk inferences be undertaken. This document remains in draft and the risk estimates have not been adopted by the agency. EPA's official unit risk remains the unit risk estimate from EPA, 1987.

reductions of other toxic compounds, like benzene and other aromatic compounds, that would offset the potential impact from increased aldehyde emissions. Uncertainties still remain regarding health effects from exposure to oxygenated fuels. Work is in progress by EPA and others to address this issue.

Alternative cancer risk estimates are also presented in Table ES-1 to illustrate the effect of alternative annual average exposure estimates and unit risk estimates. The alternative estimates are not documented in the individual chapters, although the information used to develop these estimates is extensively documented.

First, cancer incidences for 1,3-butadiene, formaldehyde, and acetaldehyde or cancer deaths for benzene and diesel particulate matter were adjusted based on a range of annual average exposures. The range of nationwide annual average exposures is obtained using the results of urban ambient monitoring studies. The lower end of the range is the lowest annual average study result, with an adjustment of 0.89 based on HAPEM-MS to account for nationwide exposure (i.e., incorporating estimated rural exposure), an adjustment applied to account for the motor vehicle fraction, and an adjustment of 0.622 to account for integrated exposure (i.e., time spent indoors at home, indoors at work, outdoors, and in motor vehicles). The upper end of the range is the highest annual average study result, with the nationwide and integrated exposure adjustments, but without the motor vehicle fraction adjustment. The motor vehicle adjustment is removed for the upper end since the relative contributions of motor vehicle and non-motor vehicle sources are not clear, especially for the nonroad contribution. The contribution of motor vehicles is likely to vary significantly from location to location and for pollutant to pollutant. Annual average exposures for each toxic from various studies are given in the individual chapters for each toxic.

Also, alternative estimates of cancer risks are provided using the single estimate of exposure from this study, but using alternative unit risk estimates either from non-EPA organizations or unapproved EPA estimates. Both the lowest and highest alternative unit risk estimates reported in this study were used to calculate the cancer risks.

Following is a synopsis of each chapter, beginning with Chapter 3.

Emission Factor Methodology

For benzene, formaldehyde, acetaldehyde, and 1,3-butadiene, available vehicle emissions data are used to estimate toxic emissions as fractions of total organic gases (TOG). TOG includes all hydrocarbons as well as aldehydes, alcohols, and

other oxygenated compounds. These fractions are then applied to an updated version of MOBILE4.1, designated MOBTOX, developed specifically to calculate in-use toxic grams per mile emission factors. (MOBTOX TOG and toxic estimates are about 25-40% lower than those that would be obtained using the recently released current version of the mobile model, MOBILE5a. As discussed later, the overall cancer risks would not change appreciably.) This approach was used because virtually all the available emission data are from low mileage, well-maintained vehicles. To simply use the g/mile data from these studies directly would likely result in a large underestimation of true emissions. Also, available data suggest relatively constant fractions (toxics/TOG) independent of TOG emission level.

For diesel particulate matter, recent analyses performed by Navistar Corporation were used to predict total grams of urban diesel particulate matter, as well as national fleet average emission factors, for base control scenarios in the years 1990, 1995, 2000, and 2010. Navistar's analyses generally agree with previous but far less comprehensive EPA analyses. These predictions utilize the most recent inputs available; thus, the particulate emission factors derived by Navistar were used with only minor adjustments to develop diesel particulate matter risk estimates. Later, EPA may develop particulate emission factors to use in developing risk estimates independently.

For gasoline particulate matter, the available emission data were reviewed. The limited data appear to indicate a correlation between exhaust HC and gasoline particulate matter emissions. Gasoline particulate matter was thus estimated to be 1.1% of exhaust HC. It should be noted, however, that this is extremely uncertain and subject to change. This percentage was then used in the MOBTOX model to calculate in-use g/mile emission factors for gasoline particulate matter.

Exposure Methodology

Annual average exposures to toxic air pollutants from motor vehicles were estimated using a model referred to as the Hazardous Air Pollutant Exposure Model for Mobile Sources, or HAPEM-MS, developed by International Technology under an EPA contract. The annual average exposures estimated by HAPEM-MS represent the 50th percentiles of the population distributions of exposure, i.e., half the population will be above and half below these values. HAPEM-MS accounts for time spent indoors and in various microenvironments. It uses carbon monoxide (CO) as a surrogate for motor vehicle emissions, since the vast majority of CO comes from motor vehicles. HAPEM-MS calculates urban and rural annual average exposure to CO for the year 1988, using data from fixed site monitors, personal monitoring studies and personal activity studies. Fixed site monitor values were adjusted using microenvironmental CO measurements from personal

exposure monitors. The MOBILE4.1 emissions model was used to estimate the corresponding CO emission factor (g/mile) for 1988. The urban and rural concentrations predicted by HAPEM-MS for 1988 were divided by the 1988 MOBILE4.1 emission factor to get g/mile to $\mu g/m^3$ conversion factors for urban and rural areas. To obtain exposure estimates for the toxic of interest, these conversion factors were simply multiplied by the emission factor for the toxic of interest. An additional adjustment factor was applied to account for the increase in vehicle miles travelled (VMT) in excess of the population increase for the year of interest relative to 1988, since HAPEM-MS does not account for changes in VMT.

The premise of the HAPEM-MS model is that the dispersion and atmospheric chemistry of the toxic of interest is similar to CO. This premise will not be valid for the more reactive pollutants such as 1,3-butadiene, in part because such pollutants typically have significant indoor sinks relative to non-reactive compounds such as CO.

Also, the reliability of the present methodology depends on the representativeness of the population by 6 cohorts which are exposed to concentrations within 5 microenvironments. Based on the study of available exposure measurements, the upper 10th percentile of the population exposures is believed to be underestimated. The present use of annual average concentrations to determine cancer risk assumes that the dose-response relationship is linear. Improved methodology must be developed before a non-linear dose-response relationship could be used. Also, assessing chronic non-cancer effects will require consideration of a distribution of annual exposures (e.g., the 90th percentile) and not simply the annual mean average.

If MOBILE5a CO emission factors were used in estimating g/mile to $\mu g/m^3$ conversion factors, the factors would be 30-35% lower. However, as discussed earlier, the toxic emission factors using MOBILE5a would be 25-40% higher; thus, the overall cancer risk estimate would not change appreciably.

To check the reasonableness of the HAPEM-MS modeling results, the urban HAPEM-MS concentrations for 1990 were compared to urban ambient monitoring data for recent years. Monitoring data from the EPA Aerometric Information Retrieval System (AIRS), the Urban Air Toxic Monitoring Program (UATMP), and the National Ambient Volatile Organic Compounds (NAVOC) Data Base were used. The monitoring data used in this study are annual average exposures (arithmetic means) for each database and year. In order to directly compare the ambient and modeled concentrations, the ambient data were adjusted in two ways. First, the ambient monitoring data were adjusted to represent the amount that is attributed to motor vehicles, using emissions inventory apportionment. Second, the estimated ambient motor vehicle level

was adjusted to account for integrated exposure, i.e., time spent indoors at home, indoors at work, outdoors, and in motor vehicles. The latter 'integrated' adjustment factor was estimated, based on CO exposure, to be 0.622. The following sections on specific air toxics compare the HAPEM-MS modeling results to the ambient data, using these adjustments.

Short-term, high level microenvironment exposures are also addressed and compared to exposures for which non-carcinogenic health effects have been observed. For many individuals, the greatest source of microenvironmental exposure is the personal garage. EPA's model for personal garage exposure is presently being reevaluated; thus, microenvironment exposure in the following sections focus on available studies where toxics concentrations have been measured in-transit and in other microenvironments where elevated levels would be expected. The inhalation Reference Concentration (RfC) methodology provides a tool making chronic noncancer assessments. The study reports RfCs for two pollutants; diesel particulate matter and acetaldehyde. New methodology must be developed before risks to acute exposures can be assessed.

Benzene

Benzene is a clear, colorless, aromatic hydrocarbon which is both volatile and flammable. Benzene is present in both exhaust and evaporative emissions. The TOG percentage of benzene in the exhaust varies depending on control technology and fuel composition but is generally about 3 to 5%. The TOG percentage of benzene in the evaporative emissions also depends on control technology (e.g., whether the vehicle has fuel injection or a carburetor) and fuel composition (e.g., benzene level and RVP) and is generally about 1%. Control techniques are available and in use for both evaporative and exhaust emissions of benzene.

Motor vehicles account for approximately 60% of the total benzene emissions, with the remainder attributed to nonroad mobile sources (25%) and stationary sources (15%). Many of the stationary sources are industries producing benzene, sometimes as a side product, and those industries that use benzene to produce other chemicals.

EPA's Total Exposure Assessment Methodology (TEAM) Study identified the major sources of exposure to benzene for much of the U.S. population. The most important source of benzene exposure is active smoking of tobacco, accounting for roughly half of the total population exposure to benzene, which is over and above that from motor vehicles. Outdoor concentrations of benzene, due mainly to motor vehicles, account for roughly one-quarter of the total. Benzene is the only motor vehicle-related toxic for which such information exists.

Benzene is quite stable in the atmosphere. The only benzene reaction which is important in the lower atmosphere is the reaction with OH radicals. Yet even this reaction is relatively slow. The

products of this reaction are primarily phenols and aldehydes, which react quickly and also are removed by incorporation into rain. Benzene itself will not be incorporated into clouds or rain to any large degree because of its low solubility. Benzene is not produced by atmospheric reactions.

Atmospheric residence times for benzene were calculated for four cities and two seasons. In the summertime, the daytime residence times under clear-sky conditions are calculated to be 1–2 days. Under these conditions, benzene can be transported far from source regions. At night, benzene can be considered essentially inert. Winter residence times in most cases are greater than summer residence times by roughly a factor of ten. The presence of cloud cover slows down photochemistry and increases the residence time for all species.

Urban Airshed Model simulations for a hypothetical day in the summer of 1990 in St. Louis demonstrated the role of atmospheric transformation in determining ambient concentrations of benzene. In the case of benzene, atmospheric transformation was shown to have only a minor effect on ambient concentrations during afternoon hours, and virtually no effect during other times of day. Simulations in the Baltimore-Washington area indicated that the motor vehicle-related concentration of ambient benzene would be higher in winter, due to less atmospheric transformation. Simulations in Baltimore-Washington predicted significant decreases in ambient levels of benzene with use of reformulated gasoline, on the order of 7 percent. However, simulations for the summer Houston episode predicted little effect on maximum daily average concentration of benzene with use of reformulated gasoline at the site of maximum concentration.

The annual average ambient level of benzene ranges from 4.13 to 7.18 $\mu g/m^3$, based on urban air monitoring data. Applying the motor vehicle adjustment factor of 0.60 and the integrated adjustment factor of 0.622, the integrated motor vehicle exposure is estimated to range from 1.54 to 2.68 $\mu g/m^3$. Since the HAPEM-MS 1990 base control number matches the upper end of the range, the HAPEM-MS 1990 base control level of 2.67 $\mu g/m^3$ will be used to estimate cancer deaths. As a result, the HAPEM-MS exposures were used as a reasonable estimate of the annual motor vehicle exposure level of benzene for all scenarios and years.

Based on the available exposure data, maximum microenvironment exposure levels to benzene range from 40 $\mu g/m^3$ from in-vehicle exposure to 288 $\mu g/m^3$ from exposure during refueling. However, information on health effects from short-term acute exposure to benzene is limited; thus, the impact of such microenvironmental exposure is difficult to assess.

Long-term exposure to high levels of benzene in air has been shown to cause cancer of the tissues that form white blood cells

(leukemia), based on epidemiology studies with workers. Leukemias and lymphomas, as well as other tumor types, have been observed in experimental animals that have been exposed to benzene by inhalation or oral administration. Exposure to benzene has also been linked with genetic changes in humans and animals. Based on this evidence, EPA has concluded that benzene is a Group A, known human carcinogen. The International Agency for Research on Cancer (IARC) has also classified benzene as a human carcinogen. EPA calculated a cancer unit risk factor for benzene of $8.3\times10^{-6}(\mu g/m^3)^{-1}$ based on the results of three epidemiological studies in benzene-exposed workers in which an increase of death due to nonlymphocytic leukemia was observed. EPA's Office of Research and Development has just recently started the process to review and update the benzene risk assessment.

Since the benzene cancer risk assessment was conducted by EPA in 1985, several new epidemiological studies have been published. Generally, these studies are updates of the studies considered by EPA. The updated studies provide continued evidence of the carcinogenicity of benzene in humans, and incorporation of increased study population sizes and improved exposure analyses in these studies may strengthen the current cancer risk assessment for benzene. New animal studies provide additional support for the carcinogenicity of benzene in animals by both the oral and inhalation routes and provide the first animal model for the type of cancer identified most closely with occupational exposure, acute myelogenous leukemia.

Recent research has also been conducted on the pharmacokinetics of benzene. These studies demonstrate that species differ with respect to their ability to metabolize benzene. These differences may be important when choosing an animal model for human exposure and when extrapolating high dose exposures in animals to the low levels of exposure typically encountered in occupational situations. The recent development of a physiologically-based pharmacokinetic model for benzene should help in performing interspecies and route-to-route extrapolations of cancer data. New information on the ability of benzene to alter the genetic material provides additional support for the occurrence of this effect with benzene and its metabolites. Furthermore, the occurrence of certain chromosomal aberrations in individuals with known exposure to benzene may serve as a marker for those at risk for contracting leukemia.

Alternate views and/or risk assessments generally concur with EPA's choice of epidemiological data upon which to base the cancer risk estimate, but differ with respect to the mathematical models and assumptions used to derive the risk estimate and the specific tumor incidence and/or exposure data to use. The CARB risk estimate is actually a range, with the number calculated by EPA serving as the lower bound of cancer risk and a more

conservative (i.e., higher) number, based on animal data, serving as the upper bound of cancer risk. The Clement Associates risk estimate (conducted for API) is also expressed as a range with the lower bound two orders of magnitude lower than the unit risk factor calculated by EPA; the upper bound is still approximately eight times lower than the EPA unit risk.

Please note that, unlike the other pollutants addressed in this study, the cancer unit risk estimate for benzene is based on human data. Cancer numbers are expressed as cancer deaths. The estimate of cancer deaths may underestimate cancer incidence associated with benzene, since survivorship rates are not included in the supporting studies. The 1990 base control scenario estimates the total annual average cancer deaths to be 70 deaths (59 urban, 11 rural). When comparing annual cancer deaths for the base control scenarios relative to 1990, there is a 39% reduction in 1995, a 50% reduction in 2000, and a 50% reduction in 2010. The reduction in per vehicle emissions is considerably higher, particularly in the later years. The projected increase in both population and vehicle miles traveled (VMT) from 2000 to 2010 appears to offset the gains in emissions reduction achieved through fuel and vehicle modifications.

The base control and expanded use scenarios within each year can be directly compared since the same VMT and populations are applied to both. In 1995, expanding the reformulated gasoline program reduces the cancer deaths by another 8% from the 1990 base control. The expanded use of reformulated fuels and the expanded adoption of the California program in the year 2000 produces another 6% reduction in cancer deaths, for both scenarios, when compared to 1990. Expanded reformulated gasoline use in 2010 reduces the cancer deaths by 6% relative to 1990 and by approximately 10% for the expanded adoption of California standards scenario. Like the base case comparison, the cancer deaths for the control scenarios are similar for 2000 and 2010 despite continued emissions reduction, due to the projected population and VMT increase.

A number of adverse noncancer health effects have also been associated with exposure to benzene. Benzene is known to cause disorders of the blood. People with long-term exposure to benzene at levels that generally exceed 50 ppm (162,500 $\mu g/m^3$) 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, such as red blood cells and blood platelets, leading to anemia and a reduced ability to clot. Exposure to benzene at comparable or even lower levels can be harmful to the immune system, increasing the chance for infection and perhaps lowering the body's defense against tumors by altering the number and function of the body's white blood cells. In studies using animals, inhalation exposure to benzene may also indicate that it

is a developmental and reproductive toxicant. Studies with pregnant animals show that breathing 10-300 ppm (32,500-975,000 $\mu g/m^3$) of benzene has adverse effects on the developing fetus, including low birth weight, delayed bone formation, and bone marrow damage.

Formaldehyde

Formaldehyde is a colorless gas at normal temperatures and is the simplest member of the family of aldehydes. Formaldehyde gas is soluble in water, alcohols, and other polar solvents. Formaldehyde is the most prevalent aldehyde in motor vehicle exhaust and is formed from incomplete combustion of the fuel. Formaldehyde is emitted in the exhaust of both gasoline and diesel-fueled vehicles. It is not a component of evaporative emissions. Use of a catalyst has been found to be effective for controlling formaldehyde emissions. The TOG percentage of formaldehyde in motor vehicle exhaust varies from roughly 1 to 4 percent depending on control technology and fuel composition.

The motor vehicle contribution to ambient formaldehyde levels contains both primary (i.e., direct emissions) and secondary formaldehyde (i.e., formed from photooxidation of volatile organic compounds, or VOCs). It appears that roughly 33% of formaldehyde in the ambient air may be attributable to motor vehicles. This was calculated based on the results of various studies using the following apportionment: 30% primary formaldehyde in the ambient air of which 28% is from motor vehicles and 70% secondary formaldehyde in the ambient air of which 35% is due to motor vehicles. Formaldehyde is produced in the U.S. by 13 chemical companies in 46 locations encompassing 18 states and it is used in the manufacture of four major types of resins. In addition, formaldehyde is produced as a by-product in the following types of processes: combustion (mobile, stationary, and natural sources), petroleum refinery catalytic cracking and coking, phthalic anhydride production, asphaltic concrete production, and atmospheric photooxidation of unburned hydrocarbons.

Formaldehyde exhibits extremely complex atmospheric behavior. It is present in emissions but is also formed by the atmospheric oxidation of virtually all organic species. It is ubiquitous in the atmosphere because it is formed in the atmospheric oxidations of methane and biogenic hydrocarbons. Formaldehyde is photolyzed readily, and its photolysis is an important source of photochemical radicals in urban areas. It is also destroyed by reaction with OH. An important carbon-containing product of all gas-phase formaldehyde reactions is carbon monoxide. Because formaldehyde is often the dominant source of radicals in urban atmospheres, formaldehyde concentrations have a feedback effect on the chemical residence time of other atmospheric species. Formaldehyde is highly water

soluble and participates in a complex set of chemical reactions within clouds. The product of the aqueous-phase oxidation of formaldehyde is formic acid.

Atmospheric residence times for formaldehyde were calculated for four U.S. cities and two seasons. In the summertime, the daytime residence times under clear-sky conditions are calculated to be 2–4 hours for formaldehyde. Winter residence times in most cases are greater than summer residence times by roughly a factor of ten. The presence of cloud cover slows down photochemistry and increases the residence time for all species, although the increase for formaldehyde is partially offset by its rapid in-cloud destruction due to its high water solubility. The physical removal processes of wet and dry deposition are important for formaldehyde, especially under wintertime conditions. Scavenging by falling raindrops will result in formaldehyde residence times of an hour or less in colder seasons.

Urban Airshed Model simulations for a hypothetical day in the summer of 1990 in St. Louis demonstrated the role of atmospheric transformation in determining ambient concentrations of formaldehyde. The UAM simulation showed that simulated formaldehyde concentrations were about twice as high as they would be in the absence of photochemical reactions, indicating that formaldehyde is formed more rapidly than it is destroyed in urban areas in the summertime. The simulation demonstrated that the component of the concentration due to primary emissions is small relative to the component due to secondary formation in the atmosphere. Simulations for the summer Baltimore-Washington area episode resulted in both increases and decreases in ambient formaldehyde with use of federal reformulated gasoline, with increases due to increased primary formaldehyde in near-source areas, and decreases due to decreased secondary formaldehyde in downwind areas. Use of California reformulated gasoline resulted in a decrease in secondary formaldehyde nearly three times as large as in federal reformulated gasoline scenarios, with similar primary formaldehyde increases. Simulations for the winter Baltimore-Washington area episode resulted in slight increases in ambient levels of formaldehyde with the use of federal reformulated gasoline, on the order of 1-2 percent, with a primary formaldehyde increase and a secondary formaldehyde decrease. Simulations for the summer Houston episode predicted slight increases in the simulated daily average concentration throughout most of the domain with use of federal reformulated gasoline.

The annual average ambient level of formaldehyde will be taken from the 1990 UATMP data since it is the only program that accounted for the interference of ozone in the measurement method. The resulting 1990 UATMP level is 1.71 $\mu g/m^3$. Applying the motor vehicle adjustment factor of 0.33 and the integrated

adjustment factor of 0.622, the integrated motor vehicle exposure is estimated to be 1.06 $\mu g/m^3$. The HAPEM-MS 1990 base control exposure level of 1.25 $\mu g/m^3$ must be multiplied by a factor of 0.848 to agree with the ambient data. All HAPEM-MS derived exposure levels will have this factor applied.

Any formaldehyde exposures projected by HAPEM-MS itself should be viewed with caution. The adjusted HAPEM-MS exposure estimates attempt to account for both primary and secondary formaldehyde; however, these estimates are based only on changes in primary emissions of formaldehyde. The reactivity of motor vehicle VOC emissions is likely to change with technology and fuel changes. Changes in the reactivity of these emissions, which would result in changes to secondary formaldehyde levels, cannot be accounted for by HAPEM-MS.

Based on available exposure data, maximum microenvironment exposure levels range from 4.9 $\mu g/m^3$ from exhaust exposure at a service station to 41.8 $\mu g/m^3$ from parking garage exposure. Formaldehyde is a known human irritant for the eyes, nose, and upper respiratory system at acute exposure levels as low as 62 $\mu g/m^3$, though levels below this are not necessarily free from risk. Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to formaldehyde causes an increase in the incidence of squamous cell carcinomas of the nasal cavity. Epidemiological exposure studies suggest that long-term inhalation of formaldehyde may be associated with tumors of the nasopharyngeal cavity, nasal cavity, and sinus. Based on this information, EPA has classified formaldehyde as a Group B1, probable human carcinogen. IARC concurs that formaldehyde is probably carcinogenic to humans. EPA calculated the present, and still official, cancer unit risk factor of 1.3×10^{-5} $(\mu g/m^3)^{-1}$ for formaldehyde based on the results of a study in rats in which an increase in the incidence of nasal tumors was observed. In a 1990 update of this 1987 cancer risk assessment (still in draft), EPA modified the cancer risk estimate to 6×10^{-7} $(\mu g/m^3)^{-1}$ by incorporating recent data on the quantification of DNA-protein cross-links (DPX) caused by formaldehyde in monkey nasal tissue. The binding of DNA to protein to which formaldehyde is bound, forming a separate entity that can be quantified, is considered a more accurate way to measure the amount of formaldehyde that is present inside a tissue. Cancer incidence estimates in this study use the 1987 unit risk factor, since the updated one is still not an official estimate and may change.

Please note that the cancer unit risk estimate for formaldehyde is based on animal data and is considered an upper bound estimate for human risk. True human cancer risk may be as low as zero.

Several studies in experimental animals have been published since EPA conducted the cancer risk assessment for formaldehyde in 1987. These studies confirm the previous findings of an increased incidence of squamous cell carcinomas of the nasal cavity in rats exposed by inhalation. In addition, the distribution of nasal tumors in rats has been better defined; the findings suggest that not only regional exposure but also local tissue susceptibility may be important for the distribution of formaldehyde-induced tumors. Recent epidemiological studies provide additional evidence that "modest" increases in nasopharyngeal and nasal cavity and sinus cancer risks, and possibly in lung cancer risks, have been observed among various occupational subgroups. However, the evidence for an association between lung cancer and occupational formaldehyde is tenuous, and collectively, the recent studies do not conclusively demonstrate a causal relationship between cancer and exposure to formaldehyde in humans.

Recent work on the pharmacokinetics of formaldehyde has focused on the validation of measurement of DNA-protein adducts, or cross-links (DPX) as internal dosimeters of formaldehyde exposure (as discussed above). An internal dosimeter for formaldehyde exposure is desirable because the inhaled concentration of formaldehyde may not reflect actual tissue exposure levels. The difference in inhaled concentration and actual tissue exposure level is due to the action of multiple defense mechanisms that act to limit the amount of formaldehyde that reaches cellular DNA. These studies have provided more accurate data with which to quantify the level of formaldehyde in the cell.

Alternate views and risk assessments have been published for formaldehyde which all use the same rat data, but differ with respect to the mathematical models and assumptions used to extrapolate from animals to humans and the methods used to estimate internal formaldehyde dose. When using only the rat data, the 1992 CARB unit risk factor delineates the lower bound of risk factors, approximately 50 percent lower than the present EPA factor, whereas, OSHA's unit risk factor, as the upper bound, is over three orders of magnitude greater than the EPA's.

The 1990 base control scenario estimates the total annual cancer incidence to be 44 cancer cases (37 urban, 7 rural). When comparing cancer incidence for the base control scenarios relative to 1990, there is a 36% reduction in 1995, a 52% reduction in 2000, and a 50% reduction in 2010. The reduction in per vehicle emissions is considerably higher, particularly in the out years. The projected increase in both population and vehicle miles traveled (VMT) from 2000 to 2010 appears to offset the gains in emissions achieved through fuel and vehicle modifications.

The expanded use scenarios provide either no decrease or a slight increase in the cancer cases. This is generally due to the fact that increased use of oxygenates in gasoline will increase direct formaldehyde emissions.

Noncancer adverse health effects associated with exposure to formaldehyde in humans include irritation of the eyes and nose (0.1-1.0 ppm or 123-1230 $\mu g/m^3$), throat (0.05-2.0 ppm or 62-2,460 $\mu g/m^3$), and lower airway at low levels (5.0-30 ppm or 6,150-36,900 $\mu g/m^3$). There is also suggestive, but not conclusive, evidence in humans that formaldehyde can affect immune function. Adverse effects on the liver and kidney have also been noted in experimental animals exposed to higher levels of formaldehyde.

1,3-Butadiene

1,3-Butadiene is a colorless, flammable gas at room temperature, is insoluble in water, and its two conjugated double bonds make it highly reactive. 1,3-Butadiene is formed in vehicle exhaust by the incomplete combustion of the fuel and is assumed not to be present in vehicle evaporative and refueling emissions. 1,3-Butadiene emissions appear to increase roughly in proportion to exhaust hydrocarbon emissions. Since hydrocarbons are decreased by the use of a catalyst on a motor vehicle, 1,3-butadiene emissions are expected to decrease proportionally. The TOG percentage of 1,3-butadiene in motor vehicle exhaust varies from roughly 0.4 to 1.0 percent depending on control technology and fuel composition.

Current EPA estimates indicate that mobile sources account for approximately 94% of the total 1,3-butadiene emissions. The remaining 1,3-butadiene emissions (6%) come from stationary sources mainly related to industries producing 1,3-butadiene and those industries that use 1,3-butadiene to produce other compounds. Approximately 59% of the mobile source 1,3-butadiene emissions (56% of total 1,3-butadiene emissions) can be attributed to onroad motor vehicles, with the remainder attributed to nonroad mobile sources.

1,3-Butadiene is transformed rapidly in the atmosphere. There are three chemical reactions of 1,3-butadiene which are important in the ambient atmosphere: reaction with hydroxyl radical (OH), reaction with ozone (O_3), and reaction with nitrogen trioxide radical (NO_3). All three of these reactions are relatively rapid, and all produce formaldehyde and acrolein, species which are themselves toxic and/or irritants. The oxidation of 1,3-butadiene by NO_3 produces organic nitrates as well. Incorporation of 1,3-butadiene into clouds and rain will not be an important process due to the low solubility of 1,3-butadiene. 1,3-Butadiene is probably not produced by atmospheric reactions.

Atmospheric residence times were calculated for 1,3-butadiene for four U.S. cities and two seasons. In the

summertime, the daytime residence times under clear-sky conditions are calculated to be one hour or less for 1,3-butadiene. Under these conditions, 1,3-butadiene will generally be present in high concentrations only near source regions. At night, the residence times for 1,3-butadiene remain short under conditions conducive to the formation of NO_3 (high O_3, high NO_2, low NO), but increase dramatically under low NO_3 conditions. Winter residence times in most cases are greater than summer residence times by roughly a factor of ten. The residence time of 1,3-butadiene can exceed one day in the winter-time, especially if clouds are present. The presence of cloud cover slows down photochemistry and increases the residence time.

Urban Airshed Model simulations for a hypothetical day in the summer of 1990 in St. Louis demonstrated the role of atmospheric transformation in determining ambient concentrations of 1,3-butadiene. The afternoon concentration of 1,3-butadiene was reduced by 90 percent due to atmospheric reactions. Simulations for the summer Baltimore-Washington area episode resulted in little change in ambient concentrations of 1,3-butadiene with the use of federal reformulated gasoline. Use of California reformulated gasoline also had little impact on ambient concentrations of 1,3-butadiene. Reformulated gasoline use had very little effect on winter 1,3-butadiene ambient concentrations. Simulations for the summer Houston episode also predicted little effect on maximum daily average concentration of 1,3-butadiene with reformulated gasoline.

The annual average ambient level of 1,3-butadiene ranges from 0.12 to 0.56 $\mu g/m^3$. Applying the motor vehicle adjustment factor of 0.56 and the integrated adjustment factor of 0.622, the integrated motor vehicle exposure is estimated to range from 0.08 to 0.35 $\mu g/m^3$. The HAPEM-MS 1990 base control level of 0.48 $\mu g/m^3$ lies above this range. The HAPEM-MS 1990 base control level must be multiplied by a factor of 0.729 to agree with the upper end of the ambient data. All the HAPEM-MS derived exposure levels have this factor applied.

Based on a single study, in-vehicle exposure to 1,3-butadiene was found to average 3.0 $\mu g/m^3$. Since data on non-cancer health effects of acute 1,3-butadiene exposure are very limited, the impact of microenvironmental exposure is difficult to assess.

Long-term inhalation exposure to 1,3-butadiene has been shown to cause tumors in several organs in experimental animals. Studies in humans exposed to 1,3-butadiene suggest that this chemical may cause cancer. These epidemiological studies of occupationally exposed workers are inconclusive with respect to the carcinogenicity of 1,3-butadiene in humans, however, because of a lack of adequate exposure information and concurrent

exposure to other potentially carcinogenic substances. Based on the inadequate human evidence and sufficient animal evidence, EPA has concluded that 1,3-butadiene is a Group B2, probable human carcinogen. IARC has classified 1,3-butadiene as a Group 2A, probable human carcinogen. EPA calculated a cancer unit risk factor of 2.8×10^{-4} $(\mu g/m^3)^{-1}$ for 1,3-butadiene based on the results of a study in mice in which an increase in the incidence of tumors in the lung and blood vessels of the heart, as well as lymphomas were observed. A special factor was incorporated into these calculations to account for the actual amount of 1,3-butadiene that is absorbed following inhalation. EPA's Office of Research and Development has just recently started the process of updating the 1,3-butadiene risk assessment.

Please note that the cancer unit risk estimate for 1,3-butadiene is based on animal data and is considered an upper bound estimate for human risk. True human cancer risk may be as low as zero.

Since EPA conducted its cancer risk assessment for 1,3-butadiene in 1985, several updates of the epidemiology studies considered by EPA and one new study in humans have been published. These studies collectively show positive, though limited evidence that 1,3-butadiene may be carcinogenic in humans. A new inhalation study was conducted in mice because the study used by EPA in 1987 was limited due to high mortality occurring early in the study. The new study demonstrates the occurrence of cancer in mice at additional sites at lower concentrations of 1,3-butadiene than those used to derive the cancer unit risk factor.

Studies in animals also indicate that 1,3-butadiene can alter the genetic material. Recent studies on the genotoxic potential of 1,3-butadiene confirm the ability of 1,3-butadiene to cause these effects. Recent studies on the fate of 1,3-butadiene in the body have focused on the mechanism behind the differences in carcinogenic responses seen between species. Recent pharmacokinetic research has found marked differences among mice, rats, and human tissue preparations in their ability to metabolize 1,3-butadiene and its metabolites. The results suggest that the effective internal dose of DNA-reactive metabolites may be less in humans than in mice for a given level of exposure.

Alternate views and/or risk assessments that have been published for 1,3-butadiene differ with respect to the mathematical models and assumptions used to extrapolate from animals to humans, the methods used to estimate internal 1,3-butadiene dose, and the specific tumor incidence data to use. The cancer unit risks range from the one calculated by EPA based on pooled female mouse tumors which represents the upper bound of unit risk estimates, to the unit risk calculated by Hattis and Watson, 1987, based on total tumors in male rats, which is approximately 2500 times lower than the EPA estimate.

The 1990 base control scenario estimates the total annual cancer incidence to be 304 cancer cases (258 urban, 46 rural). When comparing cancer incidence for the base control scenarios relative to 1990, there is a 31% reduction in 1995, a 42% reduction in 2000, and a 33% reduction in 2010, which is actually an increase when compared to 2000. The reduction in per vehicle emissions is considerably higher, particularly in the later years. The projected increase in both population and vehicle miles traveled (VMT) from 2000 to 2010 appears to offset the gains in emissions achieved through fuel and vehicle

modifications. The expanded use scenarios provide little additional reduction in the cancer cases.

Exposure to 1,3-butadiene is also associated with adverse noncancer health effects. Exposure to high levels (on the order of hundreds to thousands ppm) of this chemical for short periods of time can cause irritation of the eyes, nose, and throat, and exposure to very high levels can cause effects on the brain leading to respiratory paralysis and death. Studies of rubber industry workers who are chronically exposed to 1,3-butadiene suggest other possible harmful effects including heart disease, blood disease, and lung disease. Studies in animals indicate that 1,3-butadiene at exposure levels of greater than 1,000 ppm ($2.2x10^6$ $\mu g/m^3$) may adversely affect the blood-forming organs. Reproductive and developmental toxicity has also been demonstrated in experimental animals exposed to 1,3-butadiene at levels greater than 1,000 ppm.

Acetaldehyde

Acetaldehyde is a saturated aldehyde that is a colorless liquid and volatile at room temperature. Both the liquid and the vapors are highly flammable. Acetaldehyde as a liquid is lighter than water, and the vapors are heavier than air. It is soluble in water. Acetaldehyde is found in motor vehicle exhaust and is formed as a result of incomplete combustion of the fuel. Acetaldehyde is emitted in the exhaust of both gasoline and diesel-fueled vehicles. It is not a component of evaporative emissions. Use of a catalyst has been found to be effective for controlling formaldehyde and other aldehyde emissions. Acetaldehyde emissions are presumed to be controlled to roughly the same extent as total hydrocarbon emissions with a catalyst. The TOG percentage of acetaldehyde in motor vehicle exhaust varies from roughly 0.4 to 1.0 percent depending on control technology and fuel composition.

The motor vehicle contribution to ambient acetaldehyde levels contains both primary and secondary acetaldehyde. Data from emission inventories and atmospheric modeling indicate that roughly 39% of ambient acetaldehyde levels may be attributable to motor vehicles. Acetaldehyde is ubiquitous in the environment and is naturally released. It is a metabolic intermediate of higher plant respiration and alcohol fermentation. It is also found in many flowers, herbs, and fruits and could be available for release to the ambient air. Acetaldehyde is also produced from aliphatic and aromatic hydrocarbon photooxidation reactions. Acetaldehyde is formed as a product of incomplete wood combustion in residential fireplaces and woodstoves and is released into the atmosphere by the coffee roasting process. Together these two processes accounted for 78% of the national primary acetaldehyde emissions. Manufacturing plants that produce acetaldehyde also

emit acetaldehyde, as do manufacturing plants that produce ethanol, phenol, acrylonitrile, and acetone.

The atmospheric chemistry of acetaldehyde is similar in many respects to that of formaldehyde. Like formaldehyde, it can be both produced and destroyed by atmospheric chemical transformation. However, there are important differences between the two. Acetaldehyde photolyzes, but much more slowly than formaldehyde. Acetaldehyde reacts with OH and NO_3 radicals, and produces formaldehyde and peroxyacetyl nitrate (PAN) as reaction products. Acetaldehyde is also significantly less water soluble than formaldehyde.

Atmospheric residence times for acetaldehyde were calculated for four U.S. cities and two seasons. In the summertime, the daytime residence times under clear-sky conditions are calculated to be 5 hours or less for acetaldehyde. At night, the calculated residence time of acetaldehyde ranges from 18 hours for Los Angeles to 7 days for St. Louis. Under cloudy-sky conditions, residence times increased. The resulting climatological average residence times for July were 6 to 11 hours for acetaldehyde. In the wintertime, calculated daytime, clear-sky residence times were longer, in the range of 20 to 60 hours for acetaldehyde, and relatively inert at night. The resulting climatological average residence times for January were 3 to 8 days.

Urban Airshed Modeling simulations for a summer day in 1990 in St. Louis demonstrated the role of atmospheric transformation in determining concentrations of ALD2 (an aldehyde surrogate species composed of acetaldehyde, higher aldehydes, and lower reactivity olefins with internal double bonds). In near-source areas of the modeling domain, ALD2 behaved as a primary species, with concentration peaks in the early morning and early evening. In downwind areas, however, ALD2 behaved as a secondary species, with concentration peaks in the midafternoon. The simulation suggested that motor vehicles may be a more important contributor to ambient acetaldehyde than they are to formaldehyde levels.

For Baltimore-Washington and Houston area simulations, primary and secondary acetaldehyde were modeled explicitly. Simulations for the summer Baltimore-Washington area episode resulted in decreases in ambient acetaldehyde with the use of reformulated gasoline, with little change in primary acetaldehyde and decreased secondary acetaldehyde throughout the domain. Use of California reformulated gasoline resulted in a decrease in secondary acetaldehyde roughly twice as large as in federal reformulated gasoline scenarios. In winter, motor vehicle-related acetaldehyde emissions were about the same with reformulated gasoline use. Simulations for the summer Houston episode predicted slight decreases in simulated daily average concentration of acetaldehyde throughout most of the domain with use of reformulated gasoline.

The annual average ambient level of acetaldehyde is based on only the 1990 UATMP data due to a potential measurement method ozone interference problem with the other ambient databases. The 1990 UATMP annual average exposure of 3.10 $\mu g/m^3$ will be used for the comparison to HAPEM-MS. Applying the motor vehicle adjustment factor of 0.39 and the integrated adjustment factor of 0.622, the integrated motor vehicle exposure is estimated to be 0.75 $\mu g/m^3$. When compared to the HAPEM-MS 1990 base control level of 0.36 $\mu g/m^3$, the 1990 UATMP adjusted ambient level is observed to be approximately two times greater than the HAPEM-MS base control level. The HAPEM-MS 1990 base control exposure level of 0.36 $\mu g/m^3$ must be increased by a factor of 2.09, to 0.75 $\mu g/m^3$ to agree with the ambient data. The HAPEM-MS derived exposure levels have this factor applied.

Any acetaldehyde exposures projected by HAPEM-MS itself should be viewed with caution. The adjusted HAPEM-MS exposure estimates attempt to account for both primary and secondary acetaldehyde; however, these estimates are based only on changes in primary emissions of acetaldehyde. However, the reactivity of motor vehicle VOC emissions is likely to change with technology and fuel changes. Changes in the reactivity of these emissions, which would result in changes to secondary acetaldehyde levels, cannot be accounted for by HAPEM-MS.

There is sufficient evidence that acetaldehyde produces cytogenic damage in cultured mammalian cells. Although there are only three studies in whole animals, they suggest that acetaldehyde produces similar effects *in vivo*. Thus, the available evidence indicates that acetaldehyde is mutagenic and may pose a risk for somatic cells (all body cells excluding the reproductive cells). Current knowledge, however, is inadequate with regard to germ cell (reproductive cell) mutagenicity because the available information is insufficient to support any conclusions about the ability of acetaldehyde to reach mammalian gonads and produce heritable genetic damage.

Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to acetaldehyde causes an increase in the incidence of squamous cell carcinomas of the nasal cavity. In one epidemiological study, with occupationally exposed workers, the evidence was inadequate to suggest that long-term inhalation of acetaldehyde may be associated with an increase in total cancers. Based on this information, EPA has classified acetaldehyde as a Group B2, probable human carcinogen. IARC has classified acetaldehyde as a Group 2B, possible human carcinogen. EPA calculated the cancer unit risk factor of 2.2×10^{-6} $(\mu g/m^3)^{-1}$ for acetaldehyde based on the results of the two studies in rats in which an increase in the incidence of nasal tumors was observed.

Please note that the cancer unit risk estimate for acetaldehyde is based on animal data and is considered an upper bound estimate for human risk. True human cancer risk may be as low as zero.

An alternate view and/or risk assessment has been published by CARB as a preliminary draft for acetaldehyde and differs with respect to the mathematical model and assumptions used to extrapolate from animals to humans. CARB, like EPA, has concluded that acetaldehyde is a probable human carcinogen. The UCL for unit risk for lifetime exposure calculated by CARB is 4.8×10^{-6} ppb^{-1} (2.7×10^{-6} $[\mu g/m^3]^{-1}$). CARB also calculated a range of UCL for unit risks. This range is 9.7×10^{-7} ppb^{-1} for female rats without a scaling factor to 2.7×10^{-5} ppb^{-1} for male rats with a contact area correction (1.19×10^{-6} to 3.32×10^{-5} $[\mu g/m^3]^{-1}$).

Since the acetaldehyde cancer risk assessment was conducted by EPA in 1987, little new research in whole animals and epidemiological studies have been accomplished.

The 1990 base control scenario estimates the total annual cancer incidence to be 5.3 cancer cases (4.5 urban, 0.8 rural). Cancer cases are presented here to one decimal place due to the small numbers involved. When compared to the 1990 base control, the cancer incidence decreases by 32% in 1995, 47% in 2000, and 43% in 2010, which is actually an increase when compared to 2000. The reductions are basically due to the tighter tailpipe standards specified by the Tier 1 standards. In contrast, when compared to the 1990 base control, the emission factors decrease 32% in 1995, 57% in 2000 and 62% in 2010. The difference observed between the emission factor and cancer case reductions, and the increases observed in 2010, is due to the expected increase in population and VMT, which appear to offset the emission gains achieved through fuel and vehicle modifications.

The expanded use of reformulated gasoline and the expansion of the California standards provide no significant decrease in the cancer cases and, in several scenarios, the cancer cases increase.

The new genotoxicity studies, which utilize lower concentrations of acetaldehyde, have not produced chromosomal aberration and/or cellular mutations.

Non-cancer effects in studies with rats and mice showed acetaldehyde to be moderately toxic by the inhalation route, oral, and intravenous routes. Acetaldehyde is a sensory irritant that causes a depressed respiration rate in mice. In rats, acetaldehyde increased blood pressure and heart rate after exposure by inhalation. The primary acute effect of human exposure to acetaldehyde vapors is irritation of the eyes, skin, and respiratory tract (135 ppm for 30 minutes). At low levels of

exposure (concentrations up to 100 ppm in air), inhaled acetaldehyde is rapidly absorbed and metabolized. At high concentrations (>100 to 200 ppm), irritation and ciliastatic effects can occur, which could facilitate the uptake of other contaminants. Clinical effects include reddening of the skin, coughing, swelling of the pulmonary tissue, and localized tissue death. Respiratory paralysis and death have occurred at extremely high concentrations. It has been suggested that voluntary inhalation of toxic levels of acetaldehyde would be prevented by its irritant properties, since irritation occurs at levels below 200 ppm (360,000 $\mu g/m^3$).

Acetaldehyde is only one of two air toxics in this study with a reference concentration for chronic inhalation exposure (RfC). This RfC was recently determined to be 9×10^{-3} mg/m^3 (9.0 $\mu g/m^3$ or 5×10^{-3} ppm). An RfC is an estimate of the continuous exposure to the human population that is likely to be without deleterious effects during a lifetime. As such, it is useful in evaluating non-cancer effects. The RfC was determined based on studies done with male rats, which indicated a NOAEL (no-observed-adverse-effect-level) of 150 ppm.

Based on a single study, the in-vehicle exposure level of acetaldehyde was found to average 13.7 $\mu g/m^3$ (7.6×10^{-3} ppm). The average in-vehicle exposure level from the above study is higher than EPA's RfC. However, the RfC is based on continuous exposure whereas the level observed in the study is short-term in duration.

The research into reproductive and developmental effects of acetaldehyde is based on intraperitoneal injection, intravenous, or oral administration of acetaldehyde to rats and mice, and also *in vitro* studies. However, little or no research into effects of inhalation of acetaldehyde on reproductive and development effects was found. The *in vivo* and *in vitro* studies provide evidence to support the fact that acetaldehyde may be the causative factor in birth defects observed in fetal alcohol syndrome.

Diesel Particulate Matter

Diesel exhaust particulate matter consists of a solid core composed mainly of carbon, a soluble organic fraction, sulfates, and trace elements. Light-duty diesel engines emit from 30 to 100 times more particles than comparable catalyst-equipped gasoline vehicles. Diesel particulate matter is mainly attributable to the incomplete combustion of fuel hydrocarbons. Lubricating oil also contributes significantly to diesel particulate matter. Some may be due to other fuel components as well. The particles may also become coated with adsorbed and condensed high molecular weight organic compounds.

The control of diesel emissions can take three forms. The first is controlling emissions before they are formed with engine modifications (such as altered combustion chamber shape, modified injection systems, or improved engine manufacturer specifications and engine seals to reduce the contribution of lubricating oil). Such modifications are in various stages of development. A second way to control emissions is to add aftertreatment technologies to the exhaust system. A third way to control emissions is by reformulation of diesel fuel.

Diesel particulate matter itself has not been explicitly modeled to determine its atmospheric transformation and residence times. Residence time calculations have been done with hypothetical non-reactive particulate-phase polycyclic organic matter (POM) for four U.S. cities and two seasons. The residence time calculated for this hypothetical particle under clear-sky summer conditions was 60 hours. In the winter, the residence time increases to 120 hours. Under rainy conditions, residence times decreased dramatically for all POM that are particle based ranging from 0.5 to 4 hours. A climatological average of 12 to 70 hours was determined for the non-reactive particulate-phase POM.

The explicit Urban Airshed Modeling of the non-reactive particulate-phase POM is difficult to achieve due to the inherent complexity of diesel emissions itself. Major consideration needs to be given to the relative abundance of the various POM species in the atmosphere, the availability of emissions data, and determining an area's specific area, mobile, and point sources. Due to these many considerations and parameters, and the absence of software to implement these factors, Urban Airshed Modeling was not done for diesel particulate matter in St. Louis. However, POM was treated explicitly in the Baltimore-Washington and Houston area studies.

To obtain urban and rural annual average exposures, urban diesel particulate matter national fleet average emission factors were first multiplied by the urban and rural g/mile to $\mu g/m^3$ conversion factors obtained from HAPEM-MS for 1988. This provides an estimate of urban and rural exposure relative to the number of vehicle miles travelled (VMT) in 1988. To obtain exposure estimates for the years of interest, these values were then multiplied by incremental adjustments to allow for the VMT increase in excess of the population increase for the year of interest. Resulting nationwide annual average exposures range from 1.80 to 0.39 $\mu g/m^3$, for the period 1990 to 2010. HAPEM-MS exposure estimates compare well to adjusted ambient data; therefore, no further adjustment was made to the modeled data.

Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to high levels of diesel exhaust causes an increase in the induction of lung tumors in two strains of rats and two strains of mice. In two key epidemiological studies on railroad workers occupationally exposed to diesel exhaust, it was observed that long-term inhalation of diesel exhaust produced an excess risk of lung cancer. Collectively, the epidemiological studies show a positive, though limited, association between diesel exhaust exposure and lung cancer.

Recently published, or soon to be completed studies have concentrated on the hypothesis that the carbon core of diesel

particulate matter is the causative agent in the genesis of lung cancer. By exposing rats to carbon black and diesel soot and comparing the results to diesel exhaust itself, the tumor response to diesel exhaust and carbon black is qualitatively similar. Also, as a result of extensive studies, the direct-acting mutagenic activity of both particle and gaseous fractions of diesel exhaust has been shown. Based on the above information, EPA has classified diesel exhaust as a Group B1, probable human carcinogen. IARC concurs that diesel exhaust is probably carcinogenic to humans. EPA calculated a cancer unit risk factor for diesel exhaust based only on exposure to the carbon core of the particle from three rat inhalation studies. The unit risk (though still draft and subject to change) of 1.7×10^{-5} $(\mu g/m^3)^{-1}$ was determined from a geometric mean of the unit risks from these three studies.

An attempt was made by EPA to develop a unit risk estimate for lung cancer based on human epidemiological data. Using these data, EPA carried out more than 50 analyses of the relationship between diesel exhaust exposure and tumor incidence. None of these analyses demonstrated a pattern that was consistent with an association between diesel exhaust exposure and lung cancer. The inability to obtain an adequate dose response was attributed to the limitations regarding exposure estimates for the various job categories, coupled with the small increases in lung cancer mortality. Consequently, it was concluded that the data are inadequate for quantitative risk assessment, based on human epidemiological data.

An understanding of the pharmacokinetics associated with pulmonary deposition of diesel exhaust particles and their adsorbed organics is critical in understanding the carcinogenic potential of diesel engine emissions. The pulmonary clearance of diesel exhaust particles has multiple phases and involves several processes including a relatively rapid transport system and slow macrophage-mediated processes. The observed dose-dependent increase in the particle burden of the lungs is due, in part, to an overloading of alveolar macrophage function. The resulting increase in particle retention has been shown to increase the bioavailability of particle adsorbed mutagenic and carcinogenic components such as benzo[a]pyrene and 1-nitropyrene. Experimental data also indicate the ability of the alveolar macrophage to metabolize and solubilize the particle-adsorbed components. Although macromolecular binding of diesel exhaust particle-derived POM and the formation of DNA adducts following exposure to diesel exhaust have been reported, a quantitative relationship between these and increased carcinogenicity is not available.

Alternate views and/or risk assessments based on rat data generally concur with EPA's unit risk estimate, but differ with respect to the mathematical models and assumptions used to derive

the risk estimate. The lower bound of other risk estimates is approximately 1.5 times lower than the EPA draft unit risk, whereas, the upper bound is approximately 5 times higher than the EPA unit risk. By using the comparative potency method, all the risk estimates determined (except one) fall in the range presented by the rat data.

The 1990 base control scenario estimates the total annual cancer deaths to be 109 (92 urban, 17 rural). When comparing the annual cancer deaths for the base control scenarios relative to 1990, there is a 39% reduction in 1995, a 64% reduction in 2000, and a 75% reduction in 2010. The reduction in the emission factor is considerably higher, particularly in later years. In this case, the projected increase in both population and vehicle miles traveled (VMT) from 2000 to 2010 does not completely offset the gains in emissions achieved through fuel and engine modifications.

A number of adverse noncancer health effects have also been associated with exposure to acute, subchronic, and chronic diesel exhaust at levels found in the ambient air. Most of the effects observed through acute and subchronic exposure are respiratory tract irritation and diminished resistance to infection. Increased cough and phlegm and slight impairments in lung function have also been documented. Animal data indicate that chronic respiratory diseases can result from long-term (chronic) exposure to diesel exhaust. It appears that normal, healthy adults are not at high risk to serious noncancer effects of diesel exhaust at levels found in the ambient air. The data base is inadequate to form conclusions about sensitive subpopulations.

The reference concentration for chronic inhalation exposure (RfC) for diesel particulate matter has only recently been established. This RfC was determined to be 5.0×10^{-3} mg/m^3. As previously mentioned, an RfC is an estimate of the continuous exposure to the human population that is likely to be without deleterious effects during a lifetime. As such, it is useful in evaluating non-cancer effects. The RfC for diesel particulate matter was estimated based on studies with rats exposed to particulate matter from light-duty and heavy-duty diesel vehicles, with an NOAEL of 0.46 mg/m^3 (0.26 ppm). Details on the derivation of this RfC can be found in Chapter 9.

Recent epidemiological studies seem to indicate that PM_{10} (particulate matter less than 10 microns in diameter) might influence daily mortality rates at concentrations lower than the ranges encountered in the earlier studies. In particular, several studies that examined PM_{10} pollution found that the relative risk of daily mortality increases in a generally linear fashion with increasing concentrations of PM_{10}. In some cities, the association was seen between PM_{10} and mortality even when particle levels never violate the current standard. These recent

studies emphasize the lack of an apparent threshold, and indicate that PM_{10} may be influencing mortality even at levels well below the current standard of 150 $\mu g/m^3$.

Gasoline Particulate Matter

Gasoline exhaust particulate matter consists of a solid core probably composed mainly of carbon, a soluble organic fraction, sulfates, and trace elements. The remaining chemical and physical properties of gasoline particulate matter are very similar to those of diesel particulate matter. Gasoline particulate matter is formed as a result of incomplete combustion of gasoline. Lubricating oil and other fuel hydrocarbons may also contribute. The sulfate particles are mostly emitted from catalyst equipped vehicles using unleaded gasoline. At present, there are no motor vehicle standards being implemented for gasoline particulate matter, though new standards that take effect in 1994 will limit particulate matter to 0.08 g/mile for all light-duty engines.

Gasoline particulate matter has not been explicitly modeled to determine its atmospheric transformation and residence times. Residence time calculation for gasoline particulate matter would be expected to be similar to the non-reactive particulate-phase POM that was described under diesel particulate matter.

Simulations for the summer Baltimore-Washington area episode resulted in slight decreases in POM with the use of federal reformulated gasoline. California reformulated gasoline resulted in larger POM decreases than federal reformulated gasoline, because of reductions in the T_{90} distillation point of the fuel. Motor vehicle-related POM concentrations with federal reformulated gasoline use decreased more in winter than in summer. Simulations for the summer Houston episode predicted larger decreases than in the Baltimore-Washington area with the use of reformulated gasoline.

Because gasoline particulate matter is emitted at such low levels, it is difficult to measure accurately. The available emissions data are limited and scattered. Furthermore, all the available data, with the exception of one study, apply to 1986 and prior model year vehicles. Since this study is meant to provide a prospective look at emissions, data from the only study which includes post-1986 model year vehicles was used solely. Data from the other studies were used as support. Data from this study indicate that gasoline particulate matter is roughly 1.1% of exhaust hydrocarbons. This percentage was used as input to MOBTOX and applied to all gasoline vehicle categories.

At this time, there exists no official EPA document detailing the carcinogenicity evidence relating to gasoline particulate matter. Much of the information is found in several

sources, some relating to particles in general and others focusing on the organic compounds associated with gasoline particulate matter.

The information on the actual carcinogenicity of gasoline particulate matter is based mainly on *in vitro* and *in vivo* bioassays. This information is based on gasoline particulate matter collected from two vehicles, one using leaded fuel and the other using unleaded fuel. The organic material was extracted from the particles and used in the bioassays. In the four *in vitro* bioassays conducted to determine DNA damage (recombination, chromatid exchanges, unscheduled DNA repair, and sister chromatid exchanges), the gasoline particulate organics did produce DNA strand breaks and sister chromatid exchanges. There was no evidence to support chromosomal aberrations in any of the related studies.

In the *in vivo* bioassays, the organics extracted from the gasoline particles were able to transform embryonic cells into malignant cells. The most critical of the *in vivo* bioassays, skin tumor initiation in mice, produced both benign and malignant tumors. This assay is critical because of the fact that it is used to determine a unit risk for gasoline particulate matter using the comparative potency method.

At the present time, there is only a unit risk based on the comparative potency method (no human data) and an EPA classification does not exist. The comparative potency method uses epidemiological data from coke oven emissions, roofing tar emissions, and cigarette smoke and develops a correlation with the gasoline particulate organics based on the relative potencies in the mouse skin tumor initiation assay. This process then determines the unit risk. For the automobile with a catalyst using unleaded fuel, the unit risks are 1.2×10^{-4}(µg organic matter/m^3)$^{-1}$ and 5.1×10^{-5}(µg particulate matter/m^3)$^{-1}$. For the automobile without a catalyst using leaded fuel, the unit risk is 1.6×10^{-5}(µg particulate matter/m^3)$^{-1}$. IARC has no potency for gasoline engine
exhaust but has classified gasoline engine exhaust as a Group 2B carcinogen, i.e., possibly carcinogenic to humans.

Although gasoline engine emission particulate matter is similar to diesel exhaust in terms of chemical and most physical properties, the cancer unit risk estimate for gasoline engine exhaust is based on the comparative potency method rather than particles, for a number of reasons. The comparative potency method is believed, at present, to be the most logical approach for estimating cancer risk from gasoline engine exhaust because, first, the EPA's particle based unit risk estimate is not an official estimate and is subject to change. Also, while the composition of gasoline exhaust particulate matter may be similar to that of diesel exhaust, the particles are considerably

smaller. Cancer potency may therefore differ from diesel exhaust because of greater particle surface area per unit volume and because of altered deposition patterns. Finally, since no chronic inhalation bioassays have been carried out on gasoline engine emissions, a particle based cancer risk estimate, using the same methodology as for diesel would contain a considerable degree of uncertainty.

The cancer incidences calculated below are based on extremely uncertain emissions data, exposure estimations, and an unofficial EPA unit risk estimate. The unit risk estimate, as mentioned above, is based on the mutagenicity of the extractable organics from the particles in the comparative potency method using only the emissions from one unleaded gasoline vehicle. Due to these factors, the cancer incidences discussed below should be considered *pro forma* and will not be presented in the executive summary table which details cancer incidences/deaths due to motor vehicles.

For estimating annual *pro forma* cancer incidence, the gasoline unit risk for catalyst vehicles based on the comparative potency method was used. It should be pointed out that the unit risk is expressed in terms of whole particles, although potency is estimated based on the organic fraction. Nationwide annual average exposures for the 1990, 1995, 2000, and 2010 base control scenarios, estimated using the HAPEM-MS model, were 0.51, 0.29, 0.20, and 0.17 $\mu g/m^3$, respectively.

The 1990 base control scenario estimates the total annual average *pro forma* cancer incidence to be 93 cancer cases (79 urban, 14 rural). When comparing *pro forma* cancer incidence for the base control scenarios relative to 1990, there is a 42% reduction in cancer incidence in 1995, a 58% reduction in 2000, and a 63% reduction in 2010. The reduction in per vehicle emissions are higher, particularly in later years. The projected increase in both population and vehicle miles traveled (VMT) from 2000 to 2010 appears to offset some of the gains in emissions achieved through fuel and vehicle modifications.

No studies exist that specifically address noncancer effects of gasoline particulate matter. The studies relating noncancer effects to PM_{10} levels in general are applicable to both diesel and gasoline particulate matter.

Gasoline Vapors

Gasoline exists in two phases, liquid and vapor, with the hydrocarbon compositions being different. Gasoline vapors consist mainly of short-chained and iso-alkanes (84 to 93 percent), alkenes (2 to 6 percent), and aromatics (1 to 5 percent). In contrast, liquid gasoline consists principally of

66 to 69 percent paraffins (alkanes), 24 to 27 percent aromatics, and 6 to 8 percent olefins (alkenes).

The major sources of exposure to gasoline vapors are from service station operations and as a result of gasoline leakage from underground storage tanks. The principal exposure pathways are from the ambient air, gasoline migration into the basements of homes, and the ingestion of gasoline contaminated groundwater. The populations that receive the greatest exposure in the chain of fuel handling are refinery workers, bulk fuel truck drivers, service station attendants, self-service customers, and residents of neighborhoods close to refineries, bulk storage terminals, and service stations.

Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to wholly vaporized gasoline induced a significant increase in renal carcinomas in the kidney cortex of male rats and also a significant increase in liver carcinomas in female mice. Female rats and male mice had no significant treatment related induction of tumors at any organ site. The incidence of renal carcinomas was significantly increased only at the highest dose tested. Epidemiological studies in occupationally exposed workers suggest that long-term inhalation of gasoline vapors may be associated with certain types of cancer. However, the epidemiologic evidence for evaluating gasoline as a potential carcinogen is considered inadequate. Mutational bioassays performed *in vivo* in animals and epidemiological studies provided negative or inconclusive results on the mutagenicity of gasoline vapors. Based on this information, EPA has classified gasoline vapors as a Group B2, probable human carcinogen. EPA calculated a range of unit risk factors of 2.1×10^{-3} to 3.5×10^{-3} $(ppm)^{-1}$ for gasoline vapors based on the results of a study indicating an increase in the incidence of kidney tumors in male rats exposed to wholly vaporized gasoline.

Several studies in experimental animals have been published since EPA conducted the cancer risk assessment for gasoline vapors in 1985. These studies confirm the previous findings of an increased incidence of kidney tumors in male rats exposed by inhalation to whole gasoline vapor. Several studies tested only the lighter hydrocarbons, which would be more characteristic of the major fraction of gasoline vapor, and found no evidence of nephrotoxicity in rats. Recent epidemiological studies do not provide supportive evidence of a causal relationship between cancer and exposure to gasoline vapors in humans. Recent genotoxicity assays generally do not support the concept of the mutagenicity of gasoline vapors.

Much, but not all, of the pharmacokinetic data that have been generated since the publication of the 1985 EPA risk assessment has been devoted to trying to determine the mechanism

involved in the development of the chemically-induced kidney tumors observed in the male rat. A recent EPA report, Alpha$_{2u}$-globulin: Association with Chemically Induced Renal Toxicity and Neoplasia in the Male Rat, provided Agency-wide guidelines for evaluating renal tumors in the male rat. When evaluating a possible nephrotoxic chemical, if the nephrotoxicity involves the accumulation of the protein alpha$_{2u}$-globulin in the kidney, then the tumor incidence should not be used, since this series of events is specific to the male rat. This EPA policy is an important change in EPA's general approach to cancer risk assessment and may affect the current EPA position on gasoline vapor carcinogenicity.

Alternate views and/or risk estimates have been published for gasoline vapors since the EPA risk assessment in 1985. In a series of studies and/or evaluations, it has been found that the lighter hydrocarbons were not nephrotoxic, the epidemiological evidence is weak, and there was no proof of an association between exposure to petroleum vapors and increase in kidney cancer. NESCAUM (Northeast States for Coordinated Air Use Management) determined individual lifetime cancer risks associated with exposure to unleaded gasoline, ranging from 1.1×10^{-5} to 6.3×10^{-3} risk/person/lifetime.

The baseline average annual cancer incidence from exposure to gasoline vapor was conducted by EPA in a 1987 draft regulatory impact analysis. The gasoline vapor risk values determined in this document use the EPA unit risk for wholly vaporized gasoline. The values, presented as the average annual values for the study period of 1988 to 2020, range from a low of 1.3 cancer cases from exposure at bulk plants to a high of 51 cancer cases due to the exposure of the public at service stations.

EPA has not initiated any specific effort to re-examine the weight-of-evidence for gasoline vapors based on the new tumor evaluation criteria. It may seem timely to review the data for gasoline because of the new criteria. However, re-examination would not be limited to evaluating the kidney tumor position. EPA would also consider other newly available data relevant to the overall framework of weight-of-evidence evaluation including epidemiological data, toxicology data on non-cancer endpoints, mechanism of action, information for complex mixtures, and chemical specific information on gasoline components. It is possible that the resulting classification could be lower, higher, or unchanged, based on this comprehensive review.

When considering the other views and the recent and ongoing research it is reasonable to assume that the values mentioned above are conservative and more highly uncertain than the risk estimates for the other pollutants examined in this study. Due to this fact, these values are considered *pro forma* and will not

be presented in the executive summary table which details cancer incidences/deaths due to motor vehicles.

EPA's Integrated Air Cancer Project

The Integrated Air Cancer Project (IACP) is an EPA interdisciplinary research program aimed at identifying the major carcinogenic chemicals emitted into the air, the specific sources of these chemicals and the impact on humans of exposure to ambient concentrations of these chemicals. The IACP research strategy was designed to focus on products of incomplete combustion (PICs). PICs include polycyclic organic matter (POM), primarily absorbed to respirable particles. This POM comprises most of the human cancer risk of PICs.

The IACP has primarily taken the approach of measuring the mutagenicity of ambient air samples and apportioning this mutagenicity to sources. The IACP has looked at apportionment in Raleigh, North Carolina; Albuquerque, New Mexico; and Boise, Idaho. In Boise, the IACP has also assessed exposure from airborne carcinogens based on ambient measurements and human time-activity profiles, analyzed the role of atmospheric transformation on mutagenicity, and estimated human cancer risk using the comparative potency method. A field study has also been conducted in Roanoke, Virginia, but to date, little analysis has been done.

Mutagenicity studies focused on extractable organic material (EOM) obtained from samples. EOM is basically the amount of particulate organic material that can be extracted from ambient air samples collected on filters using methylene chloride. Some mutagenicity studies were also done on semivolatile organic compounds (SVOCs), extracted from ambient air samples using an absorbent known as XAD-2. In addition, volatile organic compounds (VOCs) were collected in canisters, and in the Boise study, mutagenicity was measured before and after irradiation to determine the effects of atmospheric transformation.

For EOM, the IACP approach involves collection of ambient air samples on filters and extraction of organic material. Then, detailed chemical characterization is done using gas chromatography and other techniques. Next, mutagenicity is determined using the *Salmonella* mutagenicity assay, and apportioned using the receptor model approach, involving the use of chemical tracers to identify sources. The procedure for measuring mutagenicity in SVOCs and VOCs varies somewhat, due to the different collecting techniques.

Human exposure estimates from the Boise study indicate that mobile sources account for about 27% of the annual EOM exposure. Furthermore, the mutagenic potency of EOM from mobile sources was roughly three times higher than for woodsmoke, and the lifetime

unit risk for mobile sources, based on the comparative potency method, was roughly two and a half times higher than for woodsmoke. Thus, mobile sources account for 56% of the mutagenicity of EOM in Boise, as well as 20% of the mutagenicity in Raleigh and 36% in Albuquerque. In larger cities, where mobile sources would be expected to contribute a greater proportion of the ambient EOM, this contribution to mutagenicity would be even higher. Finally, atmospheric transformation may greatly exacerbate the risk from mobile sources, since the contribution of VOCs to mutagenicity of ambient samples increases dramatically following irradiation in a smog chamber.

Toxics Aspects of Alternative Fuels

As a result of the centrally fueled clean fuel fleet program, the new California standards, and the Comprehensive National Energy Policy Act of 1992, more alternatively fueled vehicles could possibly be added to the fleet over the next two decades. It is likely that most of these alternatively fueled vehicles would run on high level methanol/gasoline blends, neat methanol (M100), high level ethanol/gasoline blends, neat ethanol (E100), compressed natural gas (CNG), or liquid propane gas (LPG) with a small number of electric vehicles produced to meet California's zero emission vehicle (ZEV) requirement. Thus, the potential cancer reduction benefits resulting from the combustion of these alternative fuels should be addressed. Although engine technology for these fuels is still being developed, potential cancer reduction benefits can be projected with reasonable confidence based on available data.

Use of M100 in motor vehicles will result in substantial reductions (i.e., 97% or greater) or elimination of benzene, 1,3-butadiene, acetaldehyde, gasoline refueling vapors, and particulate matter. However, tailpipe emissions of formaldehyde (i.e., primary formaldehyde) will go up by about 200% for optimized vehicles, although no formaldehyde would be associated with evaporative emissions. Conversely, the use of methanol, with its lower hydrocarbon emissions, will result in decreased levels of secondary formaldehyde resulting from exhaust emissions, which is formed in the ambient air from photochemical oxidation of hydrocarbons. In fact, when improvement in methanol engine and emission control technology are considered along with secondary formaldehyde emissions reductions, no substantial increase in overall mass of formaldehyde emissions with use of M100 in dedicated vehicles is projected. However, exposure from primary emissions of formaldehyde would likely be greater than for secondary formaldehyde.

For vehicles fueled with 85% methanol, significant reductions are also expected, although these reductions are less than that for M100 vehicles. It should be noted that primary

formaldehyde emissions are much higher than those of a dedicated methanol vehicle.

A large percentage of total exhaust and evaporative organic emissions from motor vehicles running on either M100 or methanol blends is methanol itself. There is uncertainty as to whether exposures to methanol vapors that may be encountered can result in negative health effects. EPA will assess the situation as new information is developed.

Like methanol, use of ethanol as a clean fuel would result in substantial reductions in air toxics emissions. Emissions data for higher level ethanol blends and E100 vehicles are sparse though. It is likely that substantial reductions in benzene, 1,3-butadiene, refueling vapors, and particulate matter would occur, while formaldehyde would be emitted at levels similar to gasoline vehicles. Acetaldehyde emissions, on the other hand, would increase substantially. Since the acetaldehyde cancer potency (2.2×10^{-6} unit risk) is much lower than the 1,3-butadiene potency (2.8×10^{-4} unit risk), any increase in cancer incidence due to acetaldehyde would be greatly offset by the large decrease in cancer incidence due to 1,3-butadiene exposure. It should be noted, however, that acetaldehyde is an irritant and may have some chronic and acute respiratory effects. Thus, non-carcinogenic health effects of increased acetaldehyde exposure due to ethanol combustion may be a concern (to a lesser extent, this would be a concern with methanol combustion as well).

CNG use would also yield substantial air toxics benefits. Since use of CNG as a fuel requires a closed delivery system, evaporative emissions from a dedicated CNG vehicle are assumed to be zero. Also, CNG contains no benzene, so refueling and running losses of this toxic would also be zero. Moreover, exhaust emissions of benzene and 1,3-butadiene are very low. Formaldehyde and acetaldehyde exhaust emissions are roughly the same as for gasoline.

LPG is another possible alternative fuel for motor vehicles. LPG would be expected to have very little evaporative emissions. LPG has very low 1,3-butadiene and benzene emissions, but aldehyde emissions increase substantially, as with alcohol fuels. However, these higher aldehyde emissions would likely be reduced with a catalyst specifically designed for an LPG vehicle.

Nonroad Mobile Sources

The terms "nonroad engines" and "nonroad vehicles" cover a diverse collection of equipment ranging from small equipment like lawn mowers and chain saws, to recreational equipment, farm equipment, and construction machinery. Nonroad engines are not presently regulated for emissions, and very few nonroad engines currently use emission control technology. Because of the

diversity of nonroad equipment, characterization of the emissions from nonroad engines is a complex task. As a group, nonroad engines represent the last uncontrolled mobile source. The limited availability of toxic emission data for nonroad sources makes it difficult to quantify precisely the contribution to ambient air toxic levels from nonroad sources. Many toxics such as benzene, 1,3-butadiene, aldehydes, and gasoline vapors are included in the broad category of pollutants referred to as VOCs. Measures to control VOC emissions should reduce emissions of these air toxics. However, the magnitude of reduction will depend on whether the control technology reduces the individual toxics in the same proportion that total VOCs are reduced. Since nonroad vehicles have significant VOC impacts, they are expected to have significant toxics impacts as well. While Section 202(l) of the Act addresses toxic air pollutants associated with motor vehicles and motor vehicle fuels, EPA included nonroad engines and vehicle in this study for purpose of completeness.

Approximately 30% of mobile source benzene emissions, or 25% of total benzene emissions, is attributable to nonroad sources. An estimated 13% of total formaldehyde is attributable to nonroad sources, and an estimated 5% of total particulate matter is from nonroad sources. Approximately 41% of mobile source 1,3-butadiene emissions, or about 39% of total 1,3-butadiene emissions, is attributable to nonroad sources. Neither this study nor EPA's 1991 Nonroad Engine and Vehicle Emission Study provides an estimate of the nonroad contribution to total acetaldehyde emissions.

Initial Cost Considerations

EPA has not done an independent evaluation of cost considerations associated with controlling toxic emissions. Instead, this study summarizes available cost information for various regulatory programs which may result in reductions of motor vehicle-related air toxics. Cost information will be addressed more fully in any subsequent regulatory activity.

The estimate for the dollar cost/ton of volatile organic compounds (VOC) reduction as it relates to the Tier 1 Standards ranges from $3700 to $6018/ton. For the reformulated fuel program, the estimated nationwide summertime cost per ton of VOC reduced ranges from $1500 to $3700. The estimated costs for I/M programs, based on the cost of VOC reduction per ton accounting for NO_x and CO benefits, can range from $461 to $4518. EPA has not done a cost-effectiveness analysis of the California LEV Program and has not presented information on the cost per ton of VOC or toxics reductions. The report, however, provides information for the readers' benefit that was presented to EPA by various parties as part of California's request for a waiver of federal preemption, pursuant to Section 209(b) of the Clean Air Act, for the California low-emission vehicle standards and vehicle test procedures.

EPA's recent diesel particulate matter control regulations focus to a large extent on diesel fuel desulfurization (although the diesel particulate matter bus program called for in the 1990 Clean Air Act Amendments is also an important program). The diesel fuel sulfur regulation was developed to reduce the amount of diesel particulate matter emitted by heavy-duty diesel engines. The costs are expressed as cost per ton of particles reduced and were estimated using a calendar-year approach discounted over a 33-year period (1994-2025). The estimated cost assuming no engine wear credits is $2826 to $6773/ton.

The reduction in vehicle emissions basically takes two forms, exhaust and evaporative, and the regulatory programs discussed above address either one or both of these emissions. The four toxic pollutants addressed most often, benzene, 1,3-butadiene, formaldehyde, and acetaldehyde, are all produced in

the combustion process and emitted to the environment via the tailpipe. This is also true for diesel particulate matter. Only benzene contributes to the ambient level through evaporative emissions due to its presence in gasoline. Thus, those regulatory programs that are most effective in reducing exhaust emissions will be the most successful in reducing the greatest number and mass of air toxics. This is generally true assuming that gasoline is used, but the emissions do change as the fuels are modified. With many of the new fuels there will be an immediate effect on many toxic emissions (some reduced, some increased) since these programs affect all vehicles simultaneously. The exhaust emission standards will only affect vehicles from a particular model year onward and total effects will not be seen until there is a complete fleet turnover.

Motor Vehicle Toxics in Section 112(b) of the CAA and Metallic Pollutants

The list of 189 compounds in Section 112(b) of the Clean Air Act (as amended in 1990) were reviewed to identify those compounds (29 in all) that are either known or, based on their structure, have the potential to be emitted from motor vehicles. MTBE (methyl-t-butyl ether) is one of these compounds; there are a large number of programs underway to obtain health data on MTBE. Another compound in this list that may be emitted from mobile sources is 2,3,7,8-tetrachlorodibenzo-p-dioxin. The six metals chosen are all potential fuel additives. Various health-based criteria (e.g., threshold limit value [TLV], reference dose [RfD], reference concentration [RfC]) have been developed for many of these compounds. RfCs or RfDs, as determined by EPA, do not exist for fifteen of these compounds and three of the metals. This is based on the fact that EPA considers the health information inadequate or insufficient to develop the RfC or RfD that is needed. The Occupational Safety and Health Association (OSHA) and the American Conference of Governmental Industrial Hygienists (ACGIH) have established threshold limit values (TLV), and/or short-term exposure limits (STEL) for many of the compounds where EPA has yet to determine or verify a value.

HEI Air Toxics Workshop

In December of 1992, the Health Effects Institute conducted a Mobile Air Toxics Workshop to identify priorities for research that would reduce uncertainties in risk assessments for five compounds. These compounds are benzene, aldehydes, 1,3-butadiene, methanol, and POM. Also, six cross-cutting areas were identified from the various individual compound sessions. These areas are dosimetry, high-to-low dose extrapolation, epidemiology, exposure assessment, molecular biological approaches, and neurotoxic, reproductive, and developmental

effects. The final report should be available in the spring of 1993.

Limitations

This section summarizes the major limitations of analyses done in this study. These limitations need to be considered when reviewing the results of this study.

Point estimates of risk are presented due to the difficulty in reporting a range that would accurately bound the estimates. The true risk could be as low as zero or fall above the point estimates given in Table ES-1. Thus, the cancer risk estimates are not meant to be representative of actual risk. Instead, they are meant to be used in a relative sense to compare risks among pollutants and scenarios, and to assess trends. However, the degree of uncertainty in potency, emission and exposure estimates is not the same for each pollutant. A formal uncertainty analysis would be needed to quantify the certainty of risk associated with exposure to each pollutant.

For all pollutants except benzene, the cancer risk estimates are based on upper bound estimates of unit risk, determined using animal data. Uncertainties exist with regard to animal-to-human and exposure-to-dose extrapolations. Also, different interpretations of the same health data and/or use of different models often result in wide ranges in unit risk factors. There appears to be a need for more pharmacokinetic data. Recent pharmacokinetic research for benzene, formaldehyde, and 1,3-butadiene has been conducted and summarized in this study; however, these data are not reflected in the risk estimates. EPA is currently reevaluating the health data for formaldehyde, 1,3-butadiene, and benzene. An EPA risk assessment for diesel particulate matter is also in progress.

While many of the uncertainties associated with this study are likely to result in overestimates of risk, a number of uncertainties could result in underestimates. The risk assessments in this study are limited to certain components of the mixture of chemicals in the atmosphere to which individuals are exposed. Risks from mixtures of chemicals in motor vehicle emissions and mixtures resulting from the combination of emissions from motor vehicles with emissions from other sources or atmospheric transformation products are largely uncharacterized. In addition, the role of atmospheric transformation in affecting the mutagenicity and carcinogenicity of motor vehicle emissions is uncertain. Atmospheric transformation products could be important (e.g., peroxyacetyl nitrate, or PAN, acrolein, and secondary formaldehyde), especially since available smog chamber data suggest that atmospheric transformation creates significantly increased mutagenic activity.

The discussion of non-carcinogenic effects is less quantitative than the discussion of carcinogenic effects due to the lack of available health data. No attempt has been made to synthesize and analyze the data encompassed. Also, no attempt was made to accord more importance to one type of noncancer effect over another. The objective was to research all existing data, describe the noncancer effects observed, and refrain from any subjective analysis of the data. Noncancer effects associated with exposures to the pollutants discussed in this study will be important to assess.

Toxic emissions data are limited, particularly for oxygenated fuels. Furthermore, most data are only available for low mileage and/or properly maintained vehicles. In order to estimate likely real world emissions, the available emissions data for all the toxics except diesel particulate matter were expressed as a fraction of total organic gases and used in a special version of EPA's MOBILE4.1 model, called MOBTOX, to calculate toxic emission factors. The resulting toxic emission estimates are thus derived rather than taken directly from available data. In addition, many limitations are inherent in MOBTOX and the MOBILE4.1 model on which it is based.

With a prospective study like this, many uncertainties are involved with making projections. For example, the catalyst and fuel technology mixes in the future are only projections. Also, the composition of reformulated and winter oxygenated fuels and the effect of these fuels on emissions are estimated. The study assumed MTBE fuel use in areas participating in the reformulated gasoline program and oxygenated gasoline CO program, but similar toxics benefits are expected with ethanol use. Also, this study is not intended to provide a comparison of different reformulated gasoline blends.

It should be emphasized that the expanded control scenarios included in this study are not intended to be predictive, but are instead intended to encompass a wide range of possibilities. Assumptions included in the scenarios, such as types of I/M programs, percent hydrocarbon reductions associated with oxygenated fuel use, properties of reformulated fuels, and estimates of fuel use under different scenarios were made using the best available assumptions at the time the analyses were done. The effects of these assumptions are likely to be significant. Since results are presented as national annual averages, changes in cancer incidences or deaths presented for the expanded control scenarios do not necessarily represent changes that would occur in specific areas where the strategies are implemented, such as the Northeast. Area specific analyses would be valuable, but are beyond the scope of this study. In addition, the expanded control scenarios did not assess all viable national strategies for controlling air toxics from motor

vehicles. It would be useful to evaluate the benefits of transportational control measures, for example.

Estimation of exposure is somewhat uncertain. The model used in this study for estimating annual average exposure is based on carbon monoxide (CO) as a surrogate for motor vehicle emissions. This approach is particularly uncertain for the more reactive toxics such as 1,3-butadiene. Another limitation of the exposure estimation is that the model uses CO NAAQS fixed site monitoring data; however, the purpose of siting fixed site monitoring stations is not to adequately measure ambient levels of CO but to locate exceedances of the CO standard. As pointed out by several commentors, data from fixed site monitor locations are not likely to be adequate measures of ambient outdoor CO concentration in the community as a whole. As a result, the monitor values were adjusted based on personal monitoring data obtained from one city (Denver) over a four month period during the winter of 1982-1983. There is uncertainty as to whether the resulting estimates are applicable to other areas and other seasons. The same general comment also applies to the activity pattern data, which were collected in a single city (Cincinnati). Also, the fixed site monitoring data were not adjusted to account for non-motor vehicle sources of CO, since motor vehicles are thought to be the predominant source of CO in urban areas. This assumption will serve to overestimate motor vehicle exposure. On the other hand, the cohort classification scheme in the model was not intended to account for groups of people who are both highly exposed and few in number (e.g. toll booth attendants). This may underestimate the highest exposure actually experienced by the residents of the associated study area. Finally, CO data from only two rural areas were used to extrapolate to all rural areas in the U.S. There is uncertainty regarding the representativeness of these two areas.

In all cases, the HAPEM-MS derived exposures were compared to ambient monitoring data, and adjustments made to the modeled exposures to better align them with the ambient data. However, there is also uncertainty associated with the ambient databases. The sites chosen may not be representative of nationwide exposure. Also, for 1,3-butadiene in particular, there was a wide range of ambient values, spanning over a factor of four.

EPA's Total Exposure Assessment Methodology (TEAM) study identified the major sources of exposure to benzene for much of the U.S. population as well as the contributions of these sources to personal exposure. The most important source of benzene exposure is active smoking of tobacco versus vehicle exposure. Benzene is the only motor vehicle toxic for which such integrated exposure information is available. Some rough estimates have been made on formaldehyde exposure suggesting most formaldehyde exposure occurs indoors due to a large extent from the release of

formaldehyde from consumer products (e.g., particle board, carpeting, etc.).

Clearly, many limitations are inherent in the analyses used in this study to assess the health risk from motor vehicle air toxics. The EPA welcomes comments on how to reduce these limitations. Moreover, the EPA recognizes a need to explicitly address uncertainties. Future research is necessary before critical areas
of uncertainty can be explicitly addressed. EPA will consider the comments received on this study to assist in prioritizing future research planning.

Summary of Comments on Public Review Draft of Motor Vehicle-Related Air Toxics Study

Appendix I contains a summary of comments provided on the public review draft of the Motor Vehicle-Related Air Toxics Study. Many of these comments have been incorporated into the final version of the study. The remaining comments will be considered by EPA during the subsequent regulatory decision making process. Commentors on the public review draft were: the American Automobile Manufacturers Association (in conjunction with the American Petroleum Institute, the Engine Manufacturers Association and the Association of International Automobile Manufacturers), the American Petroleum Institute, Arco Chemical Company, the California Air Resources Board, the California Environmental Protection Agency, the Chemical Manufacturers Association, Ford Motor Company, General Motors Corporation, the Health Effects Institute, Konheim and Ketcham, the Northeast States for Coordinated Air Use Management, and Zephyr Consulting.

A number of commentors stated that the study needed to deal with uncertainties more explicitly. Several commentors also pointed out the need to update EPA risk assessments for formaldehyde, acetaldehyde, and 1,3-butadiene. In addition, several commentors stated that EPA should treat diesel particulate matter carcinogenesis as a threshold phenomenon. A number of comments pertained to assumptions in the HAPEM-MS exposure model. One major comment on HAPEM-MS was that fixed site monitors are not randomly chosen, but placed in locations where high CO levels are expected. Thus, an adjustment factor should be applied to CO monitor readings to make them more representative of actual exposure levels. Another major comment was that the effect of uncertainty in exposure predictions introduced through differences between the diurnal profiles of reactive air toxics and CO should be characterized. Commentors also pointed out that EPA did not adequately account for the nonroad contribution to mobile source toxic emissions, particularly for benzene and 1,3-butadiene. Finally, two commentors expressed concern that EPA did not adequately address the issue of motor vehicles (especially diesels) as a potential source of 2,3,7,8-tetrachlorodibenzo-p-dioxin emissions.

As noted earlier, this study attempts to summarize what is currently known about motor vehicle-related air toxics and to present all significant scientific opinion on each issue. This study provides an important foundation for any future regulatory decision making in this area, including decisions under Section 202 (l)(2) of the Act. While this study does not resolve the various issues discussed herein and in the public comments, EPA will continue to explore and address these in the context of such future regulatory decision making.

1.0 INTRODUCTION

1.1 Background

The U.S. Environmental Protection Agency (EPA) initially conducted a broad "scoping" study, with the goal of gaining a better understanding of the size and causes of the health problems caused by outdoor exposure to air toxics (Haemisegger et al., 1985). This study is widely referred to as the Six-Month Study since it was meant to be conducted in a six month time period. The Six-Month Study contains quantitative estimates of the cancer risks posed by selected air pollutants and their sources. The estimates of upper bound cancer incidence ranged from 1300 to 1700 cases annually nationwide for all pollutants combined. The results further indicate that mobile sources may be responsible for a large portion (i.e., up to 60 percent) of the aggregate cancer incidence.

Based on the results of the Six-Month Study, EPA's Office of Mobile Sources conducted a study that focused on cancer risks posed by air toxics emissions from motor vehicles (Carey, 1987; Carey and Somers, 1988; Adler and Carey, 1989). The nationwide aggregate upper bound risk in 1986 was estimated to range from 586 to 1650 cancer incidences and dropped roughly 30 percent by 1995. Reasons for the projected decrease in risk in 1995 include: 1) the more stringent diesel particulate standards for both light- and heavy-duty vehicles, and 2) the increasing use of 3-way catalyst-equipped vehicles coupled with the phase out of non-catalyst-equipped vehicles. The aggregate risk in 2005 was similar to that in 1995. Even though emissions per vehicle mile were predicted to decrease in 2005 relative to 1995, this appeared to be offset by increases in vehicle miles travelled and population from 1995 to 2005.

EPA's Office of Air Quality Planning and Standards sponsored a study to define the multi-source, multi-pollutant nature of the urban air toxics problem (i.e., cancer risk) in five different areas of the U.S., to determine what reduction is likely to occur as a result of ongoing regulatory activities, and to investigate what further reductions might be possible with additional controls. The study is commonly referred to as the 5 City Study. The 5 City Study was conducted in two phases, the base year analysis for 1980 (EPA, 1989) and the projection analysis for 1995 (Pechan, 1990). Motor vehicles were found to be responsible for 53 percent of the average 5 city aggregate cancer incidence in 1980 and 31 to 54 percent in 1995, depending on the control scenario.

EPA's Office of Air Quality Planning and Standards also sponsored an analysis of cancer risks in the U.S. from outdoor exposures to air toxic pollutants (EPA, 1990). The purpose of this study was to update the 1985 Six-Month Study. Based on the pollutants and source categories examined, total upper bound excess cancer cases were estimated to be between 1,700 and 2,700

per year nationwide. In this study, motor vehicles accounted for almost 60 percent of total cancer incidence.

Collectively, the results of these studies indicate that motor vehicles could be a significant contributor to excess cancer incidence from outdoor exposure to air toxic emissions.

1.2 Congressional Mandate

Section 202(l)(1) of the Clean Air Act (CAA) as amended in 1990 directs EPA to complete a study of the need for, and feasibility of, controlling emissions of toxic air pollutants which are unregulated under the Act and associated with motor vehicles and motor vehicle fuels. The study shall also address the means and measures for such controls. The study shall focus on those categories of emissions that pose the greatest risk to human health or about which significant uncertainties remain, including emissions of benzene, formaldehyde, and 1,3-butadiene. The proposed study shall be available for public review and comment and shall include a summary of all comments. The study was due May 15, 1992.

Pursuant to Section 202(l)(2), by May 15, 1995 EPA shall, based on the study, promulgate (and from time to time revise) regulations containing reasonable requirements to control hazardous air pollutants from motor vehicles and motor vehicle fuels. The regulations shall contain standards for such fuels or vehicles, or both, which EPA determines reflect the greatest degree of emissions reduction achievable through the application of technology which will be available, taking into consideration the standards established under section 202(a), the availability and costs of the technology, and noise, energy, and safety factors, and lead time. Such regulations shall not be inconsistent with the standards under section 202(a). The regulations shall, at a minimum, apply to emissions of benzene and formaldehyde.

This study is issued pursuant to Section 202(l)(1). A Federal Register notice announcing availability of the public review draft of this study was published on January 13, 1993 (FR 58(8):4165). The deadline for comments on the public review draft was March 1, 1993.

1.3 Scope of Study

The purpose of this study is to focus on air toxics emissions from motor vehicles and their fuels. Specific pollutants or pollutant categories which will be discussed include benzene, formaldehyde, 1,3-butadiene, acetaldehyde, diesel particulate, gasoline particulate, gasoline vapors as well as selected metals and motor vehicle-related pollutants identified in Section 112(b) of the Clean Air Act as amended in 1990. The focus of the study is on carcinogenic risk. The study also discusses non-cancer effects for these and other pollutants.

The discussion of non-carcinogenic effects is less quantitative due to the lack of sufficient health data.

Two general, but important, overall guidance documents, the Habicht memo on risk characterization (EPA, 1992a) and the new exposure guidelines (EPA, 1992b) were used in this study.

Cancer incidence estimates for formaldehyde, 1,3-butadiene, acetaldehyde, and gasoline particulate matter, and cancer death estimates for benzene and diesel particulate matter are provided for the following calendar years: 1990, 1995, 2000, and 2010. The following scenarios are examined:

1) a base control scenario, which takes into account implementation of the motor vehicle-related Clean Air Act requirements,

2) a scenario involving expanded use of reformulated gasoline,

3) a scenario involving expanded adoption of California standards.

The scenarios are described in more detail in Chapter 2.

With respect to benzene, formaldehyde, 1,3-butadiene, acetaldehyde, diesel particulate, gasoline particulate, and gasoline vapors, the study discusses the chemical and physical properties of the pollutant, formation and control technology, emissions (including other emission sources), atmospheric reactivity and residence times, exposure estimation, EPA's carcinogenicity assessment, other views of carcinogenicity assessment, recent and ongoing research, carcinogenic risk, and non-carcinogenic effects from inhalation exposure. The study also describes the qualitative change in toxic pollutant levels with the use of alternative clean fuels, along with a summary of toxic emissions from nonroad mobile sources. Finally, the study discusses the costs of various existing regulatory control programs and provides a qualitative discussion of the toxics benefits of these programs.

The study attempts to summarize what is known and all significant scientific opinion on each issue. It will serve as a background and status report, to be updated during the subsequent regulatory decision making process. This study does not include a decision on whether and what standards to promulgate.

1.4 Participation by Other EPA Offices and the Public

An informal EPA work group was formed to provide review and comment on plans, inputs, and drafts of the study. The following EPA offices were represented on the work group:

Office of Air and Radiation
Office of Air Quality Planning and Standards
Office of Mobile Sources
Office of Policy Planning and Evaluation
Office of Research and Development
Office of General Counsel
Office of Pesticides and Toxic Substances

A complete list of the work group members is included in Appendix A. Comments made by work group members on both the public review draft and a previous draft of this study have been incorporated.

Also, a briefing was conducted on March 25, 1991 with representatives from the automobile and oil industries to describe plans and obtain input on the direction of the study. A similar briefing was also held on August 8, 1991 with the Environmental Risk Assessment Committee of the Motor Vehicle Manufacturers Association (MVMA). In addition, on April 18, 1991, letters providing the status of the study and an offer to hold a briefing on our plans for this study were sent to various other organizations thought to have an interest in the study. These organizations include the following:

Oxygenated Fuels Association
Environmental Defense Fund
Health Effects Institute
STAPPA/ALAPCO
NESCAUM
Natural Resources Defense Council
California Air Resources Board
Information Resources, Inc.
Citizen Action

No specific requests were received for briefings or additional information; however, the California Air Resources Board provided extensive 1,3-butadiene emission data which are used in this study.

This study incorporates material and information from four reports, three resulting from work assignments initiated specifically to provide input for this study. One summarizes the available information on the health effects of benzene, 1,3-butadiene, formaldehyde, the motor vehicle toxics in Title III of the Clean Air Act Amendments, and several metallic compounds (Clement, 1991). The second report summarizes current understanding of the atmospheric behavior of benzene, 1,3-butadiene, and formaldehyde from an air quality standpoint,

including atmospheric formation and destruction reactions, major physical and chemical atmospheric removal processes, and simulated concentrations of these toxics in an urban area (Ligocki et al., 1991). These first two reports were sent in October, 1991 to the American Petroleum Institute, Ford Motor Company, the Engine Manufacturers Association, General Motors Research Laboratory, the MVMA Environmental Risk Assessment Committee, and the other organizations listed above, requesting comments. Comments were received from the American Petroleum Institute. API's comments on the contractor reports are reflected in this study. A third report summarized current understanding of the atmospheric behavior of acetaldehyde and polycyclic organic matter, and was prepared for EPA's Office of Policy Planning and Evaluation (Ligocki and Whitten, 1991). The fourth report presents a modification of the Hazardous Air Pollution Model (HAPEM) for mobile sources, referred to as HAPEM-MS, used to predict annual average exposures to toxic air pollutants dispersing from mobile sources (Johnson, et al., 1992).

On March 25, 1992, EPA mailed copies of large documents on the following three subjects to about 100 people on a public distribution list (including the organizations mentioned previously) requesting comments:

Toxic emission factors and control scenarios

Exhaust hydrocarbon emission benefits with oxygenated fuels

The HAPEM-MS model

Comments on the toxic emission factors were received from the California Air Resources Board and these comments were incorporated in this draft of the report. Also, a briefing on this material was given to the Coordinating Research Council Auto/Oil air toxics project group on April 30, 1992. The major comments received dealt with the uncertainties and inadequacies of the EPA carcinogenic potencies. Moreover, API presented an analysis of the HAPEM-MS model at the June 10-11, 1992 Workshop on Research Status on Emissions, Models, and Exposure Assessment at Research Triangle Park, North Carolina. API's major criticisms dealt with uncertainties in CO measurement, its apportionment to sources, and the validity of assuming constant pollutant/CO ratios. Comments were also recently received from the American Automobile Manufacturers Association and the Engine Manufacturers Association on the above mentioned documents. These comments are contained in four separate contractor reports, Environ (1992a,b), Ligocki (1992), and Whitten (1992). EPA responded to these comments and incorporated many into the final study.

In addition, EPA opened a Public Docket (Air Docket A-91-19) titled, "Availability of Information on the Mobile Source-Related Air Toxics Study Required by Section 206 of Title II of the 1990 Clean Air Act Amendments" to include information related to this study with the emphasis on material received from the public.

After release of the public review draft, the American Automobile Manufacturers Association requested a meeting with EPA to discuss comments on the study. This meeting was held in Detroit, Michigan on February 10, 1993. Representatives from Ford Motor Company, General Motors Corporation, the Engine Manufacturers Association, the Association of International Automobile Manufacturers, Chrysler Corporation, the Health Effects Institute, Environ Corporation, and Caterpillar Corporation also attended.

Public Comments received on the public review draft were reviewed and incorporated as appropriate in the final version. A complete list of commentors and a summary of the comments are included in Appendix I.

1.5 References for Chapter 1

Adler, J.M. and P.M. Carey. 1989. Air Toxics Emissions and Health Risks from Mobile Sources. Ann Arbor, Michigan: Environmental Protection Agency, Office of Mobile Sources. Prepared for the Air and Waste Management Association. AWMA Paper 89-34A.6.

Carey, P.M. 1987. Air Toxics from Motor Vehicles. Ann Arbor, Michigan: Environmental Protection Agency, Office of Mobile Sources. Publication no. EPA-AA-TSS-PA-86-5.

Carey, P.M. and J.H. Somers. 1988. Air Toxics Emissions from Motor Vehicles. Ann Arbor, Michigan: Environmental Protection Agency, Office of Mobile Sources. Prepared for the Air and Waste Management Association. AWMA Paper 88-128.1.

Clement International Corporation. 1991. Motor Vehicle Air Toxics Health Information. Prepared for Environmental Protection Agency, Office of Mobile Sources. September, 1991.

E.H. Pechan & Associates, Inc. 1990. Analysis of Air Toxics Emissions, Exposures, Cancer Risks and Controllability in Five Urban Areas, Volume II. Prepared for Environmental Protection Agency, Office of Air Quality Planning and Standards. April, 1990.

Environ. 1992a. Evaluation of Procedures Used by EPA to Assess Potential Health Risks of Vehicle Exhaust Particulates. May, 1992.

Environ. 1992b. Review of U.S. EPA's Application of the HAPEM Exposure Model to Mobile Source Pollutants. July 15, 1992.

Environmental Protection Agency. 1989. Analysis of Air Toxics Emissions, Exposures, Cancer Risks and Controllability in Five Urban Areas, Volume I, Base Year Analysis and Results. Research Triangle Park, North Carolina: Office of Air Quality Planning and Standards. Publication no. EPA-450/2-89-012a. July, 1989.

Environmental Protection Agency. 1990. Cancer Risk from Outdoor Exposure to Air Toxics. Research Triangle Park, North Carolina: Office of Air Quality Planning and Standards. Publication no. EPA-450/1-90-004a.

Environmental Protection Agency. 1992a. Guidance on Risk Characterization for Risk Managers and Risk Assessors. Memo from F. Henry Habicht II to the Assistant and Regional Administrators. February 26, 1992.

Environmental Protection Agency. 1992b. EPA Guidelines for Exposure Assessment; Notice. FR Vol. 57, No. 104. May 29, 1992. pp. 22888-22938.

Haemisegger, E., A. Jones, B. Steigerwald, and V. Thomson. 1985. The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants. Environmental Protection Agency, Office of Air and Radiation, and Office of Policy, Planning and Evaluation. May, 1985.

Johnson, T.R., R.A. Paul, and J.E. Capel. 1992. Draft Report: Application of the Hazardous Air Pollutant Exposure Model (HAPEM) to Mobile Source Pollutants. Durham, North Carolina. International Technology Corporation.

Ligocki, M.P. 1992. Review of EPA Memorandum on Calculation of Toxic Emission Mass Fractions. Systems Applications International, June 23, 1992. SYSAPP-92/075.

Ligocki, M.P., G.Z. Whitten, R.R. Schulhof, M.C. Causley, and G.M. Smylie. 1991. Atmospheric Transformation of Air Toxics: Benzene, 1,3-Butadiene, and Formaldehyde. Systems Applications International, San Rafael, California (SYSAPP-91/106).

Ligocki, M.P., and G.Z. Whitten. 1991. Atmospheric Transformation of Air Toxics: Acetaldehyde and Polycyclic Organic Matter. Systems Applications International, San Rafael, California (SYSAPP-91/113).

Whitten, G.Z. 1992. Review of EPA Memorandum "Interim Analysis of HC Reduction With the Use of Oxygenated Fuel Blends With 3-Way Catalyst Vehicles". Systems Applications International, San Rafael, California (SYSAPP-92/082).

2.0 SCENARIOS STUDIED

As mentioned in Chapter 1, cancer incidence resulting from exposure to benzene, formaldehyde, 1,3-butadiene, acetaldehyde, diesel particulate and gasoline particulate was estimated for several possible control scenarios in the years 1990, 1995, 2000, and 2010. The scenarios examined in this study include a base control scenario, which takes into account implementation of requirements in the CAAA of 1990, a scenario involving expanded use of reformulated gasoline, and a scenario involving expanded adoption of California motor vehicle standards. These scenarios were chosen to compare the possible effects different control programs could have, and do not necessarily represent EPA's expectations for the scope of possible expanded implementation for these control programs. In addition, the scenarios are not intended to indicate effects in specific areas where the strategies are implemented, such as the Northeast. Area specific analyses would be valuable, but are beyond the scope of this study. Although diesel particulate emissions were examined for 1990, 1995, 2000, and 2010, individual scenarios were not studied for this toxic, since expanded use of reformulated gasoline and the expanded adoption of California standards would not affect diesel particulate.

The use of alternative clean fuels, such as 85-100% methanol, 85-100% ethanol, and compressed natural gas, was not considered as part of any of these scenarios, since it is likely to comprise only a small fraction of total nationwide fuel use under current legislation (primarily as part of California's low emission vehicle program and the federal centrally fueled clean fuel fleet program). However, the use of alternative fuels could yield substantial toxics benefits, and their potential role in reducing motor vehicle-related air toxics will be discussed in Chapter 13.

2.1 Baseline

Base control scenarios for the years examined take into account implementation of the motor vehicle-related CAAA requirements, but assume no expanded adoption of CAAA programs or California standards, and no expanded use of gasohol beyond 1990 levels.

The 1990 base control scenario includes no new CAAA programs, since none were in place at this time. The 1995 base control scenario, however, includes Phase 1 of the federal reformulated gasoline program (coverage limited to the nine major metropolitan areas mandated by Section 211 (k) of the Act), Phase 1 of the California reformulated gasoline program, and the oxygenated fuels CO program. These programs are described in greater detail in Section 3.1.3. The 2000 and 2010 base control scenarios vary from the 1995 base control scenario in that Phase

2 federal and California reformulated gasoline will be in use, rather than Phase 1. Federal and California Phase 2 reformulated gasolines differ from Phase 1 fuels primarily in that they have lower RVP standards.

2.2 Additional Control Scenarios

2.2.1 Expanded Use of Reformulated Gasoline

This scenario is considered for the years 1995, 2000, and 2010. In this scenario, all ozone nonattainment areas opt into the federal reformulated gasoline program. In Section 211(k) of the Act, ozone nonattainment areas are given the option of participating in the program. In addition, all Northeast states have expressed intent to opt into the federal reformulated gasoline program; thus, they will be considered participants in this program under the expanded use of reformulated gasoline scenario. In 1995, Phase 1 federal and California reformulated gasoline will be in use, while in 2000 and 2010, Phase 2 federal and California reformulated gasoline will be in use.

2.2.2 Expanded Adoption of California Motor Vehicle Standards

This scenario is considered for the years 2000 and 2010. California emission standards are similar to federal standards in 1995; thus, this scenario is not considered for that year. However, California standards become increasingly more stringent with time, so that in 2000 and 2010, they are markedly lower than federal standards.

In this scenario, all Northeast states and states with ozone nonattainment areas categorized as extreme, severe, or serious adopt California motor vehicle emission standards. This scenario also assumes expanded use of reformulated gasoline, as described in the previous section.

California's new emission standards also involve the use of reactivity adjustment factors which normalize the mass of non-methane organic gas (NMOG) emissions from various fuels (such as reformulated gasoline, methanol, ethanol, and compressed natural gas) according to their ozone-forming potential. Furthermore, California certifies vehicles in several different categories according to their ozone-forming potential, and any combination of vehicles and fuels in these categories can be used to meet standards. For the sake of simplicity, it was assumed that the standards would be met using gasoline. More information on the California standards can be found in Section 3.1.3.1.

3.0 EMISSION FACTOR METHODOLOGY

3.1 Methodology for Benzene, Formaldehyde, 1,3-Butadiene, and Acetaldehyde

3.1.1 Approach

In order to obtain risk estimates, emission factors must be calculated. With the approach used for this report, available vehicle emissions data are used to estimate toxic emissions as fractions of total organic gases (TOG). TOG includes all hydrocarbons as well as aldehydes, alcohols, and other oxygenated compounds. These fractions are then applied to an updated version of MOBILE4.1, designated MOBTOX, developed specifically to calculate toxic grams per mile emission factors. This same basic approach was used in previous EPA papers (Carey, 1987; Carey and Somers, 1988; Adler and Carey, 1989), where emission fractions for air toxics were applied to MOBILE4 THC output.

MOBTOX calculates in-use g/mile toxic emission factors. This approach was used because virtually all the available emission data are from low mileage, well-maintained vehicles. To simply use the g/mile data from these studies directly would likely result in an underestimation of true emissions.

The approach outlined in this section will be used for benzene, 1,3-butadiene, formaldehyde, and acetaldehyde. In order to estimate these emission factors, mass fractions of exhaust TOG emissions and evaporative emissions (for benzene) must be obtained for these toxics from actual data, to input into MOBTOX. These fractions must be calculated for various motor vehicle classes, catalyst types, fuel systems, and fuel blends. Separate sets of fractions resulting from implementation of different regulations must also be calculated. Section 3.1 describes the methodology for obtaining mass fractions for the non-particulate air toxics and for developing MOBTOX inputs. It should be noted that all mass fractions are expressed as fractions of TOG.

3.1.2 Assumptions

A number of important assumptions were made in the approach outlined in this section. Several of these assumptions were:

1) Increase in air toxics due to vehicle deterioration with increased mileage is proportional to increase in TOG.
2) Toxics fractions remain constant with ambient temperature changes.
3) The fractions are adequate to use for the excess hydrocarbons that come from malfunction and tampering/misfueling.

These assumptions can be addressed by looking at high mileage data, temperature data, malfunction data, and misfueling data. First, Carey (1987) analyzed formaldehyde and benzene data from the 46 car study (Sigsby et al., 1987), and found very little difference in fractions of these compounds among vehicles with high and low hydrocarbon emissions. Also, an earlier study (Smith and Carey, 1982) shows high mileage cars control formaldehyde roughly to the same extent as total hydrocarbons. Similarly, a General Motors study (Dasch and Williams, 1991) showed no significant increase in benzene fractions with mileage. Furthermore, the emission fractions calculated from low-mileage vehicles in the current analysis are similar to the in-use fractions in the General Motors study. Thus, it is reasonable to assume that these two compounds increase proportionally to TOG. Finally, results from a recent Auto/Oil analysis (Auto/Oil, 1993) indicated that fuel effects on toxic emissions were similar in normal and high emitting vehicles. Furthermore , the toxic fractions were similar for normal and high emitting vehicles. This analysis included the toxics formaldehyde, acetaldehyde, benzene, and 1,3-butadiene.

Stump et al. (1989, 1990, unpublished) looked at the effects of ambient temperature on exhaust toxics. Stump et al. (1989, 1990), in their low temperature study (20°F to 70°F range), found a slight increase with temperature reduction of formaldehyde emissions, but overall, the composition of hydrocarbon emissions did not vary appreciably with temperature. In the high temperature study (Stump et al., unpublished, 75°F to 105°F range), exhaust and evaporative emissions were analyzed. Formaldehyde exhaust emissions increased slightly in PFI vehicles with increased temperature, but decreased slightly for the one carbureted vehicle studied. There was no appreciable change for other aldehydes. Moreover, the authors state that tailpipe emissions for benzene and 1,3-butadiene in general followed total hydrocarbon levels. For diurnal evaporative emissions, aromatics fractions as a whole were measured. It is expected that benzene fractions would track the aromatics trend. Aromatics fractions went down with temperature, for both the fuel injected vehicles and the carbureted vehicle. Hot soak fractions of aromatics in fuel injected vehicles went up when going from 75 to 90°F, but down when going from 90 to 105°F. For the carbureted vehicle, however, aromatics went down when going from 75 to 90°F, but up when going from 90 to 105°F. Also, a separate analysis has been performed (EPA, 1992a) in which a number of MOBILE4.1 runs were done at four different temperatures in 1990 and 2000. The results indicated that the ratio of hydrocarbon and carbon monoxide from one temperature to another is relatively constant in 1990 and 2000. Based on the results of these studies, a broad generalization was made that emission fractions would not change as a function of temperature.

Carey (1987) also analyzed available malfunction and misfueling exhaust data for aldehydes and benzene. For aldehydes, Carey reviewed misfueling data available for a single vehicle (Nebel, 1981) and found only a slight increase in

percentages of aliphatic aldehydes, which should be an indicator of formaldehyde and acetaldehyde emissions. In addition, an analysis of malfunction studies (Urban, 1980a, 1980b, 1980c, 1981; Urban and Garbe, 1979, 1980) indicated roughly similar formaldehyde percentages with and without several malfunctions for vehicles with no catalyst, but small decreases in formaldehyde percentages with malfunctions in catalyst equipped vehicles. From this review, Carey (1987) concluded that, overall, formaldehyde percentages were relatively stable under malfunction and misfueling conditions.

For benzene, Carey (1987) analyzed data from the same malfunction studies analyzed for formaldehyde. A 12 percent misfire mode decreased benzene exhaust percentages appreciably, while a rich best idle mode increased benzene exhaust percentages appreciably. Since these two malfunctions were offsetting, and other malfunctions had lesser effects, no adjustments were made to benzene fractions for malfunctioning. No misfueling studies were available for benzene; thus, we assumed no misfueling effects on benzene fractions.

No malfunction or misfueling data were available for 1,3-butadiene; however, the CARB data used to determine 1,3-butadiene fractions were based on in-use vehicles, tested as received, with the same fuel as received. Thus, there was no need to address the effects of malfunction or misfueling on emission fractions for 1,3-butadiene.

3.1.3 Emission Factor Requirements

3.1.3.1 Scenario Components

Before developing exhaust and evaporative mass fractions to use in determining emission factors, it is necessary to consider the various scenarios to be included in the report. The scenarios, which are described in Chapter 2, include:

1) a base control scenario, which takes into account implementation of the motor vehicle-related Clean Air Act requirements,
2) a scenario involving expanded use of reformulated gasoline, and
3) a scenario involving expanded adoption of California standards.

The effects of the different scenarios on overall emissions will be considered for the following years: 1990, 1995, 2000, and 2010. It will not be possible to simply run MOBTOX once for each scenario/calendar year. This is because various areas of the country have different fuel and/or emission standard requirements, as well as different I/M programs. From an examination of the Clean Air Act requirements, the scenarios to be considered, and the California program, nine different fuel/emission standard combinations were identified. These

fuel/emission standard combinations will be referred to as components.

This section focuses on the fuel specifications, emission standards, and calendar years applicable for the nine components. A list of components and the fuel specifications assumed for these components is given in Table 3-1. For federal reformulated gasoline, fuel parameters are not certain at this time, particularly for Phase 2 gasoline. However, the fuel parameters used in this report for Phase 1 and 2 meet the toxic performance requirements required in the Clean Air Act. Section 3.1.3.2 provides more information on the scenarios, including which components are considered for each scenario, their relative weighting by fuel use, and the specific areas/cities covered under each scenario component.

1) Baseline Gasoline Use (Federal Emission Standards) -- Covers areas of the country using a typical 1990+ baseline gasoline. Baseline gasoline for 1990 and subsequent years was assumed to contain 1.53% benzene, 32% aromatics, and 0% oxygen, at 8.7 psi Reid Vapor Pressure (RVP). These levels are given as summertime baseline gasoline specifications for the reformulated gasoline program in Section 211(k) of the CAA. According to the national fuel survey (MVMA, 1990), regular unleaded gasoline in summer 1990 contained 1.46% benzene, 27.8% aromatics, and 0% oxygen, at 8.6 psi RVP. These specifications are similar to those given in Section 211(k).

   The federal THC/NMHC 50,000 mile emission certification standards for light duty vehicles ($\leq$ 3750 lbs.) are of interest for this analysis. The THC standard is currently 0.41 gram per mile. The Tier 1 tailpipe standard of 0.25 gram per mile for NMHC will be phased in beginning in 1994. A Tier 2 tailpipe NMHC standard of 0.125 gram per mile beginning in 2004 is contingent on determination of cost-effectiveness and feasibility by EPA. For this analysis, it is assumed that Tier 2 will not be implemented.

   For the sake of simplification, California is included in this component for 1990 since the current California exhaust NMOG 50,000 mile certification standard of .390 grams per mile NMOG is similar to the current federal THC standard of 0.41 grams per mile. This component is considered for all the calendar years of interest.

2) Baseline Fuel Use (California Emission Standards) -- Under an expanded scenario, all states with extreme, severe or serious ozone nonattainment areas adopt California emission standards. Also under this expanded scenario, all Northeast states adopt California standards. This scenario may result in attainment

Table 3-1. Fuel Specifications for the Various Components.

Components	Fuel Specifications			
	Benzene (% Vol.)	Aromatics (% Vol.)	Oxygen (% Wt.)	RVP (psi)
Baseline Gasoline Use Federal Standards	1.53	32	0	8.7
Baseline Gasoline Use California Standards	1.53	32	0	8.7
Federal/Calif. Reform. Gasoline Use Federal Phase 1 (1995-1999) Calif. Phase 1 (1992-1995) Federal/Calif. Standards	1.0	25	2.0	8.1
Federal Reform. Gasoline Use Phase 2 (2000+) Federal Standards	1.0	25	2.0	7.8
Federal Reform. Gasoline Use Phase 2 Calif. Standards	1.0	25	2.0	7.8
Winter Oxygenated Gasoline Use Federal/Calif. Standards (1995) Federal Standards (2000, 2010)	1.05	22	2.7	8.7
Winter Oxygenated Gasoline Use Calif. Standards (2000, 2010)	1.05	22	2.7	8.7
California Only Calif. Reform. Gasoline Use Phase 2 (1996+) Calif. Standards	1.0	25	2.0	7.0
Gasohol Fuel Use Federal Standards	1.4	28.8	3.5	9.7

areas in those states having baseline fuel use in conjunction with California emission standards. In 1995, this has little effect on emission factors, since federal and California light duty vehicle ($\leq$ 3750 lbs.) exhaust emission standards are similar (0.250 g/mile NMHC under federal regulations; 0.231 g/mile NMOG under California regulations). Thus there is no need to distinguish between the two sets of standards. In 2000 and 2010, however, federal and California standards are markedly different, with the federal standard remaining at 0.250 g/mile NMHC (under the assumption that Tier 2 is not implemented), while the California standard is 0.073 g/mile NMOG for 2000 and 0.062 g/mi NMOG for 2010. For these years, then, fuel use for attainment areas using baseline fuel with California

standards must be treated separately from baseline fuel use with federal emission standards.

3) Federal Reformulated Gasoline Program, Phase 1 (Federal Emission Standards) and California Reformulated Gasoline Program, Phase 1 (California Emission Standards) -- This component covers regions participating in Phase 1 of the federal reformulated gasoline program, from 1995 through 1999, under federal emission standards. It also covers Northeast states participating in the federal reformulated gasoline program and also opting into the program for California emission standards in 1995, as well as California under the California Phase 1 reformulated gasoline program (1992 - 1995) with California emission standards. Due to the timing of the Phase 1 requirements, this component is considered only for the calendar year 1995.

Phase 1 federal reformulated gasoline must contain at least 2.0% oxygen, and must not result in a NO_x increase. Reduction of both ozone forming VOCs and air toxics must be least 15%, relative to emission levels from 1990 model year vehicles with a baseline gasoline. The required 15% minimum toxics reduction for reformulated gasoline is measured on a mass basis for 5 specific pollutants -- benzene, formaldehyde, acetaldehyde, 1,3-butadiene, and POM. The toxics requirement is year-round while the VOC requirement applies during the summer months. Reformulated gasoline fuel specifications of 2.0% oxygen, 1.0% benzene, 25% aromatics and 8.1 psi RVP (for ASTM Class C areas) were assumed for CY 1995 - 1999. The oxygen and benzene specifications are minimum or maximum requirements specified in Section 211(k) of the Act. The RVP level is an estimate for Class C areas.

Based on EPA's proposed regulations for reformulated gasoline, EPA assumed maximum RVP levels for the high ozone season (June 1 through September 15) of no more than 7.2 psi in Class B areas (in Southern states) and 8.1 psi in Class C areas (in Northern states). However, a recent EPA proposal seeks comment on a decision by former President Bush to effectively grant gasohol a 1 psi RVP waiver for up to 30% of the total reformulated gasoline market in the Northern cities. The increase in VOC emissions from the higher RVP would be compensated for through a requirement that the volatility of reformulated gasoline blendstock in these cities be reduced by 0.3 psi to 7.8 psi. A similar provision would be made available for Southern cities to opt into, except that gasohol would effectively receive a 1 psi waiver for up to 20% of the total reformulated gasoline market, requiring the use of 7.0 psi RVP blendstock gasoline. Details of this waiver are presented in a recent proposed rule for standards for reformulated gasoline (EPA, 1993).

California Phase 1 reformulated gasoline in CY 1992 - 1995 has similar specifications. Thus for 1995, there is no need to distinguish between federal and California Phase 1 reformulated gasoline.

There are a number of areas where California has more stringent standards or special programs not implemented in the rest of the country. For instance, the CAAA establish provisions for a California clean car pilot program, applying to a limited number of cars starting in 1996. The pilot program is not considered in this report. Also under the CAAA, California is permitted to develop its own, more stringent vehicle control program.

The California Air Resources Board (CARB) has adopted regulations establishing increasingly stringent vehicle certification standards beginning in 1994 (CARB, 1990). Requirements for non-methane organic gas (NMOG, which is TOG less methane) begin at 0.250 grams per mile for light duty vehicle ($\leq$ 3750 lbs.) exhaust at 50,000 miles in 1994 and are progressively reduced to 0.062 grams per mile in 2003 (with a requirement of 0.231 grams per mile in 1995). CARB's new standards involve the use of reactivity adjustment factors which normalize the mass of NMOG emissions from various fuels according to their ozone-forming potential. CARB certifies vehicles in several categories based on the ozone-forming potential of their emissions. These categories are: Transitional-Low Emission Vehicles (TLEVs), Low Emission Vehicles (LEVs), Ultra-Low Emission Vehicles (ULEVs), and Zero Emission Vehicles (ZEVs). Under the 1994 standards, any combination of TLEVs, LEVs, ULEVs, ZEVs and 1993 conventional vehicles can be used to meet fleet average requirements. The 50,000 mile exhaust emission certification standards for the light duty vehicle ($\leq$ 3750 lbs.) categories are described in Table 3-2.

Although over time California emission standards are more stringent than federal standards, they are similar for 1995. Since California and federal Phase 1 reformulated gasoline specifications are also similar in 1995, all areas with combinations of federal and California Phase 1 reformulated gasoline and federal and California emission standards can be considered as one component. This includes many Northeast states which are considering participating in the federal reformulated gasoline program and also opting into the program for California standards. In these states, vehicles will be certified on California gasoline and will

Table 3-2. California Low Emission Vehicle 50,000 Mile Exhaust Emission Certification Standards for Light Duty Vehicles (≤ 3750 lbs.).

Vehicle Category[3]	Grams/Mile by Pollutant			
	NMOG[1]	NO_x	CO	HCHO
Current	0.390	0.4	7.0	none
1993	0.250	0.4	3.4	0.015[2]
TLEV	0.125	0.4	3.4	0.015
LEV	0.075	0.2	3.4	0.015
ULEV	0.040	0.2	1.7	0.008
ZEV	0.000	0.0	0.0	0.000

[1]NMHC for current and 1993 standards, NMOG with reactivity adjustment for others.
[2]Methanol-fueled vehicles only.
[3]Emission levels in this table do not include stationary source emissions related to fuel generation, including generation of electricity for ZEVs.

have to meet California standards, but for purposes of this study are presumed to be running on federal reformulated gasoline in-use.

4) Federal Reformulated Gasoline Program, Phase 2 (Federal Emission Standards) -- This component covers regions participating in Phase 2 of the federal reformulated gasoline program, under federal emission standards.

Beginning in the year 2000, under Phase 2 of the reformulated gasoline program, ozone forming VOC and toxics reductions must be at least 25%, or 20% if the 25% reduction is judged to be unfeasible. Once again, the toxics requirement is year-round while the VOC requirement applies during the summer months.

It is assumed that Phase 2 federal reformulated gasoline will have similar benzene and oxygen requirements as Phase 1 gasoline. For purposes of this study an RVP of 7.8 psi is assumed (for ASTM Class C areas), slightly lower than the 8.1 psi assumed for Phase 1. The component is considered for calendar years 2000 and 2010.

5) Federal Reformulated Gasoline Program, Phase 2 (California Emission Standards) -- This component covers non-California regions participating in Phase 2 of the federal reformulated gasoline program, under California motor vehicle emission standards.

Regions participating in Phase 2 of the federal reformulated gasoline program under California standards cannot be considered with Phase 2 of the California program, because California Phase 2 gasoline has a much lower RVP requirement. This component is only applicable for the scenario involving expanded adoption of California standards for calendar years 2000 and 2010.

6) Oxygenated Fuels CO Program, (Federal and California Emission Standards, 1995; Federal Emission Standards, 2000, 2010) -- This component covers regions participating in the seasonal oxygenated gasoline CO program, beginning November 1, 1992, while complying with federal or California motor vehicle emission standards in 1995 scenarios. It also covers regions complying with federal emission standards in scenarios for the years 2000 and 2010. Regions with California and federal standards are considered as one component in 1995 because of the similar federal and California emission standards during this year.

Section 211(m) of the Act specifies a minimum 2.7% oxygen level for gasoline in this program. Winter oxygenated gasoline, used in the oxygenated fuels CO program, was assumed to be 2.7% oxygen (15% MTBE), 22% aromatics, 1.05% benzene and 8.7 psi RVP. The estimate of 22% aromatics was chosen after examining fuel specifications of 15% MTBE blends used in various test programs. Aromatic levels in the 22% range were fairly consistent across these studies. The percent reduction in aromatics from the baseline level of 32% to 22% was then applied to the baseline benzene level of 1.53% to obtain the estimate of 1.05% benzene. 8.7 psi RVP was chosen arbitrarily. It is likely winter fuel would have a higher RVP, but changing RVP would have a minor effect on the exhaust fractions calculated.

Some regions participating in this program will also be participating in the federal reformulated gasoline program or the California reformulated gasoline program. In regions participating in two programs, fuel requirements for both the winter oxygenated and Phase 1 or Phase 2 federal or California reformulated gasoline (depending on the year) will have to be met during winter. The primary differences between these fuels which may affect toxics emission fractions are RVP and oxygen content. Since RVP is not a significant factor during winter months, and VOC control for reformulated gasoline is limited to the summer months, it was assumed for modeling purposes that winter oxygenated gasoline would be used in all of these regions during the winter months. In the modeling, use of winter oxygenated

gasoline still meets the toxics reduction requirements of the federal reformulated gasoline program. This component is considered for calendar years 1995, 2000, and 2010.

It should be noted that we assumed the oxygenated gasoline CO program would utilize a 2.7% oxygenate MTBE blend. Other oxygenated blends with ethanol (at the 2.7 oxygen level) will also be used. However, similar toxics benefits are expected with the use of gasohol in reformulated areas.

7) Oxygenated Gasoline CO Program (California Emission Standards, 2000, 2010) -- This component covers regions participating in the oxygenated gasoline CO program, while complying with California emission standards for the years 2000 and 2010. These regions will be found in California and, under an expanded scenario, in states with extreme, severe, and serious ozone nonattainment areas adopting California standards, and also Northeast states adopting California standards. Once again, some regions may also be participating in the federal reformulated gasoline program. These regions will have to meet fuel requirements for both the winter oxygenated and Phase 2 federal reformulated gasoline during the winter. It is assumed that regions in California participating in this program will have to meet requirements for winter oxygenated and California Phase 2 gasoline. As with the previous component, it was assumed for modeling purposes that winter oxygenated gasoline would be used in winter for all regions considered as part of this component. This component is considered for calendar years 2000 and 2010.

8) California Reformulated Gasoline, Phase 2 (California Emission Standards) -- This component covers California under Phase 2 California reformulated gasoline requirements (1996+), under California emission standards. Phase 2 California reformulated gasoline includes maximum limits of 1.0% benzene, 25% aromatics, 2.0% oxygen and 7.0 psi for each gallon refined (Refiners can choose instead to average production over 90 days, meeting lower averaged limits for benzene and aromatics of 22 and 0.80 percent, respectively). This component is considered for calendar years 2000 and 2010.

9) Ethanol Fuel Use (Federal Emission Standards) -- This component is based on vehicle consumption of 10% ethanol in gasoline (or gasohol). It is considered for all the calendar years.

The composition of gasohol is assumed to be 1.4% benzene, 28.8% aromatics, and 9.7 psi RVP. The composition was estimated by assuming a 10% reduction of benzene and aromatics, and an increase of 1 psi in RVP from dilution of gasoline with 10% denatured ethanol, applied to the baseline gasoline specifications. This composition is similar to the composition of the 10% ethanol blends used in the Auto/ Oil

study (1991) which had benzene levels ranging from 1.4 to 1.5%, aromatics ranging from 18 to 29%, and RVP ranging from 9.0 to 9.6 psi. The composition of the 10% ethanol blend used in another recent study used as a data source in this report (Warner-Selph and Smith, 1991) was also similar, with 1.35% benzene, 22.8% aromatics, and an RVP of 10.15 psi.

Although the CAA establishes provisions for a California pilot program and a clean fuel fleet program for centrally fueled fleets, we will not consider scenarios specifically involving components for these programs. As mentioned above, California is establishing its own standards which could effectively supplant the standards specified by the pilot program in California. Since the centrally fueled clean fuel fleet program covers a small number of vehicles (30% of new fleet purchases in 26 metropolitan areas, starting in 1998, for fleets with central refueling), it was deemed unnecessary to include a component for this program in this report. The toxics benefits associated with using 85-100% methanol, 85-100% ethanol, and compressed natural gas as alternative fuels (EPA, 1989a, 1990a, 1990b, and 1990c) are qualitatively discussed in Chapter 13.

3.1.3.2 Percent of Nationwide Fuel Use by Component for Each Scenario

Table 3-3 consists of a matrix allocating nationwide fuel use in 1990, 1995, 2000, and 2010 for the various components of each scenario. Descriptions of the three scenarios listed earlier for each calendar year are given below. These include descriptions of how fuel use percentages in a given year were determined for each component in a scenario. Assumptions made in determining these
fuel use percentages are also discussed. Also included are the specific areas/cities covered under each scenario component.

1) 1990 Base Control -- Since no new CAA programs were in effect in 1990, this scenario includes only two components -- one for baseline gasoline use, and one for gasohol fuel use. An estimate of 6% gasohol fuel use for 1990 was obtained from data compiled by the U.S. Department of Transportation (1991). These data were based on gross gallons of gasohol reported by wholesale distributors to State motor fuel tax agencies, and include highway use, nonhighway use, and losses. The remainder of fuel use in this scenario (94%) was assumed to be baseline gasoline use.

Table 3-3. Nationwide Fuel Use for the Various Components Under Different Scenarios.

Percent of Total Nationwide Fuel Use*

	1990	1995		2000, 2010		
Components/Scenarios	Base Control	Base Control	Expanded Reform. Gasoline Use	Base Control	Expanded Reform. Gasoline Use	Expanded Adoption Calif. Standards
Baseline Gasoline Use Federal Standards	94	59	27	59	27	22
Baseline Gasoline Use California Standards	0	0	0	0	0	4
Federal/Calif. Reform. Gasoline Use Federal Phase 1 (1995-1999) Calif. Phase 1 (1992-1995) Federal/Calif. Standards	0	18	50	0	0	0
Federal Reform. Gasoline Use Phase 2 (2000+) Federal Standards	0	0	0	10	42	13
Federal Reform. Gasoline Use Phase 2 Calif. Standards	0	0	0	0	0	30
Winter Oxygenated Gasoline Use Federal/Calif. Standards (1995) Federal Standards (2000, 2010)	0	17	17	12	12	3
Winter Oxygenated Gasoline Use Calif. Standards (2000, 2010)	0	0	0	5	5	14
California Only Calif. Reform. Gasoline Use Phase 2 (1996+) Calif. Standards	0	0	0	8	8	8
Gasohol Fuel Use Federal Standards	6	6	6	6	6	6

*Each vertical column totals 100 percent.

2) 1995 Base Control -- This scenario includes gasoline use under Phase 1 of the federal reformulated gasoline program and the California program, the oxygenated gasoline CO program, and gasohol fuel use. Fuel use under Phase 1 of the federal reformulated gasoline program and Phase 1 of the California reformulated gasoline program combined was estimated to be 18%. The base control scenario assumes only the 9 extreme/severe ozone nonattainment areas participate in the federal reformulated gasoline program. These areas are:

1) New York
2) Philadelphia
3) Hartford, Connecticut
4) Los Angeles
5) Baltimore
6) San Diego
7) Chicago
8) Milwaukee
9) Houston

Gasoline use data for these nine areas were obtained from the Standards Development and Support Division (RDSD), in EPA's Office of Mobile Sources, and were used by RDSD to calculate fuel consumption figures contained in the draft regulatory impact analysis for reformulated gasoline and anti-dumping regulations (EPA, 1991a). In 1990, these 9 areas were responsible for 22.2% of the annual fuel use in the United States. Fuel use percentages for the two extreme/severe ozone nonattainment areas located in California (6.7%) were subtracted from this 22.2% since California was assumed to have its own reformulated fuel program statewide. Fuel use for the extreme/severe ozone nonattainment areas outside California was adjusted to account for an estimated 15% "spillover" of reformulated gasoline into uncovered areas. This 15% estimate was obtained from RDSD's draft regulatory impact analysis cited above. Then, fuel use in California (12.0%) was added to the total. The fuel use estimate for California reformulated fuel under the California program was based on the reported gasoline consumption for California in 1990 (12.0% of fuel used), obtained from data compiled by the U.S. Department of Transportation (1990). This estimate was adjusted for the projected population increase in California between 1990 and 1995 (about 9%; Wetrogan, 1990). It was assumed the increase in fuel consumption would be proportional to the increase in population. Winter oxygenated gasoline use for areas participating in the federal and California reformulated gasoline programs (11.9%) was also subtracted from the total, thus giving the estimate of 18% of nationwide gasoline use for this component.

Winter oxygenated gasoline use was estimated using data provided by EPA's Field Operations and Support Division (FOSD) in the Office of Mobile Sources. FOSD provided percent gasoline use data for each of the 39 regions in the oxygenated fuels CO program. For the purposes of this report, it was assumed that these same 39 areas would have the winter oxygenate program in place for scenarios in 1995, 2000, and 2010. These fuel use percentages were for the entire year, so assuming that fuel use was constant through the entire year (which is admittedly an approximation since fuel usage is greater in the summer versus winter months), the percentages were multiplied by the fraction of the year each region was expected to be in the program. All regions were assumed to have four month programs, with the following exceptions: Las Vegas and Phoenix with 5 month programs, Los Angeles and Spokane with 6 month programs, and New York with a 12 month program. Winter oxygenated fuel use was estimated to be 17% for 1995. This fuel use estimate includes an adjustment to account for 15% spillover.

Gasohol fuel use was assumed to remain constant at six percent, relative to 1990, for this and all scenarios.

3) 1995 Expanded Use of Reformulated Gasoline -- In Section 211(k) of the Act, any ozone nonattainment area may opt into the federal reformulated gasoline program. In this scenario, all ozone nonattainment areas are considered to opt into the program. At the time this analysis was done, all Northeast states except Delaware and Vermont had opted into the federal reformulated gasoline program and were thus included. These states include the following:

1) Maine
2) New Hampshire
3) Massachusetts
4) Rhode Island
5) New York
6) New Jersey
7) Pennsylvania
8) Connecticut
9) Maryland
10) Virginia
11) Washington, D.C.

Delaware and Texas have since opted into the program. Northeast states and serious and above ozone nonattainment areas may also adopt California emission standards, but this will have no effect on the fuel use weightings for this scenario, because of the similarity between federal and California emission standards in 1995.

Gasoline use under Phase 1 of the federal reformulated gasoline program and Phase 1 of the California reformulated gasoline program combined was estimated to be 50% for this scenario. Phase 1 federal reformulated gasoline use in this scenario was based on data from SDSB's draft regulatory impact analysis for reformulated gasoline and anti-dumping regulations (EPA, 1991a). 1990 fuel use percentages for regions in California and the Northeast, calculated to be 29.6%, were subtracted from the total fuel use in all ozone nonattainment areas (53.8%). (To simplify the analysis, individual nonattainment areas in the Northeast were not considered and it was assumed the entire state received reformulated gasoline. While only those ozone nonattainment areas included in the state governor's opt-in request are technically included in the federal reformulated program, these typically covered the major metropolitan areas of the state. In combination with the fungible gasoline distribution system serving the Northeast, this should mean that reformulated gasoline will be distributed throughout the entire Northeast. Similarly, individual nonattainment areas in California were not considered since the entire state of California was assumed to have California reformulated gasoline). The resultant percentage (24.2%) was increased by 15% to account for spillover. Then projected statewide fuel use percentages for all opt in states and California were added (34.4%). These projected percentages were obtained by taking fuel consumption estimates from the U.S. Department of Transportation (1990). These estimates were adjusted for the projected population increases in these states between 1990 and 1995 using Department of Commerce data (Wetrogan, 1990). It was assumed increases in fuel consumption would be proportional to increases in population. Finally, winter oxygenate gasoline use in all regions and states participating in the federal reformulated gasoline program and California (12.4%) was subtracted from the total, resulting in a total fuel use estimate for this component of 50%. Fuel use for other components (except for a reduction in baseline fuel use to 27%) remained the same as in the base control scenario.

4) 2000, 2010 Base Control -- This scenario differs from the 1990 base control scenario in that Phase 2 federal and California reformulated gasoline, rather than Phase 1, will be in use. Phase 2 federal reformulated gasoline was assumed for purposes of this study to have an RVP of 7.8 psi, while Phase 2 California reformulated gasoline has an RVP of 7.0 psi. Moreover, as previously discussed, federal and California emission standards will be much different in these years. Thus, California reformulated fuel use in California (8%) and winter oxygenated fuel use in California (5%) were treated as separate components. Otherwise, it was assumed fuel use in different programs will remain the same (implying there will be no population shifts among regions). This assumption was made because of the difficulty in accurately projecting population changes in various regions within states.

5) 2000, 2010 Expanded Use of Reformulated Gasoline -- Once again, this scenario differs from the 1995 expanded reformulated gasoline use scenario, in that Phase 2 federal and California reformulated gasoline will be in use, rather than Phase 1, and federal and California emission standards will be markedly different in those years.

6) 2000, 2010 Expanded Adoption of California Standards -- Under this scenario, all Northeast states and states with ozone nonattainment areas categorized as extreme, severe, or serious adopt California emission standards. This scenario assumes expanded use of reformulated gasoline also.

Phase 2 federal reformulated gasoline use under California emission standards was estimated to be 30%. First, fuel use in all extreme, serious, and severe ozone nonattainment areas was estimated at 29.3%, based on data from SDSB's draft regulatory impact analysis for reformulated gasoline and anti-dumping regulations (EPA, 1991a). Fuel use in regions in California and Northeast states (20.7%) was subtracted from this total. Fuel use in all moderate and marginal ozone nonattainment areas in all other states with California standards was then added. The resultant 12.8% was adjusted for 15% spillover. Then, projected statewide fuel percentages for all Northeast states included in the expanded reformulated fuel use scenario were added (22.2%). Finally, winter oxygenated fuel use in all extreme, severe, and serious ozone nonattainment areas also classified as CO nonattainment areas (7.24%) was subtracted from the total, resulting in the total fuel use estimate for this component of 30%.

Phase 2 federal reformulated gasoline use under federal emission standards was estimated to be 13%. First, fuel use in all moderate and marginal ozone nonattainment areas was estimated at 24.4%, based on data from SDSB's regulatory impact analysis cited above. Then fuel use in moderate and marginal nonattainment areas with California emission standards (12.1%) was subtracted from this total, and the remaining 12.3% adjusted for 15% spillover. Finally, winter oxygenate fuel use in moderate and marginal ozone nonattainment areas with federal emission standards (0.8%) was subtracted, resulting in a 13% estimate for this component.

In this scenario, states with extreme, serious and above ozone nonattainment areas adopting California standards may have baseline fuel use under California standards outside the nonattainment areas. Fuel use for this component was estimated by subtracting fuel use in federal reformulated fuel areas with California standards (37.0%) from fuel use in all states with California standards, exclusive of California (40.8%). If there were any areas with winter oxygenated fuel use under California standards which were in ozone attainment, fuel use in these areas would also have to

be subtracted. However, no such areas exist. Thus total fuel use for this component is about 4%.

Winter oxygenated fuel use under California emission standards was estimated to be 14%, while winter oxygenated fuel use under federal emission standards was estimated to be 3%. The fuel use estimate for California reformulated fuel remained the same as under the expanded reformulated fuel use scenario for 2000 and 2010. The remainder of fuel use was assigned to baseline fuel use under federal emission standards.

3.1.3.3 Emission Fractions Associated with Components

After determining the nine components to be included for each calendar year scenario, emission fractions for the various fuels considered in these components were estimated. For baseline fuel use, emission fractions were calculated for gasoline and diesel fuel. As will be seen later, it was relatively easy to calculate the diesel numbers. For the components with federal and California reformulated fuel use, emission fractions were determined for 11% MTBE blends (2% oxygen). For the gasohol component, emission fractions for 10% ethanol were determined. For the oxygenated fuels CO program, emission fractions for 15% MTBE blends (2.7% oxygen) were determined. For the components with California emission standards, the same emission fractions for Phase 1 federal and California reformulated fuels were used, since the fuel characteristics are similar. One difference is in RVP, which is assumed to be 8.1 psi for Phase 1 federal and Phase 1 California reformulated fuel, but 7.0 psi beginning in 1996 for Phase 2 California fuel. This results in different benzene evaporative emission fractions for the two components. Also, Phase 1 federal reformulated gasoline is assumed to have a higher RVP than Phase 2 (8.1 versus 7.8) resulting in slightly different benzene evaporative emission fractions.

3.1.3.4 I/M Programs Associated with Components

The CAA requires that all ozone and carbon monoxide nonattainment areas must implement some kind of vehicle Inspection and Maintenance (I/M) program. Depending on the severity of the nonattainment problem, these areas will have to implement either a basic I/M program (required in areas with moderate ozone nonattainment, and in marginal areas with existing I/M programs) or an enhanced program (required in most serious, severe, and extreme ozone areas, as well as most carbon monoxide areas registering greater than 12.7 ppm and larger metropolitan statistical areas in the Northeast Ozone Transport Region) (EPA, 1992b). The enhanced I/M program used in modeling includes annual centralized testing of light duty vehicles and trucks, an IM240 test, antitampering tests and functional tests of the evaporative emission control system, including pressure and purge testing. The basic I/M program used in modeling was the ideal minimum I/M program recommended by the Agency.

The choice of I/M program to input into MOBTOX when modeling components affects the resultant toxics emission factors. In fact, components have a mixture of no, basic and enhanced programs in different areas. To account for this, separate MOBTOX runs for each type of I/M program were done for a component, and the resultant emission factors weighted according to the frequency of the I/M program within that component, to obtain an I/M weighted emission factor.

The I/M program weightings for each component were calculated by comparing a EPSD database compiled by EPA's Emission Planning and Strategies Division listing metropolitan statistical areas with their current and expected future I/M programs to the specific areas/cities covered under each scenario component described above, and estimating the percentage of individual scenario components covered by each type of I/M program. The breakdown of I/M programs expected in various areas has changed slightly since this database was compiled. The weightings for each component are given in Table 3-4.

3.1.3.5 Estimating Risk Under Different Scenarios

To estimate air toxics risk estimates under different scenarios, I/M weighted emission factors for each component of a scenario were weighted by the percent of total fuel use for the component on a calendar year basis, to obtain overall emission factors for each scenario. These emission factors for each scenario were then multiplied by urban and rural g/mile to $\mu g/m^3$ conversion factors, obtained from the Hazardous Air Pollutant Exposure Model for Mobile Sources (HAPEM-MS; Johnson et al., 1992), to obtain urban and rural annual average exposures. These urban and rural annual average exposures were then applied to the equation described in Section 4.1 to calculate urban and rural cancer cases in a given year for the air toxic of interest.

3.1.4 MOBTOX Emissions Model Inputs

3.1.4.1 HC Exhaust Reductions for Gasoline Oxygenated Blends

MOBTOX also requires a single input for TOG exhaust reduction for gasoline oxygenated blends. MOBTOX already calculates changes in evaporative emissions with gasoline in the same fashion that MOBILE4.1 does. MOBILE4.1 does this calculation for evaporative emissions solely as a function of RVP.

Table 3-4. I/M Program Weightings for the Various Components Under Different Scenarios.

Percent of Total Fuel Use Within Components for Each I/M Program

		1990	1995		2000, 2010		
Components/Scenarios	I/M Program	Base Control	Base Control	Expanded Reform. Gasoline Use	Base Control	Expanded Reform. Gasoline Use	Expanded Adoption Calif. Standards
Baseline Gasoline Use Federal Standards	None Basic Enhanced	32 68 0	37 39 24	49 35 16	37 39 24	48 36 16	88 12 0
Baseline Gasoline Use California Standards	None Basic Enhanced						85 15 0
Federal/Calif. Reform. Gasoline Use Federal Phase 1 (1995-1999) Calif. Phase 1 (1992-1995) Federal/Calif. Standards	None Basic Enhanced		0 15 85	17 35 48			
Federal Reform. Gasoline Use Phase 2 (2000+) Federal Standards	None Basic Enhanced				 100	20 34 46	25 75 0
Federal Reform. Gasoline Use Phase 2 Calif. Standards	None Basic Enhanced						19 18 63
Winter Oxygenated Gasoline Use Federal/Calif. Standards (1995) Federal Standards (2000, 2010)	None Basic Enhanced		2 17 81	2 17 81	2 16 82	2 16 82	9 63 28
Winter Oxygenated Gasoline Use Calif. Standards (2000, 2010)	None Basic Enhanced				0 22 78	0 22 78	0 7 93
California Only Calif. Reform. Gasoline Use Phase 2 (1996+) Calif. Standards	None Basic Enhanced				0 33 67	0 33 67	0 33 67
Gasohol Fuel Use Federal Standards	None Basic Enhanced	32 68 0	37 39 24	49 35 16	37 39 24	48 36 16	88 12 0

However, changes in exhaust hydrocarbons for gasoline oxygenated blends were not included in MOBILE4.1 even though changes in exhaust CO were included. The changes in exhaust CO were based on an analysis of the EPA emission factor data base (EPA, 1991b). A similar analysis has since been done for exhaust TOG emissions for gasoline oxygenated blends using the emission factor data (EPA, 1992c). Also, an analysis using the emission factor data was done for Phase 1 and Phase 2 reformulated gasoline for exhaust NMHC (EPA, 1992d); similar reduction would be found for TOG. These analyses were done for both normal and high emitting vehicles since the two classes of vehicles have different emission benefits (higher emitting vehicles achieve a greater benefit with the use of reformulated gasoline).

The reformulated gasoline analysis shows a 9.4% exhaust NMHC reduction for a Phase 1 reformulated gasoline (with 2.0% oxygen content) which will be assumed to be the same regardless of the type of I/M program used (none, basic, enhanced)[1]. The remaining reduction required for the minimum 15% total vehicle emission reduction comes from reduced evaporative emissions from lower RVP in the reformulated gasoline -- 8.1 psi for Class C areas compared to an 8.7 psi baseline value. The MOBTOX runs were done assuming temperature ranges (68-84°F) and RVPs for Class C areas. For the purposes of this report, where benzene is the only toxic component of evaporative emissions and the evaporative benzene contribution is small compared to the exhaust benzene, it is assumed that the same proportional reductions are obtained for Class A and B areas as for Class C. A somewhat similar assumption is being used for temperature with the summertime Class C type temperatures assumed to be representative of the country as a whole for establishing ratios of vehicle toxic emissions for the different components of the scenarios for the years examined (EPA, 1992a).

This analysis also shows that the Phase 2 exhaust NMHC reduction depends on the stringency of the I/M program. For either no I/M or a basic I/M, the exhaust reduction is 10.2% NMHC. Again, the remaining vehicle emission reductions come about from reduced evaporative emissions due to lower gasoline RVP; a 7.8 psi RVP is assumed for Class C areas. An enhanced I/M program (which catches vehicles with high evaporative emissions, resulting in necessary repairs and a lowering of these emissions) increases the need for greater exhaust emission reductions to meet the minimum 20-25% total emission reduction. A 14.4% exhaust NMHC benefit is projected for Phase 2 fuel with an enhanced I/M program. For the purposes of this report, a single emission reduction of 22.5% (the average of the 20% and 25% numbers) is being used.

---

[1]This exhaust NMHC reduction (and NMHC reductions given in the following paragraphs) was calculated relative to baseline fuel, rather than indolene. Because of limitations in the MOBTOX model, hydrocarbon emission levels for indolene and baseline fuel were assumed to be comparable.

Also, emission reduction benefits have to be assigned to Phase 2 California reformulated gasoline. Based on an EPA analysis of Arco data (DeJovine et al., 1991), an initial number to use is a 23% exhaust reduction benefit (EPA, 1992e).

For the winter oxygenate program, TOG reductions are based on a gasoline with 2.7% oxygen. These numbers come from an analysis of the EPA emission factor program (EPA, 1992c) and by extrapolating the Phase 1 reformulated gasoline analysis from 2.0 to 2.7% oxygen content. This results in a 12.7% exhaust TOG benefit for the winter oxygenate program.

Finally, an exhaust benefit is needed for use of gasohol. The recent EPA analysis (EPA, 1992c) shows approximately a 15% TOG exhaust benefit from use of ethanol. This benefit can be calculated by assuming that the emission factor data represent a typical in-use spectrum of vehicles so that all the data can be averaged. The same number is obtained if the benefits for normal, high, and very high emitters are taken and applied to the proportion of these vehicles for the 1990 in-use fleet. However, this analysis shows a lower benefit for 10% ethanol relative to 15% MTBE for PFI normal and high emitters, but a higher ethanol benefit relative to MTBE for PFI very high emitters. In 2000 and 2010, the relative number of very high emitters is expected to be lower. Also, this analysis shows a higher ethanol benefit for carbureted than fuel injected vehicles, and carbureted vehicles are likely to represent a very small portion of the fleet in 2000 and 2010. Thus, the 15% TOG exhaust benefit from use of ethanol might be an overestimate for these years. In these later years, an EPA estimate of a 9.6% NMHC exhaust benefit from use of 10% ethanol (1992f), calculated for 1990 technology type vehicles with 1990 sales weightings for each technology type, might be more appropriate. Consideration is being given to modification of MOBTOX in later years to reflect this difference.

The benefits for the winter oxygenate and gasohol components are assumed to be constant with calendar year, unlike the reformulated gasoline benefits, which increase in 2000 versus 1995. The CAAA specify increased benefits for reformulated gasoline in 2000. It is expected that fuel parameters such as lower sulfur levels and changes in distillation characteristics will give the increased benefit; it is also expected that these parameters will not change in areas of the country where non-reformulated gasoline is being used. The reformulated gasoline proposed rulemaking (EPA, 1991c) prohibits gasoline in the non-reformulated areas from deteriorating as the oil companies produce reformulated gasoline.

In all these analyses, the benefits derived from 3-way catalyst vehicles are being applied to the in-use fleet rather than using separate benefits for 3-way catalyst, oxidation catalyst, and non-catalyst vehicles. Doing a separate weighting makes little difference. First, previous EPA guidance (EPA, 1988) shows oxidation catalyst equipped vehicles obtain 14.5% and 12% exhaust emission benefits with 3.5% and 2.7% oxygen blends (gasohol and MTBE/gasoline). These numbers are remarkably close

to the 15% and 12.7% benefits for the 3-way catalyst fleet. The only years where oxidation catalyst vehicles would have any noticeable impact is the 1990 and 1995 runs; no effect would be seen for the 2000 and 2010 projections. Also, it is assumed that the benefits for the 3-way catalyst vehicles would be the same as for the 3-way plus oxidation catalyst vehicles that will be used more in the future to meet stricter exhaust emission standards. Since cars meeting future exhaust emission standards may have less open-loop operation where electronic feedback does not control exhaust TOG as much, the benefits may be slightly lower for the newer cars. However, limited or no data are available with which to make projections for benefits for future cars. Thus, the same benefits are being assumed for cars with 3-way catalysts and 3-way plus oxidation catalysts.

3.1.4.2 California LEV Standards

As mentioned previously, California has separate 50,000 mile exhaust emission certification standards for TLEVs, LEVs, ULEVs, and ZEVs, beginning in 1994. MOBTOX only accounted for these separate categories in vehicle classes less than or equal to 8500 lbs. Also, MOBTOX did not account for intermediate compliance standards. Table 3-5 lists the 50,000 mile exhaust emission certification standards, zero mile emission levels, 50,000 mile deterioration rates, and 100,000 mile deterioration rates used in MOBTOX for California vehicle emission categories with test weights less than or equal to 8500 lbs.

When California standards are combined with what EPA defines as an "appropriate" I/M program, greater emission reductions would be expected than with no I/M, basic I/M, or even enhanced I/M. Thus, lower deterioration rates would be used in modeling with California LEV standards than with federal standards. (EPA defines appropriate I/M as an I/M program that would ensure vehicles will meet California LEV standards in use.) However, since this analysis did not assume all areas with California LEV standards would have appropriate I/M, lower deterioration rates were not used. Thus, emission factors for components with California emission standards are higher than they would be if areas adopting these standards also adopted appropriate I/M concurrently.

Vehicles are classified in Table 3-5 by California emission categories within Federal weight categories, rather than the comparable California weight categories. These categories include passenger cars, or light duty gasoline vehicles (LDGVs), and four categories of light duty gasoline trucks (LDGTs 1a, 1b, 2a, 2b). ZEVs are not included in the table, since values in all categories are zero.

Table 3-6 lists the phase-in schedule used in MOBTOX for TLEVs, LEVs, ULEVs, and ZEVs. Although California has separate phase-in schedules for light duty and medium duty vehicles, both based on market shares, two phase-in schedules could not be incorporated into MOBTOX due to limitations of the model. Instead, a combined phase-in schedule was input into the model,

with fleet percentages weighted according to model year market share projections for light and medium duty vehicles. Any error introduced into the model by combining phase in schedules would be minor, since the market share of medium duty vehicles is small relative to light duty vehicles.

3.1.4.3 Toxic Exhaust Fractions

Emission fractions were disaggregated by vehicle class and catalyst type for exhaust emissions, and fuel system for evaporative emissions.

The following vehicle classes were included in the calculations: LDGVs, LDGTs, heavy-duty gasoline vehicles (HDGVs), light duty diesel vehicles (LDDVs), light duty diesel trucks (LDDTs) and heavy duty diesel vehicles (HDDVs). These vehicle classes are consistent with those in MOBTOX. LDGTs and LDDTs were assumed to have the same mass fractions as LDGVs and LDDVs, respectively. For LDGV/LDGT exhaust emissions, fractions were disaggregated by four catalyst types -- non-catalyst, oxidation catalyst, three-way catalyst, and three-way plus oxidation catalyst. For LDGV/LDGT evaporative emissions, fractions were disaggregated by fuel system -- either carbureted or fuel injection (PFI and TBI were considered to be the same so we simply pooled all the fuel injection data). HDGVs were assumed to have either no catalyst or a three way catalyst with a carbureted fuel system. Calculations were done for vehicles running on non-oxygenated gasoline, 10% ethanol, 5.5% MTBE, 9.0% MTBE, 12.5% MTBE, 15% MTBE, and 16.4% MTBE. Fuels with these MTBE levels were used in major test programs.

All exhaust mass fractions were calculated as fractions of total organic gases (TOG), on a vehicle by vehicle basis. TOG includes methane, ethane, and all oxygenated hydrocarbons, such as aldehydes, and also alcohols and ethers when oxygenated blends are used. Mass of total hydrocarbons (THC), as determined by the flame ionization detector (FID), was multiplied by a THC to TOG composite correction factor (CCF). A recent EPA analysis (1991d) described the procedure for generating THC to TOG correction factors for various vehicle class/catalyst combinations running on gasoline or diesel. These are the same correction factors used in MOBILE4.1 and MOBTOX. Although actual TOG values exist for much of the data, this approach of calculating TOG using a correction factor was used so that the emission fractions derived are consistent with the TOG values contained in MOBILE4.1/MOBTOX. A summary of CCFs for

Table 3-5. Zero Mile Levels and Deterioration Rates Based on California Exhaust Emission Certification Standards for Low Emission Vehicles.

Vehicle Emission Category	50,000 Mile Exhaust Emission Standard	Zero Mile Level	50,000 Mile Deterioration Rate	100,000 Mile Deterioration Rate
LDGV TLEV	0.125	0.1001	0.0518	0.0748
LDGV LEV	0.075	0.0600	0.0518	0.0748
LDGV ULEV	0.040	0.0320	0.0518	0.0748
LDGT1a (≤ 3750 lbs.) TLEV	0.125	0.1001	0.0518	0.0748
LDGT1a LEV	0.075	0.0600	0.0518	0.0748
LDGT1a ULEV	0.040	0.0320	0.0518	0.0748
LDGT1b (3751-5750 lbs.) TLEV	0.160	0.1281	0.0518	0.0748
LDGT1b LEV	0.100	0.0800	0.0518	0.0748
LDGT1b ULEV	0.050	0.0400	0.0518	0.0748
LDGT2a (3751-3500) (California medium duty) TLEV	0.500	0.4002	0.0768	0.0768
LDGT2a LEV	0.160	0.1281	0.0518	0.0748
LDGT2a ULEV	0.100	0.0800	0.0518	0.0748
LDGT2b (5751-8500) TLEV	0.500	0.4002	0.0768	0.0768
LDGT2b LEV	0.195	0.1561	0.0518	0.0748
LDGT2b ULEV	0.117	0.0937	0.0518	0.0748

Table 3-6. Market Share Fractions for California Low Emission Vehicle Categories.

Year	Federal Standard	TLEV	LEV	ULEV	ZEV
1994	0.91	0.09	0.00	0.00	0.00
1995	0.86	0.14	0.00	0.00	0.00
1996	0.82	0.18	0.00	0.00	0.00
1997	0.75	0.00	0.23	0.02	0.00
1998	0.50	0.00	0.46	0.02	0.00
1999	0.25	0.00	0.71	0.02	0.02
2000	0.02	0.00	0.94	0.02	0.02
2001	0.00	0.00	0.90	0.05	0.05
2002	0.00	0.00	0.85	0.10	0.05
2003+	0.00	0.00	0.76	0.15	0.09

gasoline and diesel fueled vehicles is included in Table 3-7. It should be noted that CARB uses THC to TOG CCFs which are higher than EPA's. Whereas THC as measured by FID assumes a C:H ratio of 1:1.85 for every exhaust HC compound, CARB corrects this FID calculation for the true mix of C:H ratios to more accurately report true mass. Eventually, EPA may adopt this approach. Also, in making its adjustments, CARB inaccurately assumes that all oxygenated compounds (e.g. aldehydes) are not measured by the FID.

When estimating TOG for vehicles using MTBE fuel blends, another adjustment factor had to be introduced to account for the difference in emissions when a car runs on an MTBE blend rather than standard gasoline. A relatively recent EPA analysis (1989b), calculated relative adjustment factors for 0, 11 and 15% MTBE to account for this difference. The adjustment factors are as follows:

1) 1.00 for 0% MTBE
2) 1.0144 for 11% MTBE
3) 1.0197 for 15% MTBE

Unlike the factors for gasoline vehicles, these correction factors are not technology specific. There is a linear relationship between these adjustment factors and MTBE content;

thus, a regression equation could be generated and adjustment factors then calculated for various MTBE levels. The analysis also included a relative adjustment factor for 10% ethanol (1.0232). For a vehicle class/catalyst combination, THC as measured by FID was first multiplied by the THC to TOG CCF, then by this relative oxygenate adjustment factor to account for MTBE or ethanol content.

Table 3-7. THC to TOG Composite Correction Factors.

Vehicle Class	Catalyst Technology	Adjustment Factor
LDGV/LDGT	none	1.0333
LDGV/LDGT	3-way + ox	1.0175
LDGV/LDGT	3-way	1.0125
LDGV/LDGT	ox	1.0170
LDDV		1.0490
HDDV		1.0342
HDGV	none	1.0358
HDGV	3-way	1.0178

Sources for the data used to determine emission fractions are summarized in Appendix B1. Appendix B2 contains a series of spreadsheets listing, on a vehicle by vehicle basis, exhaust emissions (and for benzene, evaporative emissions also) in mg/mile for formaldehyde, acetaldehyde, 1,3-butadiene, and benzene, TOG, and resultant fractions of TOG. For exhaust emissions, vehicles were sorted by class, catalyst type, and fuel, as listed above. For evaporative emissions, vehicles were sorted by class, fuel system and fuel. Averages were calculated for each fuel type within a vehicle class/catalyst or vehicle class/fuel system category. Appendix B3 contains summary spreadsheets listing averages for the various categories.

Because of a surfeit of extensive data on a reasonably large number of vehicles for LDGVs and LDGTs with three-way catalysts, only data from three recent Arco studies and the Auto/Oil Program were used. RDSD, in an early Notice of Proposed Rulemaking on reformulated gasoline standards (EPA, 1991c), limited its analyses to Auto/Oil data (Auto/Oil, 1990); specifically, current 1989-90 type vehicles with three way catalysts, running on an "industry average" fuel, designated fuel A. Although vehicles were tested on a number of other non-oxygenated blends, for three

way catalysts this analysis likewise only used fuel A data for current vehicles. Fuel A matches the baseline fuel specifications in Section 211 of the Act, and this fuel/vehicle technology combination is expected to be the most representative for this analysis. Arco has recently released three studies (Boekhaus et al., 1991a and 1991b, DeJovine et al., 1991), which include a large amount of data on oxygenated fuel blends; thus, it was useful to add these data to the Auto/Oil data.

For other vehicle class/catalyst categories, all available study data (even from programs where only 1 or 2 cars were tested) were used because a very limited amount was available.

3.1.4.4 Other Inputs

MOBTOX runs assumed regions modeled were low altitude regions. Also, the average speed assumed in MOBTOX runs was 19.6 miles per hour. This is the average speed in the FTP test. An average daily temperature of 75°F was assumed. As mentioned in Section 3.1.4.1, the minimum temperature assumed was 68°F and the maximum temperature was 84°F. These represent the average temperature and temperature ranges, respectively, in the FTP.

Like MOBILE4.1 (EPA, 1991e), MOBTOX requires the user to input certain assumptions about operating mode. The federal FTP has three distinct vehicle operating modes: cold start, stabilized, and hot start. The percentage of time vehicles spend in each mode affects emissions (e.g., emissions are higher in cold start mode). MOBTOX requires the percentage of time spent in cold start mode by non-catalyst vehicles, the percentage of time spent in hot start mode by catalyst equipped vehicles, and the percentage of time spent in cold start mode by catalyst equipped vehicles. The inputs in all runs for these three variables were 20.6, 27.3, and 20.6, respectively. The values used for these three variables correspond to the conditions of the FTP.

3.2 Methodology for Diesel Particulate Matter

The Environmental Protection Agency prepared an estimate of diesel particulate emissions in 1983 (EPA, 1983). In the 1983 analysis, EPA assessed the impact of "base" and "relaxed" scenarios on diesel particulate emissions in 1995, relative to those in 1980 and 1986. The base scenario assumed particulate standards would be 0.20 g/mi, 0.26 g/mi, and 0.25 g/BHP-hr for LDDVs, LDDTs, and heavy-duty diesel engines (HDDEs), respectively. The relaxed scenario assumed standards of 0.60 g/mi for LDDVs and LDDTs and 0.60 g/BHP-hr for HDDEs.

In 1986, the Motor Vehicle Manufacturers Association and Engine Manufacturers Association published an analysis of EPA's diesel particulate matter study (MVMA and EMA, 1986). While MVMA

and EMA generally agreed with EPA's methodology for estimating diesel particulate emissions, they felt that many of the inputs EPA used were outdated, and consequently, the contribution of diesel engines to particulate levels was overstated. MVMA and EMA thus estimated particulate emissions using inputs which they felt were more realistic.

Another analysis of diesel particulate emissions was done by EPA in 1987 (Carey, 1987), as part of an air toxics report. This analysis assumed that particulate standards in 1987 and later years would be 0.20 g/mi and 0.26 g/mi for LDDVs and LDDTs, respectively. It also assumed a HDDE standard of 0.60 g/BHP-hr for 1988-1990, 0.25 g/BHP-hr for 1991-1993 (except for buses, at 0.10 g/BHP-hr), and 0.10 g/BHP-hr for 1994 and later.

Recently, Sienicki and Mago (1991) updated spreadsheets from the 1986 MVMA and EMA analysis, and used these updated spreadsheets to predict the total metric tons of diesel particulate matter and concentration in urban areas from on-highway vehicle fleets for the target years of 1995 and 2015. Their analysis included more stringent standards for 1995 and later years, set by EPA, rather than those assumed in the 1983 EPA diesel particulate matter study.

Sienicki (1992a, 1992b) has also used updated analyses to predict total grams of urban diesel particulate matter, as well as national fleet average emission factors, for the years 1990, 1995, 2000, and 2010. These predictions utilize the most recent inputs available; thus, the particulate emission factors derived by Sienicki were used with only minor adjustments to develop diesel particulate matter risk estimates for the air toxics report. Later, EPA may develop particulate emission factors to use in developing risk estimates independently.

A detailed discussion of the methodology is contained in section 9.3.

3.3 Methodology for Gasoline Particulate Matter

Historically, gasoline particulate matter has been difficult to measure accurately due to the extremely low levels in exhaust. As a result, emission data for gasoline particulate matter are sparse. For this report, the available emission data were reviewed. The limited data appear to indicate a correlation between exhaust HC and gasoline particulate emissions. Gasoline particulate matter was thus estimated to be 1.1% of exhaust HC. This percentage was then used in the MOBTOX model to calculate in-use g/mile emission factors for gasoline particulate matter. An alternative approach was to assign a single g/mile value for gasoline particulate matter, based on the emission data. Unfortunately, this alternative approach would not allow expected changes to gasoline particulate emissions with either time or with changes to fuels and/or vehicle standards.

A detailed discussion of the available emission data and the derivation of the exhaust HC percentage is contained in section 10.3.

3.4 References for Chapter 3.0

Adler, J. M. and P. M. Carey. 1989. Air Toxics Emissions and Health Risks from Mobile Sources. Ann Arbor, Michigan: Environmental Protection Agency, Office of Mobile Sources. Prepared for the Air and Waste Management Association. AWMA Paper 89-34A.6.

Auto/Oil Air Quality Improvement Research Program. 1990. Phase 1 Working Data Set (published in electronic form). Prepared by Systems Applications International, San Rafael, CA.

Auto/Oil Air Quality Improvement Research Program. 1991. Technical Bulletin No. 6: Emission Results of Oxygenated Gasoline and Changes in RVP.

Auto/Oil Air Quality Improvement Research Program. 1993. Technical Bulletin No. 11. A Study of Fuel Effects on Emissions from High Emitting Vehicles.

Boekhaus, K. L., L. K. Cohu, L. A. Rapp and J. S. Segal. 1991a. Clean Fuels Report 91-02: Impact of EC-1 Reformulated Gasoline Emissions and Their Reactivity on Five 1989 Cars. Arco Products Co., Anaheim, California.

Boekhaus, K. L., J. M. DeJovine, D. A. Paulsen, L. A. Rapp, J.S. Segal and D. J. Townsend. 1991b. Clean Fuels Report 91-03: Fleet Test Emissions Data -- EC-Premium Emission Control Gasoline. Arco Products Co., Anaheim, California.

California Air Resources Board. 1990. Proposed Regulations for Low-Emission Vehicles and Clean Fuels: Staff Report. Sacremento, California: Mobile Source Division, Stationary Source Division. Release Date: August 13, 1990.

Carey, P. M. 1987. Air Toxics from Motor Vehicles. Ann Arbor, Michigan: U.S. Environmental Protection Agency, Office of Mobile Sources. Publication no. EPA-AA-TSS-PA-86-5.

Carey, P. M., and J. H. Somers. 1988. Air Toxics Emissions from Motor Vehicles. Ann Arbor, Michigan: Environmental Protection Agency, Office of Mobile Sources. Prepared for the Air and Waste Management Association. AWMA Paper 88-128.1.

Dasch, J. M., and R. L. Williams. 1991. Benzene exhaust emissions from in-use General Motors vehicles. Environ. Sci. Technol. 25:853-862.

DeJovine, J. M., K. J. McHugh, D. A. Paulsen, L. A. Rapp, J. S. Segal, B. K. Sullivan, D. J. Townsend. 1991. Clean Fuels Report

91-06: EC-X Reformulated Gasoline Test Program Emissions Data. Arco Products Co., Anaheim, California.

Environmental Protection Agency. 1983. Diesel Particulate Study. Ann Arbor, Michigan: Office of Mobile Sources. October, 1983.

Environmental Protection Agency. 1988. Guidance on estimating motor vehicle emission reductions from the use of alternative fuels and fuel blends. Ann Arbor, Michigan: Office of Mobile Sources. Publication no. EPA-AA-TSS-87-4.

Environmental Protection Agency. 1989a. Analysis of the Economic and Environmental Effects of Methanol as an Automotive Fuel. Ann Arbor, Michigan: Office of Mobile Sources. Special Report, September, 1989.

Environmental Protection Agency. 1989b. Oxygenated fuel exhaust HC, Evap, and MPG corrections. Memo from Craig Harvey to Phil Lorang, April 28, 1989.

Environmental Protection Agency. 1990a. Analysis of the Economic and Environmental Effects of Ethanol as an Automotive Fuel. Ann Arbor, Michigan: Office of Mobile Sources. Special Report, April, 1990.

Environmental Protection Agency. 1990b. Analysis of the Economic and Environmental Effects of Compressed Natural Gas as a Vehicle Fuel; Volume I: Passenger Cars and Light Trucks. Special Report, April, 1990.

Environmental Protection Agency. 1990c. Analysis of the Economic and Environmental Effects of Compressed Natural Gas as a Vehicle Fuel; Volume II: Heavy-Duty Vehicles. Special Report, April, 1990.

Environmental Protection Agency. 1991a. Draft Regulatory Impact Analysis: Reformulated Gasoline and Anti-Dumping Regulations. Ann Arbor, Michigan: Office of Mobile Sources.

Environmental Protection Agency. 1991b. CO reduction with the use of oxygenated blends. Memo from Greg Janssen to Phil Lorang, July 31, 1991.

Environmental Protection Agency. 1991c. Regulation of Fuel and Fuel Additives: Standards for Reformulated Gasoline; Proposed Rule. Federal Register 56(131): 31176-31263.

Environmental Protection Agency. 1991d. Correction factors to convert THC to TOG. Memo from Greg Janssen to Phil Lorang, September 3, 1991.

Environmental Protection Agency. 1991e. User's Guide to MOBILE4.1 (Mobile Source Emission Factor Model). Ann Arbor, Michigan: Office of Mobile Sources. July, 1991.

Environmental Protection Agency. 1992a. Ratios of emission factors at different temperatures for 1990 and 2000. Memo from Greg Janssen to Phil Lorang, February 5, 1992.

Environmental Protection Agency. 1992b. Inspection/Maintenance Program Requirements; Final Rule. Federal Register 57(215): 52950-53014.

Environmental Protection Agency. 1992c. Interim analysis of HC reduction with the use of oxygenated fuel blends with 3-way catalyst vehicles. Memo from Greg Janssen to Phil Lorang, January 23, 1992.

Environmental Protection Agency. 1992d. Emission reduction estimates of phase II reformulated gasoline. Memo from Chris E. Lindhjem to Phil Lorang, January 9, 1992.

Environmental Protection Agency. 1992e. Emission levels of target fuels. Note from Chris E. Lindhjem to Gary Dolce, February 10, 1992.

Environmental Protection Agency. 1992f. Effect of Oxygenate on Emissions. Memo from Chris E. Lindhjem to Richard Rykowski, January 7, 1992.

Environmental Protection Agency. 1993. Regulation of Fuel and Fuel Additives: Standards for Reformulated Gasoline; Proposed Rule. Federal Register 58(37): 11722-11763.

Johnson, T. R., R. A. Paul, and J. E. Capel. 1992. Application of the Hazardous Air Pollutant Exposure Model (HAPEM) to Mobile Source Pollutants. Durham, North Carolina: International Technology Corporation, August 1992.

Motor Vehicle Manufacturers Association and Engine Manufacturers Association. 1986. Analysis of the Environmental Protection Agency's Diesel Particulate Study and a Diesel Particulate Emissions Projection. September 22, 1986.

Motor Vehicle Manufacturers Association. 1990. National Fuel Surveys: Gasoline and Diesel Fuel. Summer 1990.

Nebel, G. J. 1981. The effect of misfueling on aldehyde and other auto exhaust emissions. APCA Journal 31: 877-879.

Sienicki, E. J. and R. S. Mago. 1991. Reevaluation of Diesel Engine Particulate Emission Inventories. Presented at Air and

Waste Management Association Conference, October 16-18, 1991, Detroit, Michigan.

Sienicki, E. J. 1992a. Letter from E. J. Sienicki, Navistar International Transportation Corporation, to Phil Lorang, Environmental Protection Agency, January 6, 1992.

Sienicki, E. J. 1992b. Letter from E. J. Sienicki, Navistar International Transportation Corporation, to Phil Lorang, Environmental Protection Agency, April 23, 1992.

Sigsby, J. E., S. Tejeda, W. Ray, J. M. Lang, and J. W. Duncan. 1987. Volatile organic compound emissions from 46 in-use passenger cars. Environ. Sci. Technol. 21:466-475.

Smith, L. R. and P. M. Carey. 1982. Characterization of exhaust emissions from high mileage catalyst-equipped automobiles. SAE Paper 820783.

Stump, F. D., S. Tejada, W. Ray, D. Dropkin, F. Black, R. Snow, W. Crews, P. Siudak, C. O. Davis, L. Baker and N. Perry. 1989. The influence of ambient temperature on tailpipe emissions from 1984 to 1987 model year light-duty gasoline vehicles. Atmospheric Environment 23: 307-320.

Stump, F. D., S. Tejeda, W. Ray, D. Dropkin, F. Black, R. Snow, W. Crews, P. Siudak, C. O. Davis and P. Carter. 1990. The influence of ambient temperature on tailpipe emissions from 1985-1987 model year light-duty gasoline vehicles -- II. Atmospheric Environment 24A: 2105-2112.

Stump, F. D., K. T. Knapp, W. D. Ray, R. Snow and C. Burton. The composition of motor vehicle organic emissions under elevated temperature summer driving conditions (75 to 105°F). Unpublished.

U.S. Department of Transportation. 1990. Highway Statistics 1990. Washington, D.C.: Federal Highway Administration.

U.S. Department of Transportation. 1991. Monthly Motor Fuel Reported by States, April 1991. Washington D.C.: Federal Highway Administration. Publication no. FHWA-PL-91-011.

Urban, C. M. 1980a. Regulated and Unregulated Exhaust Emissions from Malfunctioning Non-Catalyst and Oxidation Catalyst Gasoline Automobiles. Ann Arbor, Michigan: U.S. Environmental Protection Agency, Office of Mobile Sources. Publication no. EPA-460/3-80-003.

Urban, C. M. 1980b. Regulated and Unregulated Exhaust Emissions from Malfunctioning Three-Way Catalyst Gasoline Automobiles. Ann Arbor, Michigan: U.S. Environmental Protection Agency, Office of Mobile Sources. Publication no. EPA-460/3-80-004.

Urban, C. M. 1980c. Regulated and Unregulated Exhaust Emissions from a Malfunctioning Three-Way Catalyst Gasoline Automobile. Ann Arbor, Michigan: U.S. Environmental Protection Agency, Office of Mobile Sources. Publication no. EPA-460/3-80-005.

Urban, C. M. 1981. Unregulated Exhaust Emissions from Non-Catalyst Baseline Cars Under Malfunction Conditions. Ann Arbor, Michigan: U.S. Environmental Protection Agency, Office of Mobile Sources. Publication no. EPA-460/3-81-020.

Urban, C. M., and R. J. Garbe. 1979. Regulated and unregulated exhaust emissions from malfunctioning automobiles. SAE Paper 790696.

Urban, C. M., and R. J. Garbe. 1980. Exhaust emissions from malfunctioning three-way catalyst-equipped automobiles. SAE Paper 800511.

Warner-Selph, M. A., and L. R. Smith. 1991. Assessment of Unregulated Emissions from Gasoline Oxygenated Blends. Ann Arbor, Michigan: U.S. Environmental Protection Agency, Office of Mobile Sources. Publication no. EPA-460/3-91-002.

Wetrogan, S. I. 1990. Projections of the Populations of States by Ages, Sex, and Race: 1989 to 2010. Washington, D.C.: U.S. Department of Commerce, Bureau of the Census. Series P-25, publication no. 1053.

4.0 EXPOSURE METHODOLOGY

This chapter describes the methodology used to project exposure to motor vehicle air toxics. Exposure estimates have to be made for two types of situations. The first is an overall annual exposure estimate which can be used for carcinogenic risk assessments in the linear no-threshold model used by EPA to predict cancer impact. In this model, the lifetime or annual cancer impact is the product of the lifetime or annual average exposure level times the potency of the substance. The second exposure estimate needed is a localized exposure estimate for specific microenvironments highly impacted by mobile source emissions. Such microenvironments include urban street canyons, congested freeways, large commercial parking garages where many vehicles exit at once such as after a sporting event, residential garages attached to homes, and even roadway tunnels. The concern with exposure in these microenvironments generally is acute non-cancer effects.

These exposures can be estimated two ways. The first is use of models to predict either annual exposure or exposure in certain microenvironments. The second is using ambient data. Ambient data can be used to estimate annual average exposures or even localized exposure in microenvironments depending on monitor location and averaging time for exposure. However, few monitors are designed to collect short term averages of motor vehicle emissions in microenvironment areas where the highest exposures would be expected (such as residential garages).

4.1 Annual Average Population Exposure Estimation

EPA work on developing models has emphasized those that predict annual average exposure. The models predicting annual average exposure assume a person's actual exposure can be predicted by levels measured at the monitors set up to measure compliance with the National Ambient Air Quality Standards. Several years ago, EPA conducted a number of studies measuring human exposure to carbon monoxide in Washington D.C. and Denver during the winter of 1982-83 after some initial work was done in Stamford, Connecticut (Akland et al., 1985; Johnson, 1984; Clayton et al., 1985; Hartwell et al., 1984; Settergren et al., 1984, Rumba, 1981). In these studies, individuals carried carbon monoxide monitors as they went about their day to day activities. The individuals recorded their activities in a diary and the personal CO monitor recorded the CO level during each activity. When a person changed activities (as defined by guidelines given to the person carrying the monitor), the person reset the monitor so each monitor reading was associated with only one activity. The measurements were taken over approximately 100 days. On each day, about 10 different individuals were selected to use the monitors. These studies were used to determine the relationship of personal exposure levels to those found at the NAAQS monitors. This work showed very good correlation between the monitor values and ambient exposure for all groups except the top 10% of the

exposed individuals which had greater exposure than would be predicted by the NAAQS monitors.

The EPA Office of Mobile Sources has adapted two models developed by the EPA Office of Air Quality Planning and Standards to predict annual average exposure to motor vehicle carbon monoxide as a function of emission rate. The first is the NAAQS (National Ambient Air Quality Standard) Exposure Model or NEM which was originally developed to predict exposure to carbon monoxide. The second is the Hazardous Air Pollution Exposure Model (HAPEM), which was originally developed to predict exposure to air toxics generally from specific point sources. A version of HAPEM adopted for mobile sources, called HAPEM-MS (Johnson et al., 1992), is used in this study.

4.1.1 The NAAQS Exposure Model (NEM)

In order to understand HAPEM-MS, it also helps to understand the NAAQS Exposure Model (NEM). The NEM has been used by EPA in the past to estimate nationwide annual person-hours of exposure to specific levels for any mobile-source pollutant of interest. The model relies on an activity pattern model that simulates a set of population groups called cohorts as they go about their day-to-day activities. Each of these cohorts is assigned to a specific location type during each hour of the day. Each of several specific location types in the urban area is assigned a particular ambient pollutant concentration based on fixed site monitor data. The model computes the hourly exposures for each cohort and then sums up these values over the desired averaging time to arrive at average population exposure and exposure distributions. Annual average exposures are theoretically possible since a full year's data from fixed site monitors is an input to the model (Johnson and Paul, 1982).

Southwest Research Institute, under EPA contract, modified the NEM so that it would determine exposures specifically from mobile source pollutants (Ingalls, 1985). The CO NEM was selected since outdoor CO is largely a mobile source pollutant, especially in urban areas where about 80% of CO comes from motor vehicles. Since the CO monitoring data, on which the CO NEM was based, can be assumed to be related to mobile source emissions in g/mile, exposure to other mobile source pollutants can be estimated from this model, based on relative concentrations of these pollutants to total emissions. It is important to note, however, that CO is relatively non-reactive photochemically. Thus, non-reactive substances are modeled more accurately.

The CO NEM divides all non-rural areas into the following six neighborhoods:

Urban residential
Urban commercial
Urban industrial
Suburban residential
Suburban commercial
Suburban industrial

The neighborhoods were chosen to match the neighborhood descriptions used in identifying EPA ambient monitor sites. In turn, each neighborhood is divided into the following six microenvironments:

Indoors, work or school
Indoors, home or other
Inside a transport vehicle
Roadside
Outdoors
Kitchen

Each person in a city was assigned to a neighborhood type and to a microenvironment within that neighborhood for each hour of the day. The population was divided into 12 age-occupation groups with each of these groups being divided into subgroups; each group and subgroup were assigned to a particular neighborhood type and microenvironment depending on activity patterns.

Also, time spent in the following three microenvironments heavily impacted by motor vehicles was specifically accounted for:

Street canyons
Tunnels
Parking garages

A total of 99 of the 346 monitors used in the 116 largest urban areas in 1981 were used to assign ambient CO levels for this model. The monitors selected had to meet certain criteria. One was that sufficient hourly data had to be available to calculate an annual average level. Also, the monitor could not be in areas such as street canyons that would be highly impacted by mobile source emissions. Street canyons were represented separately by another set of 23 monitors that are located near street canyons. Moreover, four different microenvironment scaling factors were used (as appropriate) to adjust the ambient monitoring data to better represent CO levels in the locations used in the model (Johnson and Paul, 1982):

Microenvironment	Ambient CO Scaling Factor
Indoors	0.85
Transport vehicle	2.10
Roadside	1.20
Outdoors	0.95

CO emission factors from MOBILE3 have been used for the 1981 calendar year as inputs for each of the six neighborhoods and the three microenvironments specifically impacted by mobile sources. These emission factors are generally based on the FTP. The user assigns as input the emission factor for the compound of interest in the year of interest. In effect, the model takes the ratio of the 1981 CO emission factor to the input emission factor for the compound of interest and calculates the exposure based on the ratio of the emission factors. The output of the model is a listing of person hours exposure in the urban areas in the country as a whole for specific concentration levels.

Rural exposure levels, which are always much lower than urban levels (with the exception of Class 8 heavy duty diesel trucks which are operated mostly on interstate highways from city to city versus in urban areas themselves) can be calculated assuming exposures no greater than 2 ppm for CO.

The NEM has an input for increased population in future years and thus accounts for the greater number of people exposed. However, it does not account for increases in the number of vehicles (i.e., increases in vehicle miles traveled) which is handled separately from the model outputs.

4.1.2 Use of HAPEM-MS Model

The EPA Office of Mobile Sources decided to update the exposure model to incorporate some of the data available from the Denver CO personal monitoring studies as well as some updated personal activity data obtained by EPA in Cincinnati. Also, a model that would predict the actual annual average exposure (and number of people exposed to different annual averages) would have more long term applicability than the modified NEM mentioned above, which predicts only the number of person hours at specific levels giving no specific annual average exposure levels. Knowing the distributions of annual average exposure levels is useful in determining whether there are large numbers of people exposed to higher annual average levels balanced by a large number of people at lower levels versus having the distribution closely grouped around the overall annual average as a whole. Such information can also be useful in evaluating carcinogenic impacts from non-linear models versus the linear no-threshold model used by EPA.

The EPA Office of Air Quality Planning and Standards, in conjunction with its contractor (International Technology) that developed the NEM, developed another exposure model, the Hazardous Air Pollution Exposure Model or HAPEM (Johnson, et al., 1991). This model is generally used to predict annual average exposures to toxic air pollutants dispersing from stationary sources. However, for this study, it was modified to predict annual average exposures to toxic air pollutants from motor vehicles. The modified model is named the Hazardous Air Pollution Exposure Model - Mobile Sources or HAPEM-MS (Johnson et

al., 1992). Like NEM, HAPEM-MS is based on the assumption that CO can be used as a surrogate for motor vehicle exposure.

The first step in adapting this model is to select representative urban and rural areas for exposure estimates. The following 11 model urban areas were selected:

Boston
Denver
Houston
Los Angeles
Minneapolis/St. Paul
New York City
Philadelphia
Phoenix
St. Louis
Spokane
Washington D.C.

Paducah, Kentucky and Farmington, New Mexico were selected as rural areas with sufficient CO monitoring data.

Each urban area was then divided into exposure districts generally based on locations of the CO NAAQS monitors so that the number of exposure districts in the 11 urban areas would equal the number of CO monitors for which annual average data exist for the base year of the modified model (1988). The population was divided into the following demographic groups:

Children, 0 to 5 years old
Children, 0 to 13 years old
Children, 14 to 18 years old
Workers with low probability of outdoor work
Workers with moderate probability of outdoor work
Workers with high probability of outdoor work
Nonworking adults under 35 years old
Nonworking adults 35-54 years old
Nonworking adults 55+ years old

Each demographic group was further subdivided into cohorts such that each cohort represented a distinct combination of home and work locations. The fraction of time spent by each cohort in each exposure district and microenvironment within the exposure district was calculated based on a detailed activity pattern study conducted in Cincinnati in which over 900 subjects completed detailed three-day diaries. The data were adjusted based on season, day type (weekday or weekend), ambient temperature, and other factors (Johnson, 1990). All of the nonworking cohorts were assumed to spend all of their time in the residential exposure district. The working cohorts were assumed to spend their working time in

specific fractions of each exposure district and commuting times were specifically considered (Johnson, et al., 1991).

The model uses CO NAAQS fixed site monitoring data; however, the purpose of siting fixed site monitoring stations is not to adequately measure ambient levels of CO but to locate exceedances of the CO standard. As pointed out by several commentors, data from fixed site monitor locations are not likely to be adequate measures of ambient outdoor CO concentration in the community as a whole. As a result, the monitor values were adjusted based on personal monitoring data obtained in Denver. The personal exposure monitor CO concentrations associated with a particular microenvironment were regressed against simultaneous CO concentrations reported by fixed site monitors to obtain adjustment factors for each microenvironment.

The following five microenvironments and factors with which to adjust the NAAQS CO monitor value were incorporated into this model:

Microenvironment	Factor
Indoors - residence	0.495
Indoors - other locations (e.g., office)	0.619
Outdoors - near road	1.001
Outdoors - other locations	0.758
Inside motor vehicle	1.554

A total of 323 urban areas with a population ranging from 58,000 to 8,600,000 were modeled by grouping each of these areas with one of the 11 model urban areas. These 323 areas were qualitatively grouped with the above 11 based primarily on geographical proximity but also factors such as estimated traffic density and vehicle types used. Thus, not many areas are grouped with New York City since Manhattan and other parts of New York City have relatively unique traffic density and vehicle types used compared even to other large Northeastern urban areas such as Philadelphia, Boston, and Washington D.C.

CO exposures for areas grouped with the above 11 modeled areas are adjusted based on annual average CO levels in 1988 for the urban area of interest versus the model area with which it is being grouped. For the few areas where average annual CO levels are not available, the CO levels were estimated to be the median of those for the other areas grouped with the same model urban area. The combined population of the urban areas (334 cities total) was 189,000,000.

All rural type areas were grouped with one of two model rural areas (Paducah, Kentucky and Farmington, New Mexico). Exposure in these areas was also estimated. The rural population totaled 57,000,000.

Annual average urban and rural CO exposures in 1988, as predicted by HAPEM-MS, are 842 and 470 $\mu g/m^3$, respectively. The

1988 fleet average carbon monoxide emission factor is estimated to be 29.6 g/mile using MOBILE4.1. In MOBILE4.1 runs, all areas were assumed to be Class C. The minimum temperature was assumed to be 68°F and the maximum temperature was 84°F. Gasoline was assumed to have an RVP of 10.5 psi. 32% of the country was assumed to have no I/M and 68% was assumed to have basic I/M.

The concentrations predicted by HAPEM-MS for 1988 were divided by the 1988 MOBILE4.1 emission factor to get the g/mile to $\mu g/m^3$ conversion factors shown below for both urban and rural areas.

$$CONV_{urban} = 28.4\ (\mu g/m^3)/(g/mile)$$

$$CONV_{rural} = 15.9\ (\mu g/m^3)/(g/mile)$$

MOBILE5a, an update of MOBILE4.1, has been prepared for release since this analysis was done. If MOBILE5a CO emission factors were used in estimating the g/mile to $\mu g/m^3$ conversion factors, the factors would be roughly 30-35% lower. However, it should be noted that the toxic emission factors using MOBILE5a would be roughly 25-40% higher; thus, the overall cancer risk would not change appreciably.

To obtain exposure estimates for the scenario of interest, these conversion factors are multiplied by the emission factor for the scenario of interest. An additional adjustment factor is applied to account for the increase in vehicle miles travelled (VMT) in excess of the population increase for the year of interest relative to 1988 (EPA, 1992; Wetrogan, 1990). These adjustment factors are given below:

$$ADJ_{1990} = 1.031$$
$$ADJ_{1995} = 1.123$$
$$ADJ_{2000} = 1.218$$
$$ADJ_{2010} = 1.412$$

This additional factor is applied because HAPEM-MS does not account for changes in VMT.

There are a number of limitations inherent in HAPEM-MS that should be taken into account when reviewing the results. First, the fixed site monitoring data were not adjusted to account for non-motor vehicle sources of CO, since motor vehicles are thought to be the predominant source of CO in urban areas. This would serve to overestimate the motor vehicle exposure estimates. The microenvironment factors built into the model attempt to account for other sources of CO to some extent by using subjects that were nonsmokers and using indoor CO levels only in homes with no CO sources (e.g., gas stove, smokers).

Also, the reliability of the present methodology depends on the representativeness of the population by 6 cohorts which are exposed to concentrations within 5 microenvironments. Based on the study of available exposure measurements, the upper 10

percentile of the population exposures (e.g. tollbooth attendants) is believed to be underestimated. The present use of annual age concentrations to determine cancer risk assumes that the dose-response relationship is linear. Improved methodology must be developed before a non-linear dose-response relationship could be used. In addition, assessing chronic non-cancer effects will require consideration of a distribution of annual exposures (e.g., the 90th percentile) and not simply the annual mean average.

The microenvironment factors were estimated using data obtained from one city (Denver) over a four month period during the winter of 1982-1983. There is uncertainty as to whether the resulting estimates are applicable to other areas and other seasons. The same general comment also applies to the activity pattern data, which were collected in a single city (Cincinnati).

CO data from only two rural areas were used to extrapolate to all rural areas in the U.S. There is uncertainty regarding the representativeness of these two areas.

Finally, there are uncertainties regarding the use of CO as a surrogate for motor vehicle toxic emissions. The microenvironment factors may vary by pollutant. In addition, HAPEM-MS relies on the assumption that the ratio of emission factors for CO and the toxic of interest remains constant for the entire U.S. Any variation in these ratios between or within cities is not accounted for in HAPEM-MS. Also, the model assumes that the rates of release and chemical transformation for the toxic of interest is similar to CO. This will not be valid for the more reactive pollutants such as 1,3-butadiene. This is addressed in more detail in the individual pollutant chapters.

4.1.3 Use of Ambient Monitoring Data

Urban ambient monitoring data will be used to check the reasonableness of the HAPEM-MS modeling results. Several EPA data bases exist which contain the results of various air toxics monitoring programs. These programs have set up monitoring devices which are used to collect air samples all over the United States over a period of months or years. Scientists at EPA and elsewhere analyze these samples to determine the total mass and identity of various volatile organic compounds (VOCs) collected. These VOCs include the toxics benzene, 1,3-butadiene, formaldehyde, and acetaldehyde.

One of these programs is the Aerometric Information Retrieval System (AIRS), which became operational in 1987 and utilizes a network of monitoring stations called the State and Local Air Monitoring System (SLAMS) (EPA, 1989a). This network consists of monitoring stations set up by every state in accordance with regulations promulgated in response to requirements of the Clean Air Act. The Office of Air Quality Planning and Standards (OAQPS) administers the AIRS program using its computer facilities at Research Triangle Park, North

Carolina. OAQPS also established another network of monitoring stations called the National Air Monitoring System (NAMS). The NAMS network is part of the larger SLAMS network but must meet more stringent monitor location, equipment, and quality standards.

The AIRS program allows state and local agencies to submit local air pollution data and also have access to national air pollution data (EPA, 1989a). EPA uses data from AIRS in order to monitor the states' progress in attaining air quality standards for ozone, carbon monoxide, nitrogen oxides, sulfur oxides, and lead through the use of State Implementation Plans (SIPS). In addition to containing information about each monitoring site, including the geographic location of the site and who operates it, the AIRS program also contains extensive information on the ambient levels of many toxic compounds. These include compounds specifically discussed in this report: benzene, 1,3-butadiene, formaldehyde, and acetaldehyde. The AIRS database catalogues ambient air pollution data from 18 to 55 monitors in 15 to 23 urban areas, depending on the pollutant. These monitors collect a 24 hour sample every 12 days. However, in some cases not every target compound was detected in every sample. The samples in which this occurred for the compounds specifically mentioned above were included as half the minimum detection limit in the averaging of the data for this report.

The AIRS database also contains data from the Toxic Air Monitoring System (TAMS) (Evans, 1990; EPA, 1987, 1988). The TAMS network operated on a routine basis between 1985 and 1989. By 1989, this network included 10 monitoring sites in the metropolitan areas of Boston, Chicago, Houston, and Seattle/Tacoma. Working with state and local agencies and receiving guidance from OAQPS, EPA's Atmospheric Research and Exposure Assessment Laboratory (AREAL) in Research Triangle Park, North Carolina, administered the TAMS program. The objectives of this program included evaluating methods of sample collection and analysis specifically for toxic air pollutants, beginning to characterize ambient concentrations in selected urban atmospheres, comparing concentration profiles among and within urban areas, establishing baseline levels for trend assessments, and transferring monitoring technology and results to EPA regional offices as well as to state and local agencies. The TAMS program focused on attempting to monitor 96 volatile organic compounds, including benzene and formaldehyde. Monitoring devices collected a 24 hour sample every 12 days. Data listed and used to calculate average concentrations of benzene and formaldehyde were collected between 1987 and 1991. The minimum detection limit used in the collection of data was 0.1 ppb. If a compound was not detected in a sample, then the TAMS staff assigned one half the detection limit (0.05 ppb) as the amount of the compound detected.

Another air monitoring program is the Urban Air Toxic Monitoring Program (UATMP), which the EPA developed in 1987 to assist state and local agencies in determining the nature and

extent of urban air toxic pollution (McAlister et al., 1989, 1990, 1991; Wijnberg and Faoro, 1989). Data from the UATMP is also used in air toxic risk assessment models (EPA 1989b,c; EPA 1990 a,b). In 1989, the UATMP had 14 monitors in 12 urban areas. These urban areas included Camden, New Jersey; Washington, D.C.; Miami, Pensacola, and Ft. Lauderdale, Florida; Chicago and Sauget, Illinois; Dallas and Houston, Texas; Baton Rouge, Louisiana; Wichita, Kansas; and St. Louis, Missouri. In 1990, the UATMP had 12 monitors in 11 urban areas, of which 9 also participated in the 1989 monitoring program. These 9 urban areas are Camden, New Jersey; Washington, D.C.; Pensacola, Florida; Chicago and Sauget, Illinois; Houston, Texas; Baton Rouge, Louisiana; and Wichita, Kansas. Urban monitors added included Orlando, Florida; Toledo, Ohio; and Port Neches, Texas.

In 1989 and 1990, the UATMP network simultaneously monitored 37 non-methane organic compounds, selected metals, benzo(a)pyrene (1989 only), formaldehyde, acetaldehyde, and acetone for a 24 hour period once every 12 days. The UATMP database lists the data collected from the monitoring network using two methods. In the first method, only the concentrations above the detection limit of the compound are included in the data. In the second method, if the concentration of a compound is zero or below the detection limit, then one half of the compound's detection limit is incorporated into the data. The second method was used because it seemed more accurate and allowed a greater number of samples to be averaged. Data collected in 1989 and 1990 were studied for this report.

The 1990 UATMP ambient monitoring data presented two unique situations. The first of these was the inclusion of Port Neches, Texas in the sampling program. This urban area does not affect the overall average for benzene, formaldehyde, or acetaldehyde, but the effects are significant for 1,3-butadiene. Port Neches, Texas does possess areas with high point source concentrations and, coupled with the fact that the location of the monitor is difficult to ascertain in relation to the point sources, the decision was made to exclude the 28 samples from Port Neches from the final average ppb for the entire program. This changes the ambient mean level from 1.02 ppb to 0.14 ppb.

The second situation involves the problem of previous ozone interference when testing the carbonyl samples. Beginning with the 1990 UATMP program, ozone was removed from ambient air through the use of an ozone denuder. This ozone denuder was added to the sampling system after the heated sample probe to eliminate ozone, which is an interferant with the material used to trap the carbonyls in the sampling cartridge. The use of an ozone denuder in the sampling system results in higher and presumably more accurate reported formaldehyde concentrations; hence, only 1990 UATMP carbonyl data will be used to determine ambient levels of formaldehyde and acetaldehyde.

The National Ambient Volatile Organic Compounds (NAVOC) Data Base contains approximately 175,000 records on the observed

concentrations of 320 VOCs observed in one hour air samples taken every 24 hours between 1970 and 1987 (Shah et al., 1988; Hunt et al., 1988). However, only the most current NAVOC data, taken during 1987, is used in this report. In addition, samples which had zero concentrations of the four compounds discussed in this section were included in averaging the data for this report. These air samples were collected using indoor and outdoor monitoring devices. Personal monitors were also used. The types of locations of outdoor monitoring sites included remote, rural, suburban, and urban areas, as well as near specific point sources of VOCs. Indoor monitoring sites consisted of non-industrial workplaces and residential environments. Personal monitors are also included in the indoor category. This database was an interim precursor to the air toxics portion of (AIRS). For this report, only the outdoor urban data were used.

Table 4-1 summarizes the average concentrations (in ppb) of benzene, 1,3-butadiene, formaldehyde, and acetaldehyde found at the monitoring sites of each air monitoring program. The table also shows the total number of observations for each average and the number of sites which monitored the compounds in each program. For AIRS, the average concentrations of the four compounds are listed separately for 1987 through 1989. It should be noted that methods of averaging the data are not consistent between air monitoring databases. Also, in the NAVOC monitoring network, samples were taken for one hour in a 24 hour period while the other monitoring networks collected a 24 hour air sample every 12 days.

Table 4-1. Summary of Air Monitoring Program Results For Benzene, 1,3-Butadiene, Formaldehyde, and Acetaldehyde

	Benzene	1,3-Butadiene	Formaldehyde	Acetaldehyde
AIRS				
1987 Level				
(ppb)	2.13	----	2.79	1.34
# Obs.	422	----	100	82
# Site	23	----	14	13
1988 Level				
(ppb)	1.27	0.46[a]	2.65	1.63
# Obs.	560	12	293	253
# Sites	36	2	16	16
1989 Level				
(ppb)	1.28	----	----	----
# Obs.	373	----	----	----
# Sites	13	----	----	----
1990 Level				
(ppb)	----	0.21[a]	----	----
# Obs.	----	97	----	----
# Sites	----	6	----	----
1991 Level				
(ppb)	----	0.10	----	----
# Obs.	----	117	----	----
# Sites	----	6	----	----
UATMP				
1989 Level				
(ppb)	1.96	0.21	2.12	1.36
# Obs.	397	390	418	418
# Sites	14	13	14	14
1990 Level				
(ppb)	1.47	0.14[b]	4.21[c]	1.72[c]
# Obs.	349	321	356	356
# Sites	12	11	12	12
TAMS				
1987-89 Level				
(ppb)	1.31	----	1.75	----
# Obs.	439	----	362	----
# Sites	10	----	10	----
NAVOC				
1987 Level				
(ppb)	2.21	0.34	3.25	----
# Obs.	564	9	36	----
# Sites	31	6	1	----

[a]Average ppb from all four quarter data sites, excluding Houston, Texas.
[b]Average ppb from all sites, excluding Port Neches, Texas.
[c]Average ppb from all sites. All samples had an ozone denuder added; hence, only these ambient levels will be used later in the report, since they accounted for ozone interference.

4.1.4 Procedure for Calculating Cancer Incidences or Deaths

Urban and rural cancer incidences (for 1,3-butadiene, acetaldehyde, formaldehyde) or deaths (for benzene and diesel particulate matter) were calculated for each scenario using the following equation:

$$EXP \times UR \times POP \div 70 = CAN$$

where:

EXP = HAPEM-MS derived urban or rural annual average exposure, $\mu g/m^3$, adjusted to account for the increase in vehicle miles travelled (VMT) in excess of the population increase for the year of interest relative to 1988, as described in Section 4.1.2 above

UR = EPA unit risk in cancer cases or deaths per person exposed in a lifetime to 1 $\mu g/m^3$

POP = urban or rural U.S. population for the year of interest

	Urban	Rural
1990	187,418,000	62,473,000
1995	194,715,000	64,905,000
2000	200,811,000	66,937,000
2010	211,542,000	70,514,000

The population estimates were obtained from Wetrogan, 1990.

70 = years per lifetime

CAN = annual cancer incidences or deaths

Urban and rural cancer incidences or deaths were added to obtain total cancer incidences or deaths. In some cases, the 1990 HAPEM-MS derived exposures were adjusted to better agree with the ambient data. If an adjustment factor was deemed necessary, it was applied to the HAPEM-MS derived exposures for all years. This is discussed in more detail in the individual pollutant chapters.

4.2 Short-Term Microenvironment Exposures

While carcinogenic effects are assumed to have no threshold and are linearly related to exposure levels (even at very low exposure levels), non-carcinogenic effects are assumed to have a threshold. At low enough levels, there would be no adverse effect as would be found at higher levels; thus, the concern is with higher level exposures to these pollutants unless the threshold is low enough to encompass even the low exposure

levels. The higher level short term exposures occur in microenvironments heavily impacted by motor vehicles.

Particular attention needs to be given to the human exposures in microenvironments such as personal garages, public parking garages, in vehicles during transit, and other situations where there is relatively little dispersion of emissions. Maximum exposures are projected in personal garages, based on modeling data. The personal garage scenario was evaluated in the development of the standards for emissions from methanol-fueled motor vehicles (EPA, 1989d). It was determined in that analysis that validation data for the personal garage were not available, so that the accuracy of the model could not be determined. The number of uncertainties uncovered in this rulemaking demonstrated that more investigation into cold idle emissions and exposure modeling is necessary before accurate conclusions can be drawn regarding public health risk in the personal garage. EPA's Office of Research and Development (ORD) is presently re-evaluating the personal garage model. The determination of the health risk in microenvironments in general is also complicated by the fact that health information for non-cancer effects is limited and no RfCs have been developed by EPA for many of the compounds of concern.

The exposure to air toxics in microenvironments will be evaluated by presenting data from studies that have measured toxics concentrations for people in-transit and in various other microenvironments where elevated levels are expected. New methodology must be developed before risks to acute exposures can be assessed.

4.3 References for Chapter 4

Akland, Gerald G., T.D. Hartwell, T.R. Johnson, and R.W. Whitmore. 1985. Measuring human exposure to carbon monoxide in Washington D.C. and Denver, Colorado during the winter of 1982-83. Environ. Sci. and Technol., 19: 911-918.

Clayton, C.A., S.B. White, and S.K. Settergren. 1985. Carbon monoxide exposure of residents of Washington D.C.: comparative analysis. EPA contract report.

EPA. 1987, 1988. Toxic air monitoring system (TAMS) status report. Research Triangle Park, N.C.: Office of Research and Development, Atmospheric Research and Exposure Assessment Laboratory.

EPA. 1989a. AIRS user's guide volumes I-VII. Research Triangle Park, N.C.: Office of Air Quality Planning and Standards. June 1989.

EPA. 1989b. 1989 Urban Air Toxics Monitoring Program. Office of Air Quality Planning and Standards, Research Triangle Park, N.C. EPA Report No. EPA-450/4-91-001.

EPA. 1989c. 1989 Urban Air Toxics Monitoring Program Aldehdye Results. Office of Air Quality Planning and Standards, Research Triangle Park, N.C. EPA Report No. EPA-450/4-91-006.

EPA. 1989d. Standards for Emissions from Methanol-Fueled Motor Vehicles and Motor Vehicle Engines; Final Rule. 40CFR Part 86, Volume 54, No. 68. 14426-14613.

EPA. 1990a. 1989 Urban Air Toxics Monitoring Program. Office of Air Quality Planning and Standards, Research Triangle Park, N.C. EPA Report No. EPA-450/4-91-024.

EPA. 1990b. 1990 Urban Air Toxics Monitoring Program Carbonyl Results. Office of Air Quality Planning and Standards, Research Triangle Park, N.C. EPA Report No. EPA-450/4-91-025.

EPA. 1992. MOBILE4.1 Fuel Consumption Model. Ann Arbor, Michigan: Office of Mobile Sources.

Evans, Gary, F. 1990. Final report on the operation and findings of the toxics air monitoring system (TAMS). Research Triangle Park, N.C.: Office of Research and Development, Atmospheric Research and Exposure Assessment Laboratory.

Hartwell, T.D., C.A. Clayton, R.M. Michie, R.W. Whitmore, H.S. Zelon, S.M. Jones, and D.A. Whitehurst. 1984. Study of carbon monoxide exposure of residents of Washington D.C. and Denver, Colorado. Part I. EPA contract report.

Hunt, William, F., Jr., Robert Faoro, A.B. Hudischewskyj, and A.K. Pollack. 1988. The EPA interim database for air toxics

volatile organic chemicals. Research Triangle Park, N.C.: Systems Applications, Inc. and the U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Publication no. EPA-450/4-88-014.

Ingalls, Melvin N. 1985. Improved mobile source exposure estimation. Environmental Protection Agency. Publication no. EPA-460/3-85-002.

Johnson, Ted. 1984. A study of personal exposure to carbon monoxide in Denver, Colorado. Rsesearch Triangle Park, North Carolina: Environmental Protection Agency. Publication no. EPA-450/5-83-004.

Johnson, T.R. 1990. Estimation of ozone exposure in Houston using a probabilistic version of NEM. Paper 90-150. Air and Waste Management Association Meeting.

Johnson, Ted R., R.A. Paul, J.E. Capel, and W. Ollison. 1991. Estimation of incremental benzene exposure associated with several bulk gasoline storage facilities in North Carolina, 1st Annual meeting of the Internatinal Society of Exposure Analysis, Atlanta, Georgia.

Johnson, Ted, and R.A. Paul. 1982. The NAAQS exposure model (NEM) applied to carbon monoxide. Draft final contract reports by PEDCo Environmental for EPA.

Johnson, T.R., J.E. Capel, and D.M. Byrne. 1991. The estimation of commuting patterns in applicatins of the hazardous air pollutant expsoure model. AWMA Paper no. 91-172.6. Presented at Air and Waste Management Association Meeting, Vancouver, Canada.

Johnson, T.R., R.A. Paul, and J.E. Capel. 1992. Application of the Hazardous Air Pollutant Exposure Model (HAPEM) to Mobile Source Pollutants. International Technology Corporation, Durham, North Carolina.

McAlister, Robert, A., Wendy H. Moore, Joann Rice, Dave-Paul Dayton, Robert F. Jongleux, Phyllis L. O'Hara, Raymond G. Merrill, Jr., and Joan T. Bursey. 1989. Nonmethane organic compound monitoring program final report volume II: urban air toxics monitoring program, 1988. Research Triangle Park, N.C.: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Publication no.: EPA-450/4-89-005.

McAlister, Robert, A., Wendy H. Moore, Joann Rice, Emily Bowles, Dave-Paul Dayton, Robert F. Jongleux, Raymond G. Merrill, Jr., and Joan T. Bursey. 1990. Urban air toxics monitoring program, 1989. Research Triangle Park, N.C.: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Publication no. EPA-450/4-91-001.

McAlister, Robert, A., David L. Epperson, and Robert F. Jongleux. 1991. Urban air toxics monitoring program aldehyde results, 1989. Research Triangle Park, N.C.: Radian Corp. and the U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Publication no. EPA-450/4-91-006.

Rumba, R.G. 1981. Monitoring human exposure to carbon monoxide in Stamford, Connecticut. EPA report.

Settergren, S.K., T.D. Hartwell, and C.A. Clayton. 1984. Study of carbon monoxide exposures of residents of Washington D.C. -- additional analyses. EPA contract report.

Shah, Jitendra, J. and Emily K. Heyerdahl. 1988. National ambient volatile organic compounds data base update (project report). U.S. Environmental Protection Agency, Atmospheric Sciences Research Laboratory, Research Triangle Park, N.C. EPA/600/3-88/010(a).

Shah, Jitendra, J. and Emily K. Heyerdahl. National ambient volatile organic compounds data base update (project summary). U.S. Environmental Protection Agency, Atmospheric Sciences Research Laboratory, Research Triangle Park, N.C. EPA/600/S3-88/010.

Wetrogan, S.I. 1990. Projections of the Population of States by Age, Sex, and Race: 1989 to 2010. Washington, D.C.: U.S. Department of Commerce, Bureau of the Census. Series P-25, publication no. 1053.

Wijnberg, Louis and Robert Faoro. 1989. Urban air toxics monitoring program -- results of aldehyde monitoring fiscal year 1988. PEI Associates and the U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Research Triangle Park, N.C. September 1989.