Motor Vehicle-Related Air Toxics Study

Motor Vehicle-Related Air Toxics Study

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

Acknowledgements

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)

<u>Page</u>

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)

<u>Page</u>

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. Air 1993.[a]

Pollutant	1990 Base Control	1995 Base Control	1995 Expanded Reform. Gasoline Use	2000 Base Control	2000 Expanded Reform. Gasoline Use	2000 Expanded Adoption Calif. Stds.	2010 Base Control	2010 Expanded Reform. Gasoline Use	2010 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/m^3, 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/m^3)	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/m^3)	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/m^3) or CARB, 1984 Unit Risk (5.2×10^{-5} per µg/m^3). These are not directly comparable to the official EPA unit risk estimates.[g]								
Exposure (µg/m^3)	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.

	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 (1.3×10^{-5} per µg/m³). The draft EPA, 1991 estimate is not an official EPA estimate.[g]									
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.

	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 $\mu g/m^3$, 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] ($\mu g/m^3$)	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] ($\mu g/m^3$)	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 $\mu g/m^3$) or ICF, 1986 Upper Bound Unit Risk (3.4×10^{-3} per $\mu g/m^3$). These are not directly comparable to the official EPA unit risk estimate.[g]								
Exposure ($\mu g/m^3$)	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.

Pollutant	1990	1995		2000			2010		
	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 $\mu g/m^3$, 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] $(\mu g/m^3)$	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] $(\mu g/m^3)$	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 $\mu g/m^3$) or CARB, 1992 Upper Bound Unit Risk (2.7×10^{-6} per $\mu g/m^3$). The CARB, 1992 estimate is not directly comparable to the official EPA estimate.[g]								
Exposure $(\mu g/m^3)$	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.

Pollutant	1990 Base Control	1995 Base Control	1995 Expanded Reform. Gasoline Use	2000 Base Control	2000 Expanded Reform. Gasoline Use	2000 Expanded Adoption Calif. Stds.	2010 Base Control	2010 Expanded Reform. Gasoline Use	2010 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.

[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 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

ES-12

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 $8.3{\times}10^{-6}(\mu g/m^3)^{-1}$ 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
µg/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

EPA-420-R-93-005
April 1993

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 µg/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
(2.2×10^6 µg/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 μg/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 μg/m^3. When compared to the HAPEM-MS 1990 base control level of 0.36 μg/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 μg/m^3 must be increased by a factor of 2.09, to 0.75 μg/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} (μg/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} [μg/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} [μg/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 μg/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 $1.2 \times 10^{-4}(\mu g$ organic matter/m$^3)^{-1}$ and $5.1 \times 10^{-5}(\mu g$ particulate matter/m$^3)^{-1}$. For the automobile without a catalyst using leaded fuel, the unit risk is $1.6 \times 10^{-5}(\mu g$ 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, <u>Alpha$_{2u}$-globulin: Association with Chemically Induced Renal Toxicity and Neoplasia in the Male Rat</u>, 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

3-3

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) <u>Baseline Gasoline Use (Federal Emission Standards)</u> -- 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) <u>Baseline Fuel Use (California Emission Standards)</u> -- 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) <u>Federal Reformulated Gasoline Program, Phase 1 (Federal Emission Standards) and California Reformulated Gasoline Program, Phase 1 (California Emission Standards)</u> -- 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 ($\leq$ 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) <u>Federal Reformulated Gasoline Program, Phase 2 (Federal Emission Standards)</u> -- 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) <u>Federal Reformulated Gasoline Program, Phase 2 (California Emission Standards)</u> -- 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) <u>Oxygenated Fuels CO Program, (Federal and California Emission Standards, 1995; Federal Emission Standards, 2000, 2010)</u> -- 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

3-9

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) <u>Oxygenated Gasoline CO Program (California Emission Standards, 2000, 2010)</u> -- 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) <u>California Reformulated Gasoline, Phase 2 (California Emission Standards)</u> -- 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) <u>Ethanol Fuel Use (Federal Emission Standards)</u> -- 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*

Components/Scenarios	1990 Base Control	1995 Base Control	1995 Expanded Reform. Gasoline Use	2000, 2010 Base Control	2000, 2010 Expanded Reform. Gasoline Use	2000, 2010 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) <u>2000, 2010 Base Control</u> -- 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) <u>2000, 2010 Expanded Use of Reformulated Gasoline</u> -- 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) <u>2000, 2010 Expanded Adoption of California Standards</u> --
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

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

4-7

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.

5.0 BENZENE

5.1 Chemical and Physical Properties (EPA, 1988)

Benzene is a clear, colorless, aromatic hydrocarbon which has a characteristic sickly, sweet odor. It is both volatile and flammable. Selected chemical and physical properties of benzene are presented in Table 5-1.

Benzene contains 92.3 percent carbon and 7.7 percent hydrogen with the chemical formula C_6H_6. The benzene molecule is represented by a hexagon formed by the six sets of carbon and hydrogen atoms bonded together with alternating single and double bonds. The benzene molecule is the cornerstone for aromatic compounds, most of which contain one or more benzene rings.

Benzene is nonpolar, meaning it carries no major area of charge in any portion of the molecule and no net electrical charge considering the molecule as a whole. It is relatively soluble in water and is capable of mixing with polar solvents (solvents which carry major portions of opposing charges within the molecule) such as chloroform, acetone, alcohol, and carbon tetrachloride without separating into two phases.

Benzene is a highly stable aromatic hydrocarbon, but it does react with other compounds primarily by substitution of a hydrogen atom. Some reactions occur which can rupture or cleave the molecule.

Table 5-1. Chemical and Physical Properties of Benzene.

Property	Value
Molecular weight	78.11 g/mole
Melting point	5.5°C (41.9°F)
Boiling point	80.1°C (176.2°F)
Density at 20°C (68°F)	0.879 g/ml
Vapor Pressure at 25°C (77°F)	0.13 atm.
Flash point (closed cup)	-11.1°C (12.02°F)
Solubility in water at 25°C	1.8 g/L
Conversions at 25°C	1 ppm = 3.25 mg/m^3 1 mg/liter = 313 ppm

5.2 Formation and Control Technology

Benzene is present in both exhaust and evaporative emissions. Data show the benzene level of gasoline to be about 1.5%, with diesel fuel containing relatively insignificant levels of benzene. Some exhaust benzene is unburned fuel benzene. Some work indicates that non-benzene aromatics in the fuels can cause about 70 to 80% of the exhaust benzene formed. Some benzene also forms from engine combustion of non-aromatic fuel hydrocarbons. The fraction of benzene in the exhaust varies depending on control technology and fuel composition but is generally about 3 to 5%. The fraction 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%. These data also show that diesel vehicles account for only about 3% of the total mobile source benzene emitted (Carey, 1987).

Control techniques are available and in use for both evaporative and exhaust emissions of benzene. For example, positive crankcase ventilation (PCV) and evaporative controls reduce evaporative emissions of benzene. Fuel evaporative controls were installed on all 1971 light-duty gasoline vehicles. An absorption/regeneration system, one of the most common evaporative control techniques, is a canister of activated carbon that traps vapors such as benzene. The vapors are ultimately fed back to the combustion chamber. Catalysts on automobiles have been effective in reducing benzene exhaust emissions. The amount of reduction achieved is dependent on the type of catalyst technology used and the drive cycle of the vehicle (EPA, 1988). It is also dependent on the exhaust hydrocarbon standard to which the vehicle has been certified.

Section 202(a)(6) of the Act states that the EPA shall promulgate standards for control of refueling emissions, after consultation with the Department of Transportation. EPA decided not to promulgate such standards in March of 1992 after questions were raised by the National Highway Traffic Safety Administration on the safety of the onboard carbon canisters. This decision was also based on information concerning the effectiveness of this technology to combat ozone. The EPA then issued guidance for vapor recovery technology, known as Stage 2, to be installed on gasoline pumps (EPA, 1992a). On January 22, 1993 a Federal appellate court directed EPA to promulgate standards requiring automakers to control refueling emissions for new cars and light-duty trucks.

5.3 Emissions

5.3.1 Emission Fractions Used in the MOBTOX Emissions Model

Benzene fractions were determined using a series of equations relating fuel properties to THC percent benzene in exhaust and evaporative emissions rather than the actual vehicle data in Appendix B2. However, actual vehicle data were used to

corroborate the accuracy of these equations. Please refer to
Appendix B2 for the emission fractions used in this section.

5.3.1.1 Benzene Exhaust Emission Fractions

For benzene exhaust from gasoline vehicles, separate
equations were used for three-way catalysts, three-way plus
oxidation catalysts, and other catalyst types. For vehicles with
a three-way catalyst, running on baseline gasoline, the following
equation was used:

3-way Bz%THC = 1.077 + 0.7732*(volume % benzene)
               + 0.0987*(volume % aromatics - volume % benzene).

This equation was obtained by the EPA Regulatory Development and
Support Division (RDSD) from work done by Chevron Oil Company
(Chevron 1991). An analogous equation for NMHC is being used by
RDSD in the Supplemental NPRM, on regulation of fuels and fuel
additives in reformulated and conventional gasoline (EPA, 1991a).
For vehicles with a three-way plus oxidation catalyst, running on
baseline gasoline, the equation used was:

3-way + ox Bz%THC = 0.6796*(volume % benzene)
                    + 0.0681*(volume % aromatics) - 0.3468.

This equation was obtained from the draft Regulatory Impact
Analysis for RVP regulations (EPA, 1987a). For vehicles with no
catalyst or an oxidation catalyst, the equation used was:

other Bz%THC = 0.8551*(volume % benzene)
               + 0.12198*(volume % aromatics) - 1.1626.

This equation was also given in the draft Regulatory Impact
Analysis for RVP regulations. The same benzene fractions were
used for HDGVs. Benzene fractions for LDDVs, LDDTs, and HDDVs
were based on the benzene fractions of THC used in the 1987 EPA
motor vehicle air toxics report (0.0240 for LDDVs and LDDTs;
0.0110 for HDDVs) (Carey, 1987). These were then adjusted to
give benzene fractions of TOG using the TOG/THC ratios given in
Table 3-7.

Next, it was necessary to determine whether an adjustment
factor should be applied to the gasoline vehicle equations for
MTBE and ethanol blends. To calculate an appropriate adjustment
factor, percent exhaust benzene for individual vehicles in
various studies was compared for baseline and oxygenated blends
(Appendix B4). The comparison between fuels was done on a
vehicle by vehicle basis because of the large amount of
individual variation in emissions among vehicles. If data for
different vehicles running on a fuel type are pooled and then
compared, it is difficult to isolate trends probably due to car
to car variations. Also, if data for different MTBE or ethanol
blends (with the different aromatic, olefin content, etc.) are
pooled, fuel effects may also make comparison difficult. This
comparison was performed for 15% MTBE and 10% ethanol. Then, an

average percent change (expressed as a fraction) was calculated
for each catalyst type. This average percent change was added to
1, representing the baseline emissions with gasoline, and the
equations were then multiplied by the resultant factor. Since
the average percent change was calculated for 15% MTBE, for
blends with other MTBE levels the average percent change was
multiplied by a ratio of percent MTBE to 15. Actual benzene TOG
fractions (from Appendix B2) were compared to predicted benzene
THC, with and without the adjustment factor (Appendix B5). No
significant difference was observed in the accuracy of the
equations, with and without the adjustment factor, with both
typically predicting TOG benzene levels within +/- 20%. Based on
these comparisons, the THC equations without adjustment factors
were used to determine benzene percent TOG fractions for MTBE and
ethanol blends, since these seemed to be just as accurate.

Once the appropriate equations for benzene were chosen, the
fuel properties (% aromatics, benzene, and oxygen) to use with
the equations were then determined. The resultant emission
fractions are contained in Appendix B6.

For reformulated gasoline in CY 2000+, the fraction of
exhaust benzene (and the other toxics mentioned in CAAA Section
219) is assumed to remain the same relative to CY 1995-1999.
However, the mass of TOG will be reduced as required by the CAAA.
As a result, the mass of benzene is assumed to be reduced
proportionately to TOG for exhaust.

As mentioned earlier, under the California standards, fuel
characteristics for oxygenates are similar to those under the
reformulated gasoline regulations. However, under Phase 2 of
CARB's reformulated fuel regulations, which go into effect in
1996, RVP will be limited to 7.0 psi. Since RVP has little
effect on benzene exhaust fractions, it was assumed that benzene
exhaust fractions under the California standards are the same as
under reformulated gasoline regulations.

5.3.1.2 Benzene Diurnal and Hot Soak Evaporative Emission
Fractions

For benzene evaporative emissions from gasoline vehicles,
two equations were used to determine fractions -- one for diurnal
emissions, and one for hot soak emissions. The equation used for
diurnal emissions from vehicles running on gasoline MTBE blends
was:

Diurnal Benzene = [(1.3758 - (0.0579*(weight % oxygen/2.0)
                    - 0.080274*RVP)]*(volume % benzene).

The equation used for hot soak emissions from vehicles running on
MTBE fuel was:

Hot Soak Benzene = [(1.4448 - (0.0684*(weight % oxygen/2.0)
                    - 0.080274*RVP)]*(volume % benzene).

To calculate diurnal and hot soak emissions from vehicles running
on gasohol, the oxygen term (which was developed specifically for
MTBE) was eliminated. The oxygen term used for MTBE fuel
accounts for test data which have shown that the presence of MTBE
tends to reduce benzene's evaporative and running loss benzene
emissions. However, test data with ethanol have not shown such
an effect on benzene emissions separate from its effect on
overall evaporative VOC emissions. Thus, the diurnal and hot
soak equations for gasohol (and gasoline) are:

Diurnal Benzene = [1.3758 - (0.080274*RVP)]*(volume % benzene)

Hot Soak Benzene = [1.4448 - (0.080274*RVP)]*(volume % benzene).

For both MTBE and gasohol, these equations were derived from GM's
tank vapor emissions model (1991) for representative tank
temperatures, and were used in RDSD's reformulated gasoline NPRM,
(EPA, 1991a), and in the supplemental NPRM (EPA, 1992b). The
supplemental NPRM states that this model was derived for vehicles
typical of in-use emissions rather than vehicles meeting the
emission standards. Once again, the same emission fractions were
used for HDGVs, LDGVs, and LDGTs. Evaporative emissions from
LDDVs, LDDTs, and HDDVs were assumed to be negligible.

     The accuracy of these equations was tested in predicting
evaporative benzene levels from fuel properties in baseline
gasoline, MTBE blends, and gasohol by comparing predicted benzene
levels to benzene levels from actual vehicle data (Appendix B5).
The equations underpredicted evaporative benzene emissions
significantly (e.g., % predicted versus % observed) for vehicles
with carburetors, and even more significantly for fuel injected
vehicles. This may be because the model that the equations were
based on was derived for "typical in-use" vehicles, and almost
all the vehicles in the database were vehicles with lower
evaporative emissions. The equations were used in these
analyses, in order to be consistent with the reformulated fuels
NPRM. In any case, evaporative benzene emissions are less than
20% of total vehicle benzene emissions so this underprediction is
not serious.

     Diurnal and hot soak benzene emission fractions for various
programs included in modeling components are included in Appendix
B6. It was also assumed that the fraction of benzene in overall
evaporative emissions remains the same, regardless of
temperature, since all MOBTOX runs were done at a single
temperature range (68°-84°). Benzene evaporative emissions are
small compared to exhaust benzene so using a single temperature
range versus explicitly setting evaporative emissions of benzene
equal to zero in winter months is probably justified. Higher
benzene exhaust emissions in winter months are not being
considered, so these approximations may cancel one another.

     For exhaust benzene emissions, RVP was not part of the
equations used to predict emission fractions. RVP does affect
evaporative emission fractions, however. For example, an RVP of

8.1 was assumed for federal reformulated fuels in CY 1995-1999 for Class C areas, but an RVP of 7.8 in CY 2000+. This results in slightly higher diurnal and hot soak benzene fractions for CY 2000+ compared to 1995-1999. The overall mass of evaporative benzene decreases, however, because the reduction in overall evaporative THC is greater at lower RVPs. Also, for California standards, the benzene exhaust fractions are assumed to be the same as those for EPA 1995-1999 reformulated gasoline standards. For the 1995 scenarios, the diurnal and hot soak benzene fractions came from EPA's reformulated gasoline regulations. However, since CARB's Phase II reformulated fuel regulations, taking effect in 1996, specify an RVP of 7.0, scenarios for 2000 and 2010 used different benzene diurnal and hot soak emission fractions, calculated using the different RVP value.

5.3.1.3 Benzene Running, Resting, and Refueling Loss Evaporative Emission Fractions

Running loss evaporative emission fractions for benzene were assumed to be the same as for hot soak. Resting loss emission fractions were assumed to be the same as for diurnal. Refueling loss benzene fractions were set at 0.01, following the VOC/PM Speciation Data System (EPA, 1990a).

5.3.2 Emission Factors for Baseline and Control Scenarios

The fleet average benzene emission factors as determined by the MOBTOX emissions model are presented in Table 5-2. When comparing the base control scenarios relative to 1990, the emission factor is reduced by 46% in 1995, by 60% in 2000, and by 68% in 2010. The expansion of reformulated fuel use in 1995 reduces the emission factor by another 7% relative to 1990. In 2000, the expanded control scenarios reduce the emission factor by another 6 to 9%, and in 2010, by another 4 to 6%, relative to 1990.

Table 5-2.   Annual Emission Factor Projections for Benzene.

Year-Scenario	Emission Factor g/mile	Percent Reduction from 1990
1990 Base Control	0.0882	–
1995 Base Control	0.0472	46
1995 Expanded Reformulated Fuel Use	0.0413	53
2000 Base Control	0.0351	60
2000 Expanded Reformulated Fuel Use	0.0301	66
2000 Expanded Adoption of California Standards	0.0305	65
2010 Base Control	0.0285	68
2010 Expanded Reformulated Fuel Use	0.0248	72
2010 Expanded Adoption of California Standards	0.0228	74

5.3.3 Nationwide Motor Vehicle Benzene Emissions

The nationwide benzene metric tons are presented in Table 5-3. Total metric tons are determined by multiplying the emission factor from Table 5-2 (g/mile) by the VMT determined for the particular year. The VMT, in billion miles, was determined to be 1793.07 for 1990, 2029.74 for 1995, 2269.25 for 2000, and 2771.30 for 2010. When comparing the base control scenarios relative to 1990, the metric tons are reduced by 39% in 1995, by 50% in 2000, and remains constant at 50% in 2010.

5.3.4 Other Sources of Benzene

Mobile sources account for approximately 85% of the total benzene emissions. Of the mobile source contribution, the majority comes from the exhaust. The remaining benzene emissions (15%) come from stationary sources. Many of these are related to industries producing benzene, sometimes as a side product, and those industries that use benzene to produce other chemicals. Coke ovens are responsible for 10% of the 15% with the other 5% attributable to all other stationary sources (Carey, 1987).

Approximately 70% of mobile source benzene emissions (60% of total benzene emissions) can be attributed to onroad motor vehicles, with the remainder attributed to nonroad mobile sources. This figure is based on a number of crude estimates and assumptions. First, it was estimated that 25% of total VOC emissions are from onroad vehicles, and 10% are from nonroad sources (based on a range of 7-13%). These estimates were obtained from EPA's Nonroad Engine and Vehicle Emissions Study (NEVES) (EPA, 1991b). Thus, about 70% of mobile source VOC is attributable to onroad vehicles. This VOC split was adjusted by onroad and nonroad benzene fractions (described below) to come up with the estimate of 70% of mobile source benzene from on-road vehicles.

For nonroad vehicles, benzene was estimated to be about 3.0% of exhaust hydrocarbon emissions and 1.7% of evaporative hydrocarbon emissions, based on the NEVES report (EPA, 1991b). The 1.7% evaporative emissions estimate is actually an estimate for refueling emissions of nonroad gasoline engines. Since no estimate existed for benzene evaporative emissions, it was assumed that percent benzene evaporative emissions was the same as refueling. The split between exhaust and evaporative benzene emissions was assumed to be 80% exhaust to 20% evaporative. Thus, the overall benzene fraction of nonroad hydrocarbon emissions was estimated to be 2.74%.

For onroad vehicles, benzene was estimated to be 3.89% of exhaust hydrocarbon and 1.04% of evaporative hydrocarbon emissions. The exhaust fraction is a 1990 fleet average toxic fraction, with fractions in Appendix B2 weighted using 1990 VMT fractions. The evaporative fraction is the benzene fraction given in Appendix B6

Table 5-3. Nationwide Metric Tons Projection for Benzene.

Year-Scenario	Emission Factor g/mile	Metric Tons
1990 Base Control	0.0882	158,149
1995 Base Control	0.0472	95,804
1995 Expanded Reformulated Fuel Use	0.0413	83,828
2000 Base Control	0.0351	79,651
2000 Expanded Reformulated Fuel Use	0.0301	68,304
2000 Expanded Adoption of California Standards	0.0305	69,212
2010 Base Control	0.0285	78,982
2010 Expanded Reformulated Fuel Use	0.0248	68,728
2010 Expanded Adoption of California Standards	0.0228	63,186

for gasoline-fueled vehicles. The split between exhaust and evaporative hydrocarbon emissions was estimated to be 60% exhaust to 40% evaporative. Thus, the overall benzene fraction for onroad hydrocarbon emissions was 2.74%. If the VOC split is adjusted by these benzene fractions for onroad and nonroad emissions, 70% of benzene from mobile sources is estimated to come from on road vehicles.

Data from EPA's Total Exposure Assessment Methodology (TEAM) Study identified the major sources of exposure to benzene for much of the U.S. population. The TEAM study is described in detail in a four-volume EPA publication (EPA, 1987b). The study measured 24-hour personal exposures in air and drinking water for 20 to 25 target volatile compounds for a selected group of subjects from six cities. Subjects were selected according to census information, socioeconomic factors, and their proximity to potential industrial and mobile sources. Large numbers of homes were visited by trained interviewers to collect information on age, sex, occupation, smoking status, and other factors for each person in the household. A total of 700 subjects representing more than 800,000 residents of the various cities were sampled.

The final results of TEAM total benzene exposure (Wallace, 1989), show the most important source of benzene exposure is active smoking of tobacco. Smoking accounts for about half of the total population exposure to benzene. Personal exposures due to riding in automobiles, passive smoking, and exposure to consumer products account for roughly one-quarter of the total exposure, with outdoor concentrations of benzene, due mainly to vehicle exhaust, accounting for the remaining portion. Occupational exposures, pumping gasoline, living near chemical plants or petroleum refining operations, food, water, and beverages appear to account for no more than a few percent of total nationwide exposure to benzene.

5.4 Atmospheric Reactivity and Residence Times

Laboratory evaluations indicate that benzene is minimally reactive in the atmosphere, compared to the reactivity of other hydrocarbons. This then gives benzene long-term stability in the atmosphere. Oxidation of benzene will occur only under extreme conditions, involving a catalyst or elevated temperature or pressure. Photolysis is possible only in the presence of sensitizers and is dependent on wavelength absorption.

The information that follows on transformation and residence times has been largely excerpted from a report produced by Systems Applications International for the EPA (Ligocki et al., 1991).

5.4.1 Atmospheric Transformation Processes

A variety of atmospheric transformation processes of importance to air toxics can occur in urban atmospheres. Species can be destroyed by reaction with atmospheric oxidants, or by photolysis. The oxidant of most importance on a global scale is the hydroxyl radical (OH), which is produced photolytically everywhere in the atmosphere and reacts with nearly every organic substance. In urban atmospheres, ozone (O_3) can also be an important oxidant. At night, OH concentrations drop off significantly because little OH is produced in the absence of sunlight, but concentrations of the nitrate radical (NO_3) can increase to fairly high levels when high concentrations of nitrogen oxides (NO_x) are present. Other atmospheric oxidants are the hydroperoxyl radical (HO_2), the oxygen atom, and the chlorine atom (Cl), which may be important under some circumstances. A few atmospheric species react directly with nitrogen dioxide (NO_2).

Photolysis refers to decomposition following absorption of ultraviolet radiation. While reaction with oxidants is common to virtually all organic molecules, photolysis usually involves oxygenated intermediates containing the carbonyl (C=O) bond, such as formaldehyde and acetaldehyde. (Whitten, 1983).

Many atmospheric species react rapidly in the aqueous phase of clouds, fogs, and aqueous aerosols. For highly soluble and highly reactive species, this can be a major atmospheric transformation pathway.

Atmospheric transformation can also include the condensation of gaseous species onto atmospheric aerosols. This process is a function of the vapor pressure of the species, the amount of aerosol present in the atmosphere, and the temperature. Although benzene, 1,3-butadiene, formaldehyde, and acetaldehyde exhibit sufficiently high vapor pressures that they will not condense onto aerosols to any significant degree, this process can be of major importance for other types of air toxics such as polycyclic organic matter associated with diesel and gasoline particulate.

5.4.2 Gas Phase Chemistry of Benzene

The aromatic ring structure of benzene is extremely stable and resistant to chemical attack. Therefore, of all the toxic species to be addressed in this report, benzene is the least reactive in the atmosphere. Not only does benzene oxidize slowly, but one of its key oxidation products, phenol, suppresses ozone formation under NO_x-limited conditions because it acts as a free radical scavenger.

5.4.2.1 Gas Phase Reactions

The only benzene reaction which is important in the lower atmosphere is reaction with the OH radical. Yet even this reaction is relatively slow. The reaction proceeds by OH addition, forming a complex which can decay back to the original reactants. At relevant tropospheric temperatures, this decay rate is negligible. The temperature dependence of this reaction is not well known. Benzene reacts more slowly with OH radicals than do most other aromatic species. Toluene and m-xylene react five times and 19 times as fast as benzene, respectively (Atkinson, 1990).

The reactions of benzene with oxygen atoms, ozone (O_3), and nitrate (NO_3) have been measured. Since the rate of these reactions are slower than rate of reaction of benzene with OH, and/or their concentrations in the atmosphere are generally much lower than OH concentrations, these reactions are not important in the atmospheric transformation of benzene.

Reactions with Cl atoms are known to be important in the stratosphere, where they are associated with the ozone depletion cycle. However, Cl concentrations in the troposphere are low, roughly three orders of magnitude smaller than OH concentrations (Singh and Kasting, 1988). Since the reaction rate is only a factor of ten larger than the OH rate, this reaction is not important in the lower atmosphere.

5.4.2.2 Reaction Products

The observed stable products from the atmospheric oxidation of benzene are phenols (phenol and nitrophenol), and aldehydes (mainly glyoxal [CHO]$_2$) with reported yields of 24 percent for phenol (Atkinson et al., 1989) and 21 percent for glyoxal (Tuazon et al., 1986). Nitrophenol yields of 3 percent at low NO_x concentrations have been reported (Atkinson, 1990). Thus, the known products do not completely account for all the mass reacted. Phenol is highly reactive under smog conditions and will react rapidly with OH radicals during the daytime and with NO_3 radicals at nighttime. Glyoxal is also highly reactive, with a chemistry similar to that of formaldehyde. Both phenol and glyoxal, besides being highly reactive, are also highly water-soluble, and will be removed rapidly by incorporation into clouds or rain.

5.4.3 Aqueous Phase Chemistry of Benzene

Benzene reacts rapidly in aqueous solution with the OH radical and the sulfate radical (SO_4^-), forming products that are removed by their incorporation into rain. Despite the rapid reaction of benzene in aqueous solution, its low solubility limits the importance of aqueous-phase processes for this compound and it will not be incorporated into clouds or rain to any large degree.

5.4.4 Atmospheric Residence Times

5.4.4.1 Definition and Limitations

In assessing the potential impact of emissions of toxic species into the atmosphere, it is important to have some measure of their atmospheric persistence. Species which persist for long periods of time can accumulate to high concentrations during stagnation periods and can be transported further from their sources than species which are destroyed rapidly. Common measures of atmospheric persistence are the residence time, or lifetime (τ), and the half-life, both of which are measures of the time required for a fixed concentration of a species to decay to a certain percentage of its initial concentration. The residence time and the half-life are times at which the concentration has been reduced to 37% and 50% of its original value, respectively. The atmospheric residence time is thus a mathematical formulation which provides a common ground for comparison of the persistence of different chemical species.

One limitation of residence time calculations is that they cannot be used to predict ambient concentrations of toxic species. Concentrations are determined by atmospheric dispersion characteristics combined with emissions patterns, formation, and removal rates. In urban areas, the effective residence time of toxic species in the atmosphere may be determined by the time required to transport emissions out of the air basin, rather than the time required for their chemical or physical removal within the air basin. Also, residence time calculations do not incorporate chemical production rates for secondary species. Thus, residence time calculations may indicate that a species such as formaldehyde is removed rapidly during the daytime, when actually formaldehyde is being produced more rapidly than it is being removed. Finally, residence time calculations consider atmospheric reactions as destruction processes and do not consider the possible transformation of toxic species into equally toxic products.

Despite these limitations, atmospheric residence time calculations can be valuable when viewed in context with these other issues.

5.4.4.2 Chemical and Physical Processes

A variety of chemical and physical processes must be taken into consideration when determining the residence time of a compound. Chemical processes include gas-phase chemical reactions, photolysis, and in-cloud chemical destruction. Physical processes include wet and dry deposition. With regard to gas-phase chemical reactions, typical atmospheric oxidant concentrations are required for residence time calculations. Concentrations of OH radicals are of particular importance, since chemical residence times for many atmospheric species are determined by their rate of reaction with the OH radical. At night, photolysis is absent and OH radical concentrations are

very low. Other chemical reactions, such as reaction with NO_3 radical or O_3, may be important at night.

For species which photolyze, photolysis can compete with the OH reaction as the dominant daytime removal mechanism. Photolysis rates depend only upon the amount of ultraviolet (UV) radiation reaching the lower troposphere, and thus can be determined on the basis of latitude, altitude, and time of year.

Cloud cover is often neglected in atmospheric residence time calculations. Yet, many areas of the United States experience a significant degree of cloud cover throughout much of the year. Cloud cover affects the residence time of atmospheric pollutants in two major ways. First, clouds attenuate the solar UV radiation at ground level, slowing photolysis rates and decreasing radical concentrations. Second, clouds are themselves a reactive medium in which chemical transformation will take place. Therefore, the presence of clouds may increase or decrease the atmospheric residence time of specific pollutants.

The physical processes of wet and dry deposition can also be significant removal routes for some atmospheric pollutants. Wet deposition refers to the capture and removal of species by hydrometers including rain, snow, hail, etc. Dry deposition refers to the loss of atmospheric species to surfaces by diffusion, sedimentation, impaction, etc. The atmospheric residence time due to physical processes depends upon whether the species is present in the atmosphere only as a vapor, or partially adsorbed to particles. This partitioning is determined by the vapor pressure of the species. For calculation purposes, all precipitation was assumed to be in the form of rain, since partitioning of organic compounds from the atmosphere to snow or other forms of frozen precipitation is less well understood.

The rate of dry deposition of volatile organic compounds is highly uncertain. A method proposed for incorporation into regional air quality models was used to calculate dry deposition rates, although its validity has not been demonstrated for organic species.

For species which are present in the atmosphere as gases or vapors, deposition processes may be reversible. For instance, volatile compounds present in rain which falls on a surface such as a street or sidewalk and subsequently evaporates will return to the atmosphere. It has been proposed that formaldehyde rapidly deposits to dew-covered surfaces overnight and in the early morning, and then is released when the dew evaporates at mid-morning (Ireson et al., 1990). To the extent possible, these types of reversible processes should not be considered in atmospheric residence time calculations.

5.4.4.3 Generation of Input Values

The oxidant concentrations required for the residence time calculations were obtained from trajectory model simulations for the four cities, Los Angeles, St. Louis, New York, and Atlanta.

These locations were chosen to represent a variety of regions within the United States, and were also chosen because summer model input data were available for these cities. The simulations were conducted using the Ozone Isopleth Plotting Model, Version 4 with Carbon Bond Mechanism IV (OZIPM-4) (Hogo and Gery, 1988). This is a model which is used routinely to predict ozone formation as a function of VOC and NO_x emissions; however, as an intermediate step, it calculates radical concentration such as OH.

Simulations began at 9 a.m. and continued through 4 a.m. the following day. The calculations were conducted for daytime and nighttime, and then weighted by the length of the day and night to obtain 24-hour averages. Because these simulations were for severe ozone episodes, the oxidant concentrations generated may be somewhat larger than seasonal average values.

For each city, calculations were conducted for both the summer (July) and winter (January) seasons. For each season, residence time calculations were also conducted for clear-sky and cloudy conditions.

The winter simulations used the same summer input files except for the following: (1) the time zone was increased 1 hour to convert to standard time, (2) the temperatures were changed to start at the average winter low and smoothly reach the average winter high at about 1400 hours, (3) the date was set to 15 January, and (4) the mixing height maximum was adjusted downward. Each of the residence time calculations was conducted for clear-sky conditions and cloudy conditions. Cloudy conditions take into account the UV transmission factor, the in-cloud OH concentration, the gas-phase oxidant concentrations, and the cloud liquid water content.

The residence times are most useful for comparison purposes rather than as absolute numbers, because of the necessary assumptions and simplifications which went into the calculations. More details regarding the model input files and parameters used in calculating residence times, such as oxidant concentrations and rates of reaction, are given in Ligocki et al., 1991.

5.4.4.4 Benzene Residence Times

Residence times for benzene were calculated by considering gas phase chemical reactions with OH and NO_3, in-cloud chemical reaction with OH, and wet and dry deposition. The results of the residence time calculation for benzene are presented in Table 5-4.

TABLE 5-4. Atmospheric residence time calculation for benzene. All times are in hours unless otherwise noted.

	Los Angeles		St. Louis		Atlanta		New York	
	July	Jan	July	Jan	July	Jan	July	Jan
Clear sky – day	40	300	30	500	30	500	50	900
Clear sky – night	3000	14000	4000	18000	3000	14000	4000	18000
Clear sky – avg	70	700 (30 d)	50	1100 (46 d)	50	1100 (45 d)	90	2200 (92 d)
Cloudy – day	80	600	60	800	50	800	100	1600
Cloudy – night	800	7000	900	8000	300	7000	1500	12000
Cloudy – avg	120	1300 (56 d)	90	1800 (75 d)	80	1700 (71 d)	150 (6 d)	3600 (150 d)
Monthly Climatological Average	80	900 (37 d)	70	1500 (62 d)	60	1400 (58 d)	110	2900 (120 d)

Calculated residence times ranged from 2 days under summer, clear-sky conditions, to several months under winter, cloudy-sky conditions. These values can be compared to estimated benzene half-lives of 4 days under summer, urban conditions (CARB, 1984) and 6 days under summer conditions at 60°N latitude (Nielsen et al., 1983).

The main atmospheric destruction pathway for benzene is the reaction with OH radical. Even at night, the residence time of benzene was found to be determined primarily by the reaction with OH, with a slight contribution from in-cloud destruction. The reaction with NO_3 was found to be unimportant for benzene.

As discussed above, estimates of residence times due to dry deposition should be regarded as highly uncertain. The residence times of benzene due to dry deposition are estimated to be on the order of 20 days for summer, daytime conditions and one year or more for all other conditions.

In-cloud chemical destruction and wet deposition will not be rapid removal processes for benzene. The residence times due to in-cloud chemistry ranged from 11 days in the summer to over 2 years in the winter. The calculated residence times due to wet removal ranged from 3 years in the winter to 10 years in the summer.

Residence times for different cities within a given season varied by factors of 2-3. A much larger effect was predicted for the difference between summer and winter conditions at all sites, with winter residence times 10-30 times greater than summer residence times.

The major uncertainties in these calculations for benzene are the OH radical concentrations, which vary from day to day by roughly a factor of two. The uncertainty in the OH rate constant is much smaller than this (about 20 percent). Although the uncertainty in the deposition velocity is much larger than a factor of two, it does not have a large effect on the overall uncertainty because dry deposition is only of minor importance as a removal mechanism.

These results suggest that, on an urban scale, atmospheric transformation of benzene would not be expected to be a significant determinant of ambient benzene concentrations. Under all conditions examined, the calculated residence time of benzene was greater than one day. Therefore, significant day-to-day carryover of benzene concentrations would be expected.

5.4.5 Limited Urban Airshed Modeling of Air Toxics

Much of the information below on the Urban Airshed Model and the benzene results are excerpted from reports conducted for two EPA offices (Office of Mobile Sources and Office of Policy, Planning, and Evaluation) by Systems Applications International

(SAI) (Ligocki et al., 1991, Ligocki and Whitten, 1991, Ligocki et al., 1992). The modified version of the UAM used in these reports, with explicit treatment of several toxics, will be referred to as UAM-Tox. UAM-Tox in Ligocki et al. (1991) and Ligocki and Whitten (1991) which was used to model St. Louis, did not include explicit chemistry for acetaldehyde and POM. UAM-Tox in Ligocki et al. (1992) which was used to model the Baltimore-Washington area and Houston, does treat these toxics explicitly, however. Details of inputs and modifications for the UAM are presented in the above references. The treatment of each toxic in UAM-Tox is discussed in the results section for each toxic.

The Urban Airshed Model (UAM) is a three-dimensional grid model designed to simulate all important physical and chemical processes which occur in the atmosphere. In a grid model, the region of interest (domain) is divided into grid cells which are equally spaced in the horizontal directions, and may have varying heights depending upon the atmospheric mixed-layer height. Within each grid cell, concentrations are assumed to be uniform, and any emissions which are injected into that cell will instantaneously spread throughout the cell. The model incorporates mathematical representations of the processes of transport, diffusion, chemical reaction, and deposition. Based upon inputs such as emissions, winds, mixing heights, initial concentrations of each species, and concentrations of each species on the boundaries of the domain, the model computes concentrations for each species for each grid cell for each hour of the simulation.

The UAM has been used primarily for the simulation of ozone and the development of control strategies for ozone precursors. It has been evaluated in terms of its ability to predict concentrations of ozone and a few other species such as NO_x and peroxyacetyl nitrate (PAN). The UAM has not been evaluated for the prediction of concentrations of air toxics, and such an evaluation was beyond the scope of the study summarized here. Until such an evaluation is conducted, the model results are most useful for the comparisons they provide of the importance of atmospheric transformation.

To illustrate the effects of atmospheric persistence and transformation on ambient concentrations in an urban area, an initial urban airshed modeling study of benzene, 1,3-butadiene, formaldehyde, and acetaldehyde was conducted for a hypothetical day in the summer of 1990 in the St. Louis area (Ligocki et al., 1991; Ligocki and Whitten, 1991). A summer day was selected in order to maximize the potential effects of atmospheric transformation. The St. Louis urban area was selected primarily because the necessary model inputs were readily available; however, St. Louis is also of interest because relatively high benzene concentrations have been measured there (McAllister et al., 1990). Only one city was modeled due to resource constraints. Understanding how the calculated results may vary in different cities with different emissions and air quality patterns would help address some of the uncertainty in the

results. Subsequently, additional urban airshed modeling was done for multi-day episodes in the Baltimore-Washington area and Houston, as part of another study (Ligocki et al., 1992). Both of these areas are severe ozone nonattainment areas, and will participate in the federal reformulated gasoline program. Modeling was conducted for hypothetical episodes in 1995 and 1999, and took into account provisions of the CAA. Since toxics provisions of the reformulated gasoline program are year round, a winter episode was simulated for Baltimore. The Baltimore and Houston areas represent opposite ends of the spectrum in terms of expected air quality benefits of reformulated gasoline.

The St. Louis episode selected for the initial study was an historical episode from July 13, 1976. The meteorological and air quality inputs for that episode were originally developed for the EPA as part of the St. Louis Ozone Modeling Project (Schere and Sheffler, 1982; Cole et al., 1983). This episode also was modeled by SAI as part of the EPA Five Cities Study (Morris et al., 1989). Levels of air pollutants have declined significantly in most cities over the past 15 years. Although the available inputs for this simulation were for a 1976 episode, it was judged to be more useful to conduct the simulation for current conditions. Therefore, the emission inventory was updated to a summer weekday in 1990. The episode represents a hypothetical day in 1990 in which the dispersion characteristics correspond to an actual day in 1976. Details of other inputs and modifications for the UAM are presented in detail in Ligocki et al. (1991) and Ligocki and Whitten (1991). The treatment of each toxic in the UAM is discussed in the results sections for each toxic.

For modeling in the Baltimore-Washington area, the episode selected was an historical episode from July 5-7, 1988. The July 5-7 episode is part of a larger, regional-scale ozone episode that has been modeled with the Regional Oxidant Model (Possiel et al., 1990). A number of simulations were conducted for this episode in the base year of 1988, 1995, and 1999. Base, federal reformulated gasoline, California phase 2 reformulated gasoline and reduced motor vehicle NO_x simulations were conducted. Simulations were also done for both summer and winter, and with motor vehicles removed. For modeling in the Houston area, the episode selected was an historical episode from September 3-5, 1987. Simulations for summer were conducted for this episode in the base year of 1987, and for base case, reformulated gasoline, and no motor vehicle scenarios in 1995.

5.4.5.1 General Results From the UAM Simulations

Two base-case UAM simulations were conducted for the St. Louis study. The simulations used identical input parameters, but in one of them all chemistry was "turned off" assuming the toxic species of concern to be inert. The second simulation assumes all "chemistry on", referred to as the reactive simulation. The UAM simulations began at 1 a.m. local daylight time and ran through 11 p.m.

Results are presented as time-series plots of concentration at a specific grid cell. The time-series plots are presented in Appendix D and include predicted total concentrations of each toxic from both the reactive and inert simulations, and also include concentrations of the mobile- and stationary-source components from the reactive simulation. All values presented in the time series plots are hourly averages.

The simulations indicated that summertime concentrations of primary toxic species derived from mobile sources will be greatest during morning commute hours, when emissions are maximized, atmospheric dispersion is poor, and photochemistry is slow. The afternoon commute hours are less likely to produce peaks in mobile-source toxics in the summertime because they occur while mixing heights are higher and photochemistry is at its peak.

The Baltimore-Washington area and Houston simulations also indicated that concentrations of primary toxics species will be greatest during morning commute hours.

Federal reformulated gasoline simulations for 1995 and 1999 in the Baltimore-Washington area indicated a decrease in peak ozone of 0.2 pphm in 1995 (1.1% of total) and 0.15 pphm in 1999 (0.85% of total). This decrease corresponded to 20% of the peak ozone attributed to motor vehicles. For Houston, federal reformulated gasoline usage produced smaller ozone benefits, with a decrease in peak ozone of 0.013 pphm in 1995 (0.04% of total). This decrease corresponds to only 2% of the peak ozone attributable to motor vehicles. In both the Baltimore-Washington area and Houston, use of federal reformulated gasoline resulted in reductions of ambient benzene, acetaldehyde, and POM concentrations. For butadiene, there was virtually no effect on ambient concentrations. For formaldehyde, there were both decreases and increases, depending on the simulation.

The combination of the UAM results with results from the residence time calculations provides an estimate of the differences in concentrations which might be expected under wintertime conditions. Differences in emission rates and atmospheric dispersion parameters will also be important factors in determining wintertime concentrations. A comparison of summer and winter simulations in Baltimore indicated that, although benzene emissions from motor vehicles were lower in winter than in summer, motor vehicle-related concentrations of benzene were higher. Even so, the motor vehicle fraction of the simulated concentrations was

similar in winter, due to an increase in stationary source concentrations.

5.4.5.2 UAM Results for Benzene

Benzene was treated explicitly in the UAM-Tox. Mobile and stationary emissions of benzene were tagged separately and carried through simulations separately in the model. The gas phase reactions discussed previously were added to the chemistry subroutines. Because the focus of the study was on destruction of the toxic species rather than on the subsequent chemistry of their reaction products, no products were included in the UAM modifications for benzene.

St. Louis Simulation

A time-series plot of predicted benzene concentrations in St. Louis at the grid cell with the largest mobile-source benzene concentration is presented in Figure D-1 of Appendix D. At the time of the mobile-source benzene concentration peak, mobile-source benzene contributed roughly half of the total benzene concentration of 0.54 ppb. As the day progressed, the mobile-source benzene concentration decreased, while the total benzene increased to a peak of 0.7 ppb at 11 a.m. There was no evidence of a peak in the mobile-source concentration during the afternoon commute, probably due to the fact that the mixing height during the afternoon commute was still roughly 1500 m, compared to 400 m in the morning. Thus all emissions would be diluted into a much larger air volume in the afternoon.

The low reactivity of benzene is apparent from the comparison of the "inert" benzene and total benzene curves in Figure D-1. There is no difference between the two curves until mid-morning, and even in the mid-afternoon the difference between the two curves is less than 0.1 ppb. Thus, 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. This illustrates the conclusion drawn from the residence time calculations, that atmospheric chemical transformation of benzene in a urban environment is less important than location of sources and atmospheric dispersion characteristics in the assessment of benzene concentrations. Little seasonal effect would be expected for benzene.

The benzene concentration at the end of the simulation was 0.7 ppb (Figure D-1). Because benzene is not destroyed chemically at night (Table 5-2), in the absence of strong winds this concentration would be expected to persist into the following day. Therefore, the initial concentration of benzene of 0.1 ppb used for this simulation is likely to be too low. Future benzene simulations should be conducted for multiple days in order to

quantify the importance of day-to-day carryover of benzene concentrations.

The effect of initial concentration assumptions for benzene was examined in a sensitivity study in which the concentration fields from the end of the base-case simulation were used as the initial concentrations. This has the effect of increasing the initial concentrations of benzene. The peak concentrations within the city do not increase substantially from their base-case values. The afternoon maximum concentration only increases by 0.1 ppb. This result indicates that the meteorology of the simulated episode was such that concentrations were dominated by local emissions. For other episodes and other locations, more stagnant conditions might exist, and the importance of the initial concentrations might be greater.

When a comparison of simulated concentrations of benzene is made with ambient measured concentrations, the simulated concentrations were much lower than typical measured concentrations. This discrepancy may be due to uncertainties in the emission inventory for benzene. Another possibility is that the ambient monitors were located in areas not represented well in the UAM. The American Petroleum Institute (API) has stated that these differences may also be due to the fact that the UAM is not able to predict the concentrations and residence times of reactive air toxics well, and concentrations of the more reactive compounds show better agreement due to compensating errors in the model (API, 1991). For a full accounting of API's analysis please consult API, 1991.

Houston and Baltimore-Washington Area Simulations

Simulations for the summer Baltimore-Washington area episode resulted in significant decreases in ambient levels of benzene with use of federal reformulated gasoline, amounting to as much as 12 percent of ambient benzene concentrations. Use of California reformulated gasoline resulted in slightly larger decreases in ambient benzene. Maximum daily average benzene concentration for the 1988 base scenario was 2.2 ppb. Motor-vehicle related benzene accounted for about 58% of total benzene emissions. This agrees with the 60% estimate obtained in Section 5.3.4 for motor vehicles.

The summer Baltimore-Washington area simulations do not significantly underpredict benzene like the St. Louis simulation. In fact, simulated benzene concentrations were in good agreement with the average measured values from the UATMP data base. Ligocki et al. attribute this to an effort made to improve the emission mass fractions in the motor vehicle, area, and point source speciation profiles.

In the winter 1988 base scenario, the maximum daily average benzene concentration was 3.6 ppb, about 40 percent higher than in summer. Motor-vehicle related benzene accounted for about 37% of total benzene emissions. Simulations for the winter Baltimore-Washington area episode resulted in significant

decreases in ambient levels of benzene with use of reformulated gasoline, on the order of 7 percent. Motor vehicle benzene emissions were about 30 percent lower with reformulated gasoline use, and comprised a smaller fraction of total benzene emissions. However, the motor vehicle-related concentration of ambient benzene would be higher in winter, due to less atmospheric transformation. Comparison of simulated concentrations with measured concentrations indicate that the model may underpredict winter benzene concentrations.

For the summer 1987 base scenario in Houston, the maximum daily average benzene concentration was 41.4 ppb. Motor-vehicle related benzene accounted for about 21% of total benzene emissions. The maximum motor vehicle contribution to ambient benzene was 25%, based on the 1995 no motor vehicle scenario. Thus, motor vehicle-related benzene contributed less to overall ambient benzene in Houston than in Baltimore. 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, since in Houston maximum daily average concentrations are primarily influenced by point sources due to many large industrial facilities. However, for the entire Houston modeling domain, the maximum decrease in daily average concentration was about 8 percent. Comparison of simulated concentrations with measured concentrations suggest the model accurately predicts benzene concentrations.

5.5 Exposure Estimation

5.5.1 Annual Average Exposure Using HAPEM-MS

The data presented in Table 5-5 represent the results determined by the HAPEM-MS modeling that was described previously in Section 4.1.1. These numbers have been adjusted to represent the increase in VMT expected in future years.

The HAPEM-MS exposure estimates in Table 5-5 represent the 50th percentiles of the population distributions of exposure, i.e., half the population will be above and half below these values. High end exposures can also be estimated by using the 95th percentile of the distributions. According to the HAPEM-MS sample output for benzene, the 95th percentile is 1.8 times higher than the 50th percentile for urban areas, and 1.2 times high for rural areas. Applying these factors to the exposure estimates in Table 5.5, the 95th percentiles for urban areas range from 1.69 $\mu g/m^3$ for the 2010 expanded adoption of the California standards scenario to 4.81 $\mu g/m^3$ for the 1990 base control scenario. The 95th percentiles for rural areas range from 0.61 to 1.74 $\mu g/m^3$, respectively.

Table 5-5. Annual Average HAPEM-MS Exposure Projections for Benzene.

Year-Scenario	Exposure ($\mu g/m^3$)		
	Urban	Rural	Nationwide
1990 Base Control	2.67	1.45	2.36
1995 Base Control	1.56	0.84	1.40
1995 Expanded Reformulated Fuel Use	1.37	0.74	1.20
2000 Base Control	1.25	0.68	1.10
2000 Expanded Reformulated Fuel Use	1.08	0.58	0.98
2000 Expanded Adoption of California Standards	1.10	0.59	0.98
2010 Base Control	1.18	0.64	1.05
2010 Expanded Reformulated Fuel Use	1.04	0.56	0.93
2010 Expanded Adoption of California Standards	0.94	0.51	0.84

5.5.2 Comparison of HAPEM-MS Exposures to Ambient Monitoring Data

As stated in section 4.1.2, four national air monitoring programs/databases contain data on benzene. The Aerometric Information Retrieval System (AIRS), the Toxic Air Monitoring System (TAMS), the Urban Air Toxic Monitoring Program (UATMP), and
the National Ambient Volatile Organic Compounds Data Base (NAVOC) all have a significant amount of data for benzene. The urban exposure data for benzene from all four databases is summarized in Table 5-6. The AIRS data base contains data on benzene from 1987 to 1989 (EPA, 1989). The location and number of the sites varies between years. Referring back to Table 4-2 in Section 4.1.2 and to Table C-1 in Appendix C, 23 sites monitored benzene in 1987, 36 in 1988, and 13 in 1989. The cities where monitoring sites are located are listed below.

Birmingham, AL	St. Louis, MO
Oakland, CA	Louisville, KY
Fresno, CA	Atlanta, GA
Bakersfield, CA	Chicago, IL
Los Angeles, CA	Baton Rouge, LA
Merced, CA	Lowell, MA
Riverside, CA	Boston, MA
Sacramento, CA	Detroit, MI
San Bernadino, CA	Port Huron, MI
San Diego, CA	Dearborn, MI
San Francisco, CA	Lansing/E. Lansing, MI
Stockton, CA	New York, NY
Santa Barbara, CA	Cleveland, OH
San Jose, CA	Dallas, TX
Modesto, CA	Houston, TX
Oxnard, CA	Deer Park, TX
Miami, FL	Burlington, VT
Jacksonville, FL	Tacoma, WA

The average level of benzene (averaged equally by the number of sites) was 6.92 µg/m^3 (2.13 ppb) in 1987, 4.13 µg/m^3 (1.27 ppb) in 1988, and 4.16 µg/m^3 (1.28 ppb) in 1989. Because the number of sites differs from year to year and the number of samples taken at the various sites varies greatly, it is misleading to directly compare these numbers. However, these numbers do provide a general idea of the amount of benzene being emitted.

Looking at the data on an individual site basis, St. Louis had the highest level of benzene, 31.0 µg/m^3 (9.54 ppb) in 1987 at an industrial suburban site. However, only 5 samples were collected at that site in 1987. The lowest level of benzene was found in Boston, 2.50 µg/m^3 (0.77 ppb) in 1987 at an industrial urban site in the downtown area; however, only 4 samples were collected. In 1988, a commercial urban downtown site in Cleveland had the highest local average of all the sites monitoring benzene, 11.25 µg/m^3 (3.46 ppb) with 4 samples collected. Two commercial suburban sites

Table 5-6. Air Monitoring Results for Benzene.

Program	Years	Ambient Data[a] µg/m^3	Estimated Motor Vehicle Contribution[b] µg/m^3
AIRS	1989	4.16	2.50
	1988	4.13	2.48
	1987	6.92	4.15
UATMP	1989	6.37	3.82
	1990	4.78	2.87
TAMS	1987–89	4.26	2.55
NAVOC	1987	7.18	4.31

[a]Caution should be taken in comparing these numbers. The methods of averaging the data are not consistent between air monitoring databases and the sampling methodology is also inconsistent.

[b]The ambient data are adjusted to represent the motor vehicle contribution to the ambient concentration, which for benzene is estimated to be 60%, based on emissions inventory apportionment.

in Jacksonville, Florida had the lowest sample levels of benzene, both 1.82 µg/m^3 (0.56 ppb) with 17 and 5 samples collected. A residential suburban site in Houston had the highest average levels of benzene, 6.34 µg/m^3 (1.95 ppb) in 1989 with 20 samples collected. Also in 1989, Lowell, Massachusetts had the lowest average benzene level at a residential suburban site, 2.28 µg/m^3 (0.70 ppb), with 17 samples collected.

Referring to Table 4.2 and Table C-2, ten sites in the Toxic Air Monitoring System (TAMS) collected samples of benzene between 1987 and 1989. Boston, Chicago, and Houston each had three sites and Seattle/Tacoma had one. Averaged together, these sites had a benzene level of 4.26 µg/m^3 (1.31 ppb). The highest local average level of benzene was at an urban industrial area in Houston, 6.66 µg/m^3 (2.05 ppb). The lowest average local level of benzene was found at an industrial area in Tacoma, 2.02 µg/m^3 (0.62 ppb). It should be noted that Tacoma was added as a site in TAMS later than the other sites. Therefore, data were collected for benzene starting in 1988 instead of 1987. As stated in Section 4.1.2, TAMS is a subset of AIRS and so it has a limited number of sites.

In the 1989 Urban Air Toxics Monitoring Program (UATMP), 397 measurements of benzene were taken at 14 sites. These sites were in the cities listed below.

Baton Rouge, LA	Chicago, IL
Camden, NJ	Dallas, TX
Fort Lauderdale, FL	Houston, TX
Miami, FL	Pensacola, FL
St. Louis, MO	New Sauget, IL
Washington, D.C.	Wichita, KS

The highest average was 12.9 µg/m^3 (3.97 ppb) at an urban commercial site in downtown St. Louis, Missouri. Thirty samples were collected at this site. The lowest average was 1.95 µg/m^3 (0.60 ppb) at a suburban industrial site in Pensacola, Florida. Only seven samples were collected at this site. The next lowest average was 2.99 µg/m^3 (0.92 ppb) at a urban commercial site in Dallas, Texas. Twenty-five samples were collected at this site, providing a statistically more valid average. The overall average of the averages for each site was 6.37 µg/m^3 (1.96 ppb). For more detailed information on UATMP, please refer to Table C-3.

In the 1990 Urban Air Toxics Monitoring Program (UATMP), 349 measurements of benzene were taken at 12 sites. These sites were in the cities listed below.

                    Baton Rouge, LA   Chicago, IL
                    Camden, NJ   Houston, TX
                    Orlando, FL   Pensacola, FL
                    Port Neches, TX   Sauget, IL
                    Toledo, OH   Washington, D.C.
                    Wichita, KS

The highest average was 8.74 µg/m^3 (2.69 ppb) at an suburban
residential site in Houston, Texas. Twenty-eight samples were
collected at this site. The lowest average was 2.73 µg/m^3 (0.84
ppb) at a suburban residential site in Toledo, Ohio. Twenty-one
samples were collected at this site. The overall average of the
averages for each site was 4.78 µg/m^3 (1.47 ppb).

     In the National Ambient Volatile Organic Compounds (NAVOC)
program, 564 measurements of benzene were taken in 31 cities.
These cities are listed below.

                    Bakersfield, CA   Chula Vista, CA
                    Citrus Heights, CA   Concord, CA
                    El Cajon, CA   El Monte, CA
                    Fremont, CA   Fresno, CA
                    Long Beach, CA   Los Angeles, CA
                    Merced, CA   Modesto, CA
                    Richmond, CA   Rubidoux, CA
                    San Francisco, CA   San Jose, CA
                    Santa Barbara, CA   Simi Valley, CA
                    Stockton, CA   Upland, CA
                    Philadelphia, PA   San Leandro, CA
                    Livermore, CA   San Rafael, CA
                    Napa, CA   Vallejo, CA
                    Santa Rosa, CA   Redwood City, CA
                    Mountain View, CA   Oakland, CA
                    Baton Rouge, LA

The highest measurement was 11.7 µg/m^3 (3.60 ppb), which was
found at an urban site in San Francisco. However, this was only
one sample instead of an average. The highest average was 11.47
µg/m^3 (3.53 ppb), which was found at an urban site in Fresno and
consisted of 11 samples. The lowest measurement of benzene was
2.60 µg/m^3 (0.80 ppb), which was found at an urban site in
Oakland. Once again, this was only one measurement instead of an
average of multiple measurements. The lowest average was 3.51
µg/m^3 (1.08 ppb), which was found at an urban site in Livermore,
California, and consisted of 8 samples. The overall average of
the averages from the 31 cities was 7.18 µg/m^3 (2.21 ppb). For
more detailed data, please refer to Table C-4.

     The premise of the HAPEM-MS model is that the dispersion and
atmospheric chemistry of benzene is similar to CO. The average
atmospheric lifetime of CO ranges from one to four months (EPA,
1990b). Since both benzene and CO have long atmospheric
lifetimes, the HAPEM-MS model should be a reliable indicator of
benzene exposure from motor vehicles. To test the reasonableness
of the HAPEM-MS modeling results, the HAPEM-MS results for 1990

are compared to ambient monitoring results for recent years. Before comparing the HAPEM-MS results to the ambient data, the ambient monitoring data should be adjusted in two ways. First, the ambient monitoring data should be adjusted to represent the amount that is attributed to motor vehicles. The data derived from emission inventories estimate that 60% of the ambient benzene can be apportioned to motor vehicles. The numbers in the second column of Table 5.6 are 60% of the ambient levels and thus represent estimated ambient motor vehicle levels.

Second, the estimated ambient motor vehicle level should be adjusted to account for integrated exposure, i.e., time spent indoors and in various microenvironments. Pezda et al. (1991) refer to data collected in California (Robinson et al., 1989), which indicate that people spend 5.9% of their time outdoors, 61.9% indoors at home, 24.6% at work, and 7.6% during some form of transportation (car, bus, train, etc.). Using these activity patterns, it is next necessary to estimate how much of the ambient mobile source level people in these microenvironments are exposed to. HAPEM-MS provides the following microenvironment factors: indoors residence - 0.495; indoors other - 0.619; outdoors - 0.758; and inside motor vehicle - 1.554. These microenvironment factors are based on correlations between CO measured by personal exposure monitors and CO measured by fixed site monitors located within 10 km.

Combining the activity patterns and microenvironment factors, an adjustment factor to the ambient motor vehicle level to account for integrated motor vehicle exposure is calculated as shown below:

$$[(0.059)(0.758)+(0.619)(0.495)+(0.246)(0.619)+(0.076)(1.554)] = 0.622$$

The ambient motor vehicle level ranges from 2.48 to 4.31 $\mu g/m^3$. Applying the factor of 0.622 to this range, the integrated motor vehicle exposure is estimated to range from 1.54 to 2.68 $\mu g/m^3$. Since the unit risk estimate for benzene is an upper bound estimate, and 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. See Section 5.3.3 for the discussion of total integrated benzene exposure in the TEAM study (EPA, 1987; Wallace, 1989).

5.5.3 Short-Term Microenvironment Exposures

The primary emphasis for benzene and other exposures in microenvironments are relatively localized scenarios which are highly impacted by motor vehicle emissions. These microenvironments include in-vehicle exposure, parking garage exposure, and exposure to vehicle refueling emissions. The information contained in Table 5-7 is excerpted from four studies that have measured microenvironment exposures to benzene. These four studies are the EPA's Total Exposure Assessment Methodology

(TEAM) Study (EPA, 1987b), Commuter's Exposure to Volatile Organic Compounds, Ozone, Carbon Monoxide, and Nitrogen Dioxide (Chan et al., 1989), In-Vehicle Air Toxics Characterization Study in the South Coast Air Basin (Shikiya et al., 1989), and Air Toxics Microenvironment Exposure and Monitoring Study (Wilson et al., 1991). See the information in Section 4.2 for more details about the methodology.

The TEAM Study (EPA, 1987b; Wallace, 1989) was planned in 1979 and completed in 1985. The goals of this study were: 1) to develop methods to measure individual total exposure (exposure through air, food and water) and resulting body burden to toxic and carcinogenic chemicals, and 2) to apply these methods with a probability-based sampling framework to estimate the exposures and body burdens of urban populations in several U.S. cities. This was achieved through the use of small personal samplers, a specially designed spirometer (used to measure the chemicals in exhaled breath), and a survey designed to insure the inclusion of potentially highly exposed groups.

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

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

Table 5-7. Microenvironment Exposure to Benzene (µg/m^3).

Scenarios	In-Vehicle		Service Station		Parking Garage		Office Building	
	Mean	Max.	Mean	Max.	Mean	Max.	Mean	Max.
TEAM Study (EPA, 1987b)	--	40-60[a]	--	3000[b]	--	--	--	--
Raleigh, NC Study[c] (Chan et al., 1989)	10.9	42.8	--	--	--	--	--	--
SCAQMD Study[d] (Shikiya et al., 1989)	42.5	267.1	--	--	--	--	--	--
SCAQMD Study[e] (Wilson et al., 1991)	--	--	--	288	--	67.1	--	16.0

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

commuters from a non-industrial park were sampled for in-vehicle concentrations of 14 toxic air pollutants, carbon monoxide, and lead.

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

Maximum microenvironment exposure levels of benzene related to motor vehicles were determined in these studies to range from 40 $\mu g/m^3$ from in-vehicle exposure to 288 $\mu g/m^3$ from exposure during refueling. This compares to ambient levels of 4.13 to 7.18 $\mu g/m^3$ determined through air monitoring studies and presented in Table 5-6. Since for the majority of the population these are short-term acute exposures to benzene, the concern would be with non-cancer effects. Health information for non-cancer effects is limited and no RfC has been developed by EPA. Several studies recently conducted in rats and mice have observed depressed cell proliferation in specific bone marrow cells at short-term exposures of 3.2×10^4 $\mu g/m^3$ benzene. Please see Section 5.8 for more information on non-cancer effects.

Due to more stringent fuel and vehicle regulations, short-term exposure to benzene in these microenvironments is expected to decrease in future years.

5.6 Carcinogenicity of Benzene and Unit Risk Estimates

5.6.1 Most Recent EPA Assessment

The information presented in Section 5.6.1 has been abstracted from EPA's Interim Quantitative Cancer Unit Risk Estimates Due to Inhalation of Benzene (EPA, 1985), EPA's Integrated Risk Information System (EPA, 1992c), and the Motor Vehicle Air Toxics Health Information (Clement, 1991). The carcinogenicity risk assessment for benzene was last updated on IRIS in January 1992, and contains data published through 1987. However, it is essentially unchanged from the risk assessment published in 1985. EPA's Office of Research and Development has just recently started the process to review the benzene risk assessment. Data published since the 1985 risk assessment for benzene is summarized in Section 5.6.3.

5.6.1.1   Description of Available Carcinogenicity Data

Genotoxicity

        Benzene has been found to induce chromosomal aberrations
(i.e., abnormalities in the chromosomes) in bone marrow cells
from rabbits (Kissling and Speck 1973), mice (Meyne and Legator
1980), and rats (Anderson and Richardson 1979). Several
investigators have reported positive results for benzene in mouse
micronucleus assays (Meyne and Legator 1980). The micronucleus
assay is a laboratory method in which blood cells are examined to
determine if broken chromosomes have formed small extra nuclei in
the cytoplasm of the cell. Benzene was not mutagenic (i.e., did
not cause changes in the genetic material) in several bacterial
and yeast systems (e.g., Crebelli et al. 1986; De Flora et al.
1984; Glatt et al. 1989; Lee et al. 1988; Tanooka 1977), in the
sex-linked recessive lethal mutation assay with *Drosophila
melanogaster* (fruit fly) (Kale and Baum 1983) or in the mouse
lymphoma cell forward mutation assay (Oberly et al. 1984).

Animal Studies

        Exposure of rodents to benzene either by gavage (compound is
administered directly into the stomach by means of a stomach tube
inserted down the throat) or inhalation has resulted in tumor
formation. Maltoni and Scarnato (1979) and Maltoni et al. (1983)
administered 0, 50, 250, and 500 mg/kg benzene by gavage to
Sprague-Dawley[1] rats (30-40/sex/dose) for life. Rats
demonstrated dose-related increases in the incidence of mammary
tumors (females), Zymbal gland carcinomas (a malignant tumor of a
gland that surrounds the ear canal in rats that secretes an oily
substance), oral cavity carcinomas, and leukemias/lymphomas in
both sexes. Leukemia is an acute or chronic disease that is
characterized by unrestrained growth of leukocytes (white blood
cells) and their precursors in the tissues[2]. Lymphoma is a
general

---

        [1]The names and/or numbers preceding rats or mice throughout this document
denote specific laboratory strains.

        [2]Leukemia may be divided into granulocytic leukemias (which include
myelocytic, monocytic, and erythroblastic cell types) and lymphocytic leukemias.
Both granulocytic and lymphocytic leukemia may, in turn, be separated into acute
and chronic forms. In acute myeloid leukemia (AML) there is diminished
production of normal erythrocytes, granulocytes, and platelets which leads to
death by anemia, infection, or hemorrhage. These events can be rapid. In
chronic myeloid leukemia (CML) the leukemic cells retain the ability to
differentiate (i.e., be responsive to stimulatory factors) and perform function;
later there is a loss of the ability to respond.

term for inappropriate growth of new tissue or neoplastic[3] growth in the lymphatic system.

In a National Toxicology Program (NTP 1986) study, benzene was administered by gavage to Fischer-344/N rats (50/sex/dose) at doses of 0, 50, 100, or 200 mg/kg and to B6C3F$_1$ mice (50/sex/dose) at doses of 0, 25, 50, or 100 mg/kg . The animals were treated 5 times/week for 103 weeks. There were significant increases in the incidence of various neoplastic growths in both sexes of both rats and mice. Both species had an increased incidence of carcinomas of the Zymbal gland. Male and female rats had oral cavity tumors, and males showed an increased incidence of skin tumors. Males were observed to have tumors of the Harderian (a gland located within the eye of the rat) and preputial gland (a small gland located near the head of the penis that secretes an odiferous discharge important to mating), and females had tumors of the mammary gland and ovaries. In general, the increased incidence was dose-related.

Inhalation exposure of male C57B1 mice to 300 ppm benzene on a workday schedule (6 hours/day, 5 days/week) for 488 days resulted in slight increases in the incidence of hematopoietic neoplasms (Snyder et al. 1989). However, there was no increase in tumor incidence in male AKR mice exposed to 100 ppm or male CD-1 mice exposed to 100 or 300 ppm benzene. Likewise, male Sprague-Dawley rats exposed by inhalation to 300 ppm benzene were not observed to have an increased incidence of neoplasia.

Maltoni et al. (1983) treated male and female Sprague-Dawley rats in the following manner. Starting at 13 weeks of age, rats were exposed to 200 ppm benzene by inhalation 4 hours/day, 5 days/week for 7 weeks. Animals were then exposed to the same concentration for 7 hours/day, 5 days/week for 12 weeks, and finally 300 ppm 7 hours/day, 5 days/week for 85 weeks. A time-weighted average (TWA) of 241 ppm for an 8 hours/day, 5 days/week exposure was calculated. In this study, a statistically significant increase in the incidence of liver tumors (hepatomas) and carcinomas of the Zymbal gland was found.

Goldstein et al. (1980) conducted studies in male Sprague-Dawley rats exposed to 0 ppm (67/group), 75 ppm (40/group), or 225 ppm (45/group) benzene by inhalation for an unreported period of time. In this study, one animal contracted leukemia in the 75 ppm concentration group. In addition, AKR, C57BL, and CD-1 mice were exposed to 0 ppm (210/group), 75 ppm (50/group), or 225 ppm (160/group) benzene again for an unreported period of time. After this treatment, two animals in the high-concentration exposure group developed leukemia.

Human data

------

[3]Neoplastic growth is characterized by new and abnormal formation of tissue, usually as a tumor. By custom, this refers to the pathological process in tumor formation, i.e., cancer.

Rinsky et al. (1981) followed 748 Pliofilm® (a film made of rubber hydrochloride) workers (all white males) exposed to benzene at levels that averaged from 10-100 ppm over an 8-hour workday (8-hour time-weighted average, TWA) for at least 24 years (17,020 person-years, an expression of cumulative dose). Seven deaths resulted from leukemia in this group. This increased incidence was statistically significant with a standard mortality ratio (SMR) of 560. The standard mortality ratio is the number of deaths, either total or cause-specific, in a given group expressed as a percentage of the number of deaths that would have been expected in that group if they were the same as the age-and-sex-specific rates in the general population. For the 5 leukemia deaths that occurred among workers with more than 5 years of exposure, the SMR was 2,100. Exposures were described as less than the recommended standards (25 ppm) for the time period of 1941-1969. A computer tape containing follow-up information for the Rinsky population through the year 1978 was used in addition to the original Rinsky et al. (1981) data to develop unit risk estimates. No effort was made to correct for smoking or other potential confounding exposures.

Ott et al. (1978) studied 594 white male workers occupationally exposed to benzene in a chemical manufacturing facility at concentrations ranging from 2 to 25 ppm (8-hour TWA). This group was followed for at least 23 years in a retrospective cohort mortality study. A retrospective cohort is a group of people, defined by arbitrary criteria as alike in some way, some of whom are known to have experienced particular exposures as well as particular health effects at some time prior to the start of the investigation. Although three leukemia deaths were observed, the increase was not statistically significant when compared to an unexposed population.

Wong et al. (1983) studied 4,062 male (both white and nonwhite) chemical workers who had been exposed to benzene for at least 6 months between 1946-1975. The study population was drawn from seven chemical plants, and jobs were categorized with respect to peak exposure. Subjects with at least 3 days/week exposure (3,036 individuals) were further categorized on the basis of an 8-hour TWA and were compared to controls who held jobs at the same plants for at least 6 months without exposure to benzene. The range of exposures experienced by these workers was <1 ppm to >50 ppm (8-hour TWAs). Statistically significant dose-dependent increases in the incidence of leukemia, lymphatic, and hematopoietic cancer (i.e., cancers of the blood forming organs) were found when the data were analyzed in terms of cumulative exposure (i.e., exposure level multiplied by duration of exposure). The incidence of leukemia was responsible for a majority of the increase, due largely to the fact that the incidence of mortality due to neoplasia in unexposed subjects was lower than expected.

Aksoy et al. (1974) reported effects of benzene exposure among 28,500 Turkish workers employed in the shoe industry who used benzene-containing adhesives. Mean duration of employment

was 9.7 years (1-15 year range) and mean age was 34.2 years. Peak exposure was reported to be 210-650 ppm. Twenty-six cases of leukemia and a total of 34 leukemias or preleukemias (blood conditions that are thought to precede the onset of leukemia) were observed, corresponding to an incidence of 13/100,000 (by comparison to 6/100,000 for the general population). This represents a statistically significant increase in the incidence of leukemia among the shoe workers. The possibility of concomitant exposure to other agents was not discussed. A follow-up paper (Aksoy 1980) reported eight additional cases of leukemia as well as evidence suggestive of increases in other malignancies in exposed workers.

The leukemogenic (i.e., the ability to induce leukemia) effects of benzene exposure were studied in 748 white males employed from 1940-1949 in the manufacturing of rubber products in a retrospective cohort mortality study (Infante et al. 1977a,b). Statistics were obtained through 1975. A statistically significant increase in the incidence of leukemia was found by comparison to the general U.S. population. The worker exposures to benzene were between 100 ppm and 10 ppm during the years 1941-1945. There was no evidence of solvent exposure other than benzene.

There are many other epidemiologic and case studies that report increased incidence or a causal relationship between leukemia and benzene exposure. In addition, numerous investigators have found significant increases in chromosomal aberrations of bone marrow cells and peripheral lymphocytes from workers with exposure to benzene (IARC 1982).

5.6.1.2 Weight-of-Evidence Judgment of Data and EPA Classification

The weight-of-evidence indicates that benzene is a Group A, known human carcinogen. This is based on sufficient human epidemiologic evidence (Rinsky et al. 1981; Ott et al. 1978; Wong et al. 1983) demonstrating an increased incidence of nonlymphocytic leukemia from occupational inhalation exposure, in addition to supporting animal evidence (Goldstein 1980; NTP 1986; Maltoni et al., 1983) in which there was an increased incidence of neoplasia in rats and mice exposed by inhalation and gavage.

5.6.1.3   Data Sets Used For Unit Risk Estimate

    The data sets used to estimate the unit risk[4] for benzene were obtained from a reorganization of the 1981 Rinsky et al. data (followup from 1940 to 1978), Wong et al. (1983), and Ott et al. (1978). These three studies are summarized in Table 5-8.

    The Rinsky data used were from an updated tape that reports one more case of leukemia than was published in 1981. It should be noted that a recently published paper (Rinsky et al. 1987) reported 2 additional cases of leukemia from the study population but was not used for the current risk estimate. Updates of other cohorts used in the current EPA assessment are discussed in Section 5.6.3. Generally, the updates report increased cohort size, improved exposure analyses, and/or alternative methods to analyze the cancer incidence data.

    Although the data from Aksoy et al. (1974) and Aksoy (1978) indicated an association of leukemia with benzene, it was decided by EPA that the exposure information was so imprecise that it was not suitable for quantitative estimation.

    Selection of the models used in the EPA estimate of unit risk was "a matter of judgement." The estimates were based on the most extensive and inclusive body of data available that is of acceptable quality, so all three epidemiologic studies were used. The choice of the studies in which the species (human) and route of exposure (inhalation) most closely corresponded to the environmentally exposed population were given the most weight. Animal studies were merely used for confirmation purposes.

5.6.1.4 Dose-Response Model Used

    The unit lifetime risk estimate was determined by using the relative risk model and the absolute risk model with three different measures of dose (total of six models) to develop 21 maximum likelihood estimates (MLEs). These 21 MLEs were then used to calculate a geometric mean to determine the unit risk estimate.

_____

    [4]Under an assumption of low-dose linearity, the unit cancer risk is the excess lifetime risk due to continuous constant lifetime exposure to one unit of carcinogen concentration. Typical exposure units include ppm or ppb in food or water, mg/kg/day by ingestion, or ppm or $\mu g/m^3$ in air (EPA 1986b).

Table 5-8. Summary of Data Sets Used to Calculate the Unit Risk Factor For Benzene[a].

Study	Population Studied/Years of Follow-up	Duration of Exposure	Exposure Level(s)	Effect(s)
Rinsky et al. (1981)	748 Pliofilm® workers (all white males)/ 38 years	At least 24 years	10-100 ppm (8-hour TWA)	Statistically significant increased incidence of leukemia
Ott et al. (1978)	594 chemical workers (white and nonwhite)/ at least 23 years	Not specified	2-25 ppm (8-hour TWA)	Increased incidence of leukemia (not statistically significant)
Wong et al. (1983)	44,062 male chemical workers (white and nonwhite)/ 29 years	At least 6 months	<1ppm->50 ppm	Statistically significant increased incidence of leukemia, lymphatic, and hematopoietic cancer

[a]Various subsets of these studies were used to develop the 21 unit risk factors.

5.6.1.5 Unit Risk Estimates[5]

As stated above, six models and various combinations of epidemiological data sets were used to derive a total of 21 MLEs,with their 95% statistical confidence bounds. Because the EPA had no basis for choosing one model over another, the geometric mean of these 21 unit risk estimates was taken to obtain a pooled model average estimate, resulting in a maximum likelihood estimate (MLE) unit risk of 2.7×10^{-2} for leukemia due to a lifetime exposure of 1 ppm benzene in the air (8.3×10^{-6} $[\mu g/m^3]^{-1}$).

The actual 95% upper bound (UCL) was calculated for each MLE derived with each of the 21 models. These data are presented in Tables E-1 through E-6 in Appendix E, which were reproduced from (EPA, 1985). A geometric mean of the UCLs was not calculated.

5.6.2 Other Views and Risk Estimates

This section presents alternative views and/or risk assessments for benzene. These alternative risk assessments are summarized in Table 5-9.

International Agency for Research on Cancer (IARC)

IARC has classified benzene as a Group 1 carcinogen. A Group 1 carcinogen is defined as an agent that is carcinogenic to humans. This classification is based on sufficient evidence for carcinogenicity in humans (IARC, 1987). IARC (1987) based this conclusion on the fact that numerous case reports and follow-up studies have suggested a relationship between exposure to benzene and the occurrence of various types of leukemia.

In addition, IARC (1987) considers the evidence for carcinogenicity to animals to be sufficient. No unit risk was determined by IARC for benzene.

California Air Resources Board (CARB)

The California Department of Health Services (DHS, 1984) (which provides technical support to CARB) has also determined that there is sufficient evidence to consider benzene a human

---

[5]For any dose-response model, one typically obtains risk estimates for various dose levels. It is possible to obtain maximum-likelihood estimates (MLEs) and upper confidence limits (UCLs) for those risks. The MLEs represent the best description of the observed data that can be obtained for any given dose-response model. However, because there are many sources of error that affect the observation of responses (including, but not limited to, random error) it is often desirable to determine upper bounds on the risks. The UCLs are statistical estimates of those upper bounds; they determine the highest levels of risks associated with specific dose levels that are consistent with the observed responses, the dose-response model, and the level of certainty required by the user.

Table 5-9. Comparison of Benzene Inhalation Unit Risk Estimates.

Source	Tumor Type	Classification	Cancer Unit Risk Estimate $(\mu g/m^3)^{-1}$	Cancer Unit Risk Estimate $(\mu g/m^3)^{-1}$ MLE
EPA (1985)	Nonlymphocytic leukemia in occupational studies	Group A[a]	6.7×10^{-6} – 1.5×10^{-4} [b]	8.3×10^{-6} [c]
IARC (1987)	Leukemia in occupational studies	Group 1[d]	–[e]	–[e]
CARB (DHS 1984)	Leukemia in occupational studies (for lower bound on risk) and preputial gland tumors in mice and rats (for upper bound on risk)	Human Carcinogen	5.2×10^{-5} [f]	8.3×10^{-6} [g]
CARB (CAPCOA 1991)	--	--	2.9×10^{-5} "best value"	
Clement (1988)	Leukemia in occupational studies	–[h]	–[i]	4.3×10^{-8} – 1.1×10^{-6} [j]

[a]Group A = Human Carcinogen
[b]Range of 21 95% UCLs. Geometric mean not calculated
[c]Geometric mean of 21 MLEs
[d]Group 1 = Human Carcinogen
[e]IARC did not conduct a quantitative risk assessment
[f]Lower bound of cancer risk
[g]Upper bound of cancer risk
[h]Clement did not classify benzene
[i]Clement did not calculate UCLs
[j]Range of MLEs calculated using different assumptions and data

carcinogen. CARB performed a risk assessment of benzene that was very similar to EPA's risk assessment. DHS, like EPA, assumed that there is no threshold for benzene-induced carcinogenicity, and that the multistage theory most appropriately describes the phenomenon of benzene-induced carcinogenesis.

The CARB potency factor for benzene is actually a range of potency factors. For the lower end of the range, DHS calculated a MLE potency estimate for benzene, like EPA had originally done, based on the reevaluation of three epidemiological studies using the linearized multistage model and cumulative exposure averaged over the individual's lifetime (see Appendix F for a lay description of this model). However, whereas EPA calculated a geometric mean of each of the three study's estimated slopes to obtain one slope factor, DHS used combined input data (i.e., background rate of leukemia, relative risk, and lifetime average exposure level) from the three studies to calculate one slope factor. As a result, the MLE slope factor calculated by EPA yielded an increase in risk of 2.7×10^{-2} due to a continuous lifetime exposure of 1 ppm benzene in the air (8.3×10^{-6} [$\mu g/m^3$]$^{-1}$) whereas the DHS MLE slope factor corresponds to a lifetime risk of 4.8×10^{-2} (ppm)$^{-1}$ (1.5×10^{-5} [$\mu g/m^3$]$^{-1}$). CARB chose to use EPA's MLE value of 2.7×10^{-7} ppm^{-1} as the lower bound for the benzene cancer potency factor range.

For the upper end of the range, a 95% UCL was calculated based on the most sensitive site in rats and mice, the preputial gland, using the data from the NTP (1983) study. This data set yielded a risk of 1.7×10^{-1} per ppm in air (5.2×10^{-5} [$\mu g/m^3$]$^{-1}$), which is 3.5-7 times as great as the risk estimated from human mortality data. Thus, the CARB potency estimate for benzene ranges from 2.7×10^{-2} to 1.7×10^{-1} ppm^{-1} ($8.3x10^{-6}$ to $5.2x10^{-5}$ [$\mu g/m^3$]).

CARB has also used what is termed as a "best value" for the benzene estimate provided by the California Department of Health Services in conjunction with the California Air Pollution Control Officers Association (CAPCOA, 1991). The CARB "best value" for benzene is 2.9×10^{-5} ($\mu g/m^3$)$^{-1}$ (9.4×10^{-2} ppm^{-1}) which falls within a range of unit risk factors, 0.75×10^{-5} to 5.3×10^{-5} ($\mu g/m^3$)$^{-1}$, (2.4×10^{-2} to 1.7×10^{-1} ppm^{-1}) recommended by DHS for health effects assessments which were prepared for the State's Toxic Air Contamination Program.

Motor Vehicle Manufacturer's Association (MVMA)

MVMA contracted with Environ (Environ 1987) to evaluate the risk assessment issues in EPA's technical report "Air Toxics Emissions from Motor Vehicles" (Carey 1987). It is important to note that the Environ document is not actually a risk assessment of benzene; rather, it is a critique of EPA's risk assessment of motor vehicle air toxics.

In a discussion of the possible impact of alternative approaches, Environ (1987) mentioned a risk assessment it performed for the Western Oil and Gas Association, in which it

calculated an estimate of benzene risk that was approximately one-fourth of that developed by EPA. This estimate was based upon the Rinsky et al. (1987) cohort, and assumptions about their exposure as developed by Crump and Allen (1984). No unit risk was determined in this study by Environ for the MVMA.

American Petroleum Institute

The American Petroleum Institute (API) states that the Yin et al. (1987) study should not be used in evaluating human health risks associated with benzene exposure because of the technical limitations of the study. Technical problems cited by API include exposure to mixtures of chemicals, retrospective benzene exposure measurements, lack of information on other exposures, and no confounding factors (such as smoking) were taken into consideration. API also states that there are problems in the comparison of the chosen exposed and non-exposed cohorts.

API also does not support the conclusion of the most recent data that benzene could be a developmental and reproductive toxicant. API states that its preliminary analysis of these studies indicates that there is a lack of quality data necessary to support this conclusion. API specifically cites the study by Savitz et al. (1989) which reports an elevated risk of still births when fathers were exposed to benzene and specifically finds fault with the exposure methodology. In this study though, Savitz et al. (1989) do discuss the limitations and suggest further evaluation while assuming no definitive relationships.

API has also taken the results of the Lange et al. (1973b) immunological effects study and performed its own analysis. Lange et al. (1973b) indicated a relationship between benzene and an allergic blood disease. API claims that the data in Lange et al. (1973b) indicate non-significant differences between exposed and non-exposed groups.

Clement Associates, Inc.

Under the sponsorship of the American Petroleum Institute, the Chemical Manufacturers Association, and the Western Oil and Gas Association, Clement Associates, Inc. (Clement 1988) performed a quantitative re-evaluation of the human leukemia risk associated with inhalation exposure to benzene. Clement's risk assessment differed from EPA's in the following ways:

1)   The analysis of risk is based solely on the Rinsky epidemiology cohort, rather than Rinsky, Ott, and Wong. The justification for this was that the Rinsky study was the only one that was not confounded by exposure to other chemicals and had an observed statistically significant dose relationship. Furthermore, the Clement reanalysis made use of three more years of follow-up data on this cohort (Rinsky et al. 1987), and corrected several job code errors that existed in the data used in 1985.

2)   The absolute risk model, rather than a combination of the absolute and relative risk models, was used in the Clement reanalysis.

3)   The Clement reanalysis selected the weighted cumulative form as the most realistic biological latency distribution, rather than both a cumulative dose and a weighted cumulative dose weighting function.

4)   The Clement reanalysis uses the exact time-dependent exposure for each of the 1,740 individuals in the cohort as the critical information necessary to calculate the transition rate per unit of exposure parameter (i.e., the probability that one unit of exposure will result in the biological event that leads to leukemia). EPA calculated transition rate by relying on an aggregation of data that grouped all person-years observed in the epidemiology studies into six exposure intervals rather than treating each exposed individual separately.

5)   The Clement reanalysis adopts a different definition of the types of diseases (i.e., acute leukemia and myelodysplastic syndrome/chronic "aplastic" anemia) associated with benzene exposure and adjusts background rates accordingly.

6)   The Clement reanalysis adds a quadratic model based on the hypothesis that two hits (i.e., two molecules of a benzene metabolite) are required to induce the biological event that leads to leukemia and calculates a linear quadratic model[6] as an upper bound on the two-hit model. The results of these differences in approach are summarized in Table E-7 in Appendix E.

---

[6]Dose-response functions are often referred to based on the mathematical equations that define them. Many of the equations that are used are polynomials, which can be expressed in the general form

$$a_0 + a_1 * d + a_2 * d^2 + ... + a_k * d^k.$$

Each of the groups of symbols between the plus signs is referred to as a term of the polynomial. Polynomials are referred to by their degree, which is the highest power to which dose, d, is raised. The equation shown above has degree k. Multistage models that have been applied to cancer risk assessment are based on such polynomial expressions.

A linear-quadratic model is a polynomial-based model that has degree 2. That is, the polynomial on which a linear-quadratic model is based includes a term for the background (a_0), a term that is linear in dose (dose raised to the power 1, which is often represented with no exponent, i.e., d^1 is the same as d), and a term with dose raised to the second power ($a_2 * d^2$).

A pure quadratic model is similar to the linear-quadratic model in that it has degree 2. However, the pure quadratic model dose not have a linear term; it includes only the background term (a_0) and a term with dose raised to the second power. One can think of the pure quadratic model as a submodel of the linear-quadratic model, where the coefficient for the linear term (a_1) has been set to zero.

EPA Rebuttal to Clement (API) Risk Assessment of Benzene

EPA (Chen et al. 1989) raised several issues in response to the Clement (API) risk assessment of benzene. These issues are summarized below:

1) **Use of Rinsky's Cohort as the Sole Data Base.** Chen et al. stated that the Rinsky study lacks adequate exposure information during the early but critical years of employment of the cases. Also, none of the three epidemiological studies used by EPA is considered to be superior to any other.

   Furthermore, it was stated that Clement gave "an incomplete picture of other studies and therefore reduced their usefulness by leaving out important details about those studies that do not support the use of the Rinsky study as the sole data source." Therefore, Chen et al. (1989) does not agree with choosing the Rinsky cohort as the sole data base for the benzene risk assessment.

2) **Differences Between Clement (API) and Rinsky himself in the Use of Rinsky Data Tapes.** Chen et al. (1989) stated that there appears to be some differences between the Rinsky data tapes used by API and the Rinsky data tapes used by Rinsky himself in his 1987 published paper.

3) **Only Certain Types of Leukemia are Induced by Benzene.** Chen et al. (1989) did not agree that acute myelogenous leukemia and aplastic anemia were the only disease end points associated with benzene exposure. They stated that there is also evidence linking acute and chronic forms of lymphocytic leukemia as well as acute nonlymphatic leukemia and multiple myeloma to benzene exposure, and that these should be included in a risk assessment of benzene.

4) **Blood Counts and the Crump-Allen Exposure Estimate.**
Chen et al. (1989) stated that the evidence provided by
Clement to justify the use of the Crump and Allen
(1984) exposure estimate is disputed by Rinsky, and
that both the Rinsky and Crump and Allen exposure
estimates should be considered. Clement stated that
the Crump and Allen exposure estimate was superior to
Rinsky's because higher blood counts are correlated
with lower exposure estimates, while no correlation was
found using the Rinsky estimate.

5) **Benzene has a Non-Linear and Threshold Dose Effect.**
Clement stated that the Rinsky study showed a strong
non-linearity of leukemia mortality rate with dose
using either the Rinsky or the Crump and Allen exposure
estimates. EPA's view is that linear low-dose
extrapolation is preferred, unless low dose data and/or
mechanism/metabolism knowledge show otherwise.

6) **Clement's (API's) Model is Superior to EPA's 1985
Model.** As discussed above, Clement stated that their
model represents an improvement over EPA's 1985 model
because it incorporates latency period data and
individual exposure information. Chen et al. (1989)
maintains that the way the latency is incorporated in
the model is not appropriate, and that the equation
used by Clement to estimate benzene-induced age-
specific cancer rate is not accurate (see item 7
below).

7) **Problems in the Clement (API) Procedures for Risk
Calculation.** Chen et al. (1989) stated that the way
the latency is incorporated by API into the Moolgavkar
(MVK) model is both mathematically and biologically
inappropriate because it assumes that one and only one
single tumor cell will eventually lead to leukemia
death. Furthermore, it is stated by Chen et al. (1989)
that the argument provided in the Clement assessment to
support the use of an absolute risk model over a
relative risk model is not convincing.

5.6.3 Recent and Ongoing Research

5.6.3.1 Genotoxicity

Benzene and its metabolites have been shown to cause
clastogenic effects (damages or breaks of the genetic material
that can be observed at the chromosome level) such as sister
chromatid exchange (SCE), micronuclei, and chromosomal
aberrations in both *in vivo* and *in vitro* systems in both humans
and animals. However, studies attempting to show mutagenic
activity of benzene have generally been negative (Shahin and
Fournier 1978; Lebowitz et al. 1979; Bartsch et al. 1980;
Nestmann et al. 1980; Shimizu et al. 1983; Nylander et al. 1978).
Recent work by Glatt et al. (1989) has shown, using a closed
desiccator system, that benzene is mutagenic in *Salmonella*

typhimurium (a type of bacteria) strain TA1535 in the presence of activated microsomal enzymes. These results suggest that only the metabolites of benzene are mutagenic.

Conflicting results have been obtained regarding the ability of benzene to form DNA adducts *in vivo*. Although DNA adducts have been demonstrated in *in vitro* experiments with a variety of benzene metabolites, Reddy et al. (1989) have not observed DNA adducts in samples of liver, kidney, bone marrow, and mammary gland obtained from Sprague-Dawley rats following oral administration of benzene (500 mg/kg/day, 5 days/week, for up to 10 weeks). The only potential adducts identified were observed in isolated rat Zymbal glands. In contrast, Snyder et al. (1989) observed a peak upon HPLC analysis of bone marrow DNA from rats treated with 1 mL/kg of benzene, 1 time per day for 4 days, with a longer retention time than any of the deoxynucleotide standards, suggesting covalent binding of benzene/benzene metabolite with the DNA.

Recent research has also examined the genotoxicity (i.e., the ability to damage the chromosomes at the DNA level) of the recently identified benzene metabolite, *trans,trans*-muconaldehyde (Latriano et al. 1986). *Trans,trans*-muconaldehyde has been demonstrated to form stable DNA adducts when reacted with deoxyguanosine monophosphate (Latriano et al. 1989). Deoxyguanosine monophosphate is a nucleic acid that is one of the building blocks of DNA. Also, it has been shown to be strongly mutagenic in Chinese hamster V79 cells and weakly mutagenic in bacterial systems (Glatt and Witz 1990). When administered to mice, *trans,trans*-muconaldehyde increased sister chromatid exchanges (Witz et al. 1990).

Recent work has also demonstrated that 1,4-benzoquinone and 1,2,4-benzenetriol, which are metabolites of benzene, inhibit DNA synthesis in a cell-free DNA synthetic system (Lee et al. 1989). The inhibitory effect was concluded to be due to inhibition of polymerase α by these metabolites.

Chromosomal aberrations occurring in humans with leukemia thought to be associated with exposure to benzene have been reported. A recent letter to The Lancet by Lumley et al. (1990) described the case of a 58-year old heavy-goods-vehicle driver who had heavy exposure to gasoline (and thus, benzene) who developed thrombocytopenia, neutropenia, and acute myeloid leukemia. He was found to have multiple chromosomal abnormalities including deletion of the long arm of chromosome 5, which the authors describe as a cytogenetic hallmark of secondary leukemia. The authors cite this example of non-random chromosomal changes in individuals with known benzene exposure as useful for early detection of those at risk for developing leukemia.

In a recent study, Irons et al. (1992) tested the effects of benzene metabolites on the growth of myeloid progenitor cells (bone marrow cells that are the precursor to granulocytes and macrophages). The benzene metabolite, hydroquinone, has been shown in previous studies to cause malignant transformations such as inhibition of microtubule assembly (essential for cell division) and nondisjunctional events (a loss of all or part of chromosomes 5 and 7) in these progenitor cells. Irons et al. (1992) hypothesized that if agents with leukemogenic potential (such as hydroquinone) have the ability to produce alterations in the regulation of these cells (i.e., increased growth), the absolute number of dividing progenitor cells would be increased. The increased size of this dividing cell population would serve to increase the probability of malignant transformations occurring. Actively dividing cells are also more susceptible to transformations due to their nature.

In vitro pretreatment of murine (mouse) bone marrow cells with hydroquinone and the stimulating factor that is required for their differentiation and replication results in an enhancement of granulocyte/macrophage colony formation. The magnitude of hydroquinone-enhanced colony formation equals or exceeds that described for the synergistic action of other compounds known to cause cell differentiation with the stimulating factor. The potential of hydroquinone to alter growth response and induce differentiation in a myeloid (bone marrow) progenitor cell population may be important in the pathogenesis of acute myelogenous leukemia secondary to benzene exposure. Benzene leukemogenesis may result from the dual ability of its metabolites to promote progenitor cell growth and differentiation and also induce cytogenetic changes in replicating cells. If other leukemogenic agents act similarly, alterations in myeloid progenitor cell differentiation may be important in the pathogenesis of secondary acute myelogenous leukemia in general.

These new studies provide additional support for the clastogenic ability of benzene metabolites and provide new evidence for the potential mutagenic activity of some of these 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.

5.6.3.2 Pharmacokinetics

The tumor diversity observed in different strains and species of rodents has been proposed to be due to the production of a number of potentially carcinogenic metabolites of benzene that may act singly or in combination (although the specific "active" metabolites have not yet been identified) (Huff et al. 1989).

A number of recent studies have examined the effects of dose, dose rate, route of administration, and species on benzene metabolism. For example, Sabourin et al. (1987) demonstrated that administration of bolus doses $\geq$ 50 mg/kg by oral or

intraperitoneal injection to rats and mice exceeded the metabolic capacity of these rodents and resulted in a portion of the dose being exhaled as benzene and a decrease in conversion to reactive benzene metabolites. As the dose was increased above 50 mg/kg, proportionately more was exhaled and less converted to benzene metabolites. With inhalation exposures, a similar phenomenon was observed in rats and mice. However, mice had a more rapid metabolic rate than rats, resulting in higher metabolite production in mice after an extended inhalation exposure (i.e., 6 hr). In mice the toxic pathways became saturated, whereas, in rats there was a relative increase in nontoxic pathways as the dose increased.

In addition to the higher metabolic rate in mice than in rats, Sabourin et al. (1988) demonstrated that mice and rats use the various metabolic pathways for benzene to differing degrees. In mice, detoxification also predominated, but substantial conversion to toxic metabolites was apparent. In rats, no saturation of toxic pathways was evident with increasing dose rate; however, increases in the mouse exposure level resulted in a shift from toxic metabolic pathways to detoxification pathways (Sabourin et al. 1988). These results indicate that the net result of exposures to high concentrations (200 ppm by the inhalation route) is to decrease the proportion of toxic metabolites formed relative to the dose administered in both mice and rats, with low level, long duration exposures of mice producing more toxic metabolites.

Age-related differences in benzene pharmacokinetics also appear to occur. McMahon and Birnbaum (1991) found that the disposition of benzene differed between 3- and 18-month-old male B6C3F1 mice administered as a single oral dose of either 10 or 200 mg/kg benzene. These differences include increased urinary and fecal elimination, increased expiration of benzene derived CO_2, and an effect on the metabolism of benzene to specific metabolites. While these differences may be due to the physiological effects of aging rather than direct age-related differences in the metabolism of benzene, these results have important implications with respect to the extrapolation of data obtained in young or old animals to young or old humans.

Using information about the relationship of the exposure conditions with the internal dose of various metabolites, computer simulations can be generated to estimate metabolite concentrations after differing exposure regimens (Medinsky et al. 1989) and derive internal doses that may be correlated with observed carcinogenic responses (Bailer and Hoel 1989) for risk assessment. Recently, Bois and Spear (1991) attempted to correlate circulating levels of phenol and hydroquinone with the onset of cancer in rats and mice using a computer model of benzene metabolism. No correlation was observed, indicating that other metabolites or combinations of

metabolites may be important in the initiation of cancer following benzene exposure.

Two benzene metabolites that have been shown to interact metabolically are phenol and hydroquinone to produce 1,4-benzoquinone *in vitro* (Eastmond et al. 1987). The observation that phenol and hydroquinone, when administered together in mice, produced a much greater decrease in bone marrow cellularity than did administration of either metabolite alone, suggests that a similar enhancement of the formation of the myelotoxic metabolite, 1,4-benzoquinone, may also occur *in vivo*.

A physiologically based pharmacokinetic model (PBPK) model for benzene has been developed by Travis et al. (1990). PBPK models are designed to allow more accurate prediction of actual internal doses across species. This particular model was developed to quantitatively predict the fate of benzene in mice, rats, and humans following several routes of exposure. One of the advantages to having a highly predictive PBPK model for benzene is that exposure data from benzene-induced cancers in animals may be directly compared to exposure data from human epidemiological studies in terms of metabolized dose, and therefore, route-to-route extrapolations can be made with a higher degree of confidence.

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 exposures and when extrapolating high dose exposures in animals to the low levels of exposure typically encountered in occupational situations. The development of a PBPK model for benzene should help in performing interspecies and route-to-route extrapolations of cancer data. Furthermore, metabolite interactions should be considered in developing PBPK models.

5.6.3.3 Carcinogenicity - Animal Studies

Recent studies examining the carcinogenicity of benzene in rodents have demonstrated that benzene is a potent carcinogen in a number of organs in a variety of species and strains of mice and rats, whether administered orally or by inhalation. In a recent NTP bioassay (Huff 1986), administration of benzene by gavage produced a variety of types of tumors in male and female F344/N rats and B6C3F1 mice. Male rats were administered 0, 50, 100, or 200 mg/kg benzene and female rats and male and female mice were administered 0, 25, 50, 100 mg/kg in corn oil by gavage for 103 weeks. Female rats administered benzene at 25 mg/kg and above caused significantly increased incidences of Zymbal gland carcinoma and at 50 mg/kg and above, squamous cell carcinomas and papillomas of the oral cavity. In male rats, at 100 mg/kg and above, Zymbal gland carcinomas, squamous cell carcinomas (malignant tumors of the skin), and papillomas (benign tumors) of the oral cavity were

significantly increased. Also in male rats, skin papillomas were increased at 200 mg/kg and above.

In mice, significantly increased incidences of Zymbal gland carcinomas, malignant lymphomas, and alveolar/bronchiolar carcinomas at 50 mg/kg and above were observed. Harderian gland adenomas increased at 25 mg/kg and above, and squamous cell carcinomas of the preputial gland increases at 50 mg/kg and above were observed in male mice. At 25 mg/kg and above, malignant lymphomas increased and ovarian granulosa cell tumors (tumor of the ovary), carcinomas of the mammary gland, and alveolar/bronchiolar carcinomas increased at 50 mg/kg and above in female mice. Zymbal gland carcinomas at 100 mg/kg were also observed in females. Alveolar tumors are located in the deepest part of the lung in the tissue where air exchange with blood takes place. Bronchiolar tumors are located in the bronchial tubes in the lungs. In general, mice were more sensitive to the carcinogenic effects of benzene than were rats.

Similar results were presented by Maltoni et al. (1989). When administered benzene (0, 50, 250 mg/kg or 0, 500 mg/kg) by gavage in olive oil for 104 weeks, benzene-exposed Sprague-Dawley rats had increased incidences of tumors of the Zymbal gland, oral cavity, nasal cavity, skin, forestomach, liver angiosarcomas (malignant tumors in blood vessels in the liver) and marginal increases in carcinomas of the mammary glands, hepatomas (liver tumors), and leukemias. Wistar rats administered 0 or 500 mg/kg by gavage in olive oil for 104 weeks had increased incidences of carcinomas of the Zymbal gland, oral cavity, and nasal cavity in benzene-exposed animals. Swiss mice administered 0 or 500 mg/kg by gavage in olive oil for 78 weeks had increased incidences of carcinomas of the mammary gland, lung tumors, and carcinomas of the Zymbal glands in those mice exposed to benzene. RF/J mice administered 0 or 500 mg/kg by gavage in olive oil for 52 weeks had increased incidences of mammary carcinomas, lung tumors, and leukemias in those mice exposed to benzene. When adult Sprague-Dawley rats inhaled either 0 or 200 ppm 4 hr/day, 5 days/week for 7 weeks followed by 200 ppm 7 hr/day, 5 days/week for 12 weeks and then 300 ppm 7 hr/day, 5 days/week for 85 weeks, an increased incidence of carcinomas of the Zymbal glands and oral cavity were observed with marginal increases in carcinomas of the nasal cavity, mammary glands, and hepatomas in benzene-exposed rats. Slightly greater numbers of tumors were observed when inhalation exposure at the above concentrations began on day 12 of gestation.

Recently, an increased incidence of myelogenous leukemias (the type of cancer associated with benzene exposure in humans) was reported in mice exposed to benzene by inhalation (Cronkite et al. 1989). CBA/Ca mice were used in this study. These mice come from the same stock as a strain (CBA/H) known to have a low incidence of acute myeloblastic leukemia (a type of myelogenous leukemia), but which respond to ionizing radiation with a high incidence of these tumors. Inhalation of 300 ppm, 6 hr/day, 5 days/week, for 16 weeks significantly decreased survival and increased the incidence of myelogenous neoplasms in male and

female CBA/Ca mice. Also, an increased incidence of neoplasms other than hepatic and hematopoietic cancers such as squamous cell carcinoma, mammary adenocarcinoma (tumors of the mammary gland), Zymbal and Harderian gland tumors, and papillary adenocarcinomas of the lung was observed in these mice. These tumors (myelogenous neoplasms and other neoplasms) were observed earlier in benzene-treated animals than in the controls. Hepatic neoplasms also appeared sooner in the benzene-exposed mice; however, both hepatic and lymphomatous neoplasms were significantly decreased in benzene-treated mice. At lower concentrations (100 ppm), an increased incidence of tumors other than hematopoietic and hepatic neoplasms was also observed although no significant increase in myelogenous neoplasms was seen. Preliminary results indicated that exposure of these mice to much higher concentrations of benzene (3,000 ppm for 8 days) did not produce similar increases in mortality or cancer incidence. The absence of neoplastic effects at this high dose is consistent with the much lower hematotoxicity (blood disease) observed with exposure to 3,000 ppm for 2 days as compared with exposure to 316 ppm for 19 days (exposures designed to yield similar total doses of benzene).

These new 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 neoplasm identified most closely with occupational exposure, acute myelogenous leukemia. Benzene has been shown to be carcinogenic in both sexes, at multiple sites, in several strains of rats and mice.

5.6.3.4 Carcinogenicity - Epidemiological Studies

Several studies have become available since the 1985 EPA carcinogenicity assessment, and have not been considered in the derivation of the cancer potency factor for benzene. The study by Rinsky et al. (1981) has been updated through December 31, 1981 (Rinsky et al. 1987). An additional two deaths attributable to leukemia were included in the update, bringing the total number of leukemia deaths to nine. The standardized mortality ratio (SMR) for leukemia calculated in the update was 337 (95% confidence interval = 154-641). Also, a significant increase in multiple myeloma[7] was observed in the updated cohort (SMR=409, 95% confidence interval=110-1047). Latency for the leukemia deaths ranged from 5-30 years with seven of the nine deaths occurring with a latency of <20 years. In contrast, latency for all of the cases of multiple myeloma was >20 years. A matched case-control analysis was also performed using conditional logistic regression analysis. Conditional logistic regression is used in a case-control study when the cases (i.e., exposed individuals) and controls (i.e., non-exposed individuals) have

---

[7]Myeloma is a tumor originating in the cells of the blood-forming portion of bone marrow. Multiple myeloma is a type of cancer characterized by the infiltration of bone and bone marrow with myeloma cells that form multiple tumor masses. This disease is usually progressive and fatal, and is accompanied by anemia, renal lesions, and high globulin levels in the blood.

been matched (i.e., matched pairs). It then can provide an unbiased estimate on a number of factors of the relative risk. Although the information reported in the Rinsky et al. (1987) update do not qualitatively change the current EPA risk assessment for benzene (i.e., they support the conclusion that benzene exposure is associated with an increased incidence of leukemia), the analytical methods used in this update, the improved exposure data, and the larger cohort size may impact the quantitative assessment of cancer risk based on this cohort.

The study by Ott et al. (1978) has also been updated (Bond et al. 1986) expanding the cohort size from 594 to 956 and increasing the period of observation to 1940-1982. An additional death was reported in this update, bringing the total to four leukemia deaths (all of the myelogenous type). Myelogenous leukemias are diseases where there is unrestrained growth of myelocytes, which are large cells found in the bone marrow that develop into white blood cells. Although the SMR for leukemia deaths was not significantly elevated, the mortality due to acute myelogenous leukemia was significantly increased. However, a positive dose-relationship between benzene exposure and leukemia was not observed.

A conditional logistic regression case-control analysis of the Ott et al. (1978) and Bond et al. (1986) studies was performed by the American Petroleum Institute (API) similar to that described in the Rinsky et al. (1987) update. However, API failed to observe a statistically significant relationship between increasing cumulative benzene exposure and increased risk of leukemia (Peterson 1986).

The study by Wong et al. (1983) has also been updated (Wong 1987). Two additional leukemias have been added to the cohort, bringing the total to seven. When compared to workers with no occupational exposure to benzene, those with at least 720 ppm-months of exposure to benzene had a relative risk of 3.93 for lymphatic and hematopoietic cancer. Workers with <180, 180-719, and ≥720 ppm-months of exposure had a borderline significantly increased incidence of non-Hodgkin's lymphopoietic cancer with increased exposure.

A new retrospective mortality study was published by Yin et al. (1989) of 28,460 benzene-exposed workers from 83 factories in China. Mortality of workers with at least six months of exposure to benzene between January 1, 1972 and December 31, 1981 was compared with mortality of a similar number of workers from these factories who had not been exposed to benzene. Significantly increased SMRs for leukemia (SMR=5.74) and lung cancer (SMR=2.31) were observed among exposed males and an increased SMR for leukemia was observed among exposed females. A higher proportion of acute nonlymphocytic leukemias were observed and a lower proportion of acute lymphocytic leukemias were seen than in the general population. The risk of leukemia increased with exposure duration up to 15 years and then declined with additional years of exposure. Cumulative exposure estimates were also performed in this study although measurements of ambient benzene levels

were not complete for all of the subjects. The cumulative exposure estimates supported the findings by Rinsky et al. (1987) that leukemia was, in many cases, seen in workers with continuous low dose exposure less than 400 ppm-yr of exposure. Smoking histories were determined in this study and the results demonstrated that smoking had no effect on leukemia mortality. Smoking increased the mortality due to lung cancer, but significantly greater lung cancer mortality was observed in exposed nonsmokers than in nonexposed nonsmokers, suggesting that lung cancer may also be associated with benzene exposure.

The updated studies provide continued evidence of the carcinogenicity of benzene in humans, and incorporation of increased cohort sizes and improved exposure analyses in these studies may strengthen the current cancer risk assessment for benzene. Furthermore, the observation of significantly increased lung cancer, as well as increased acute myelogenous leukemia, in the new study by Yin et al. (1989), suggests that benzene might be a multisite carcinogen in humans, as has been indicated in animal studies.

Morris and Seifter (1992) hypothesize that the increase in breast cancer incidence observed in urban areas may be due to the increased exposure to aromatic hydrocarbons found in urban pollution. Aromatic hydrocarbons are capable of inducing breast cancer in animals and benzene is a known cause of leukemia in humans.

Most aromatic hydrocarbons and benzene are readily soluble in fatty tissue (e.g., breast tissue) where they are stored, concentrated, and metabolized in the breast tissue to carcinogenic compounds. Some of these aromatic hydrocarbons produce electron seeking metabolites which can adduct to the DNA, causing mis-replication which can lead to tumor production. Other metabolites can function in the role of tumor promoter by producing an oxidant through their metabolic detoxification pathways. These oxidants, oxygen free radicals (activated forms of oxygen), consume glutathione (an anti-oxidant in the cells) that would otherwise protect against tumor promotion. Some aromatic hydrocarbons can react with cell membrane receptor sites causing oxygen free radicals to peroxidant the polyunsaturated lipids of the cell membrane. These lipid peroxidases and their degradation by-products cause chromosomal breaks in the related cell and also in remote tissues. The consequence of long term hydrocarbon exposure is the possibility of an increased pro-oxidant state which destabilizes DNA, causes chromosomal breaks, and allows for initiation and promotion of breast cancer.

In urban communities, there is increased exposure to hydrocarbons due to the use of fossil fuels and, concurrently, there is an increased personal exposure to hydrocarbons. It is the authors' contention that this low dose, long term exposure to many mammary specific hydrocarbon carcinogens and to the promotional effects of perhaps hundreds of other carcinogenic and non-carcinogenic hydrocarbon metabolites accounts for the urban factor in breast cancer.

5.7 Carcinogenic Risk for Baseline and Control Scenarios

Since the benzene unit risk estimate is based on human epidemiology of death data, cancer numbers should be expressed as cancer deaths. The estimate of cancer deaths may underestimate cancer incidence associated with benzene, since survivorship rates were not included in the supporting studies. Table 5-10 summarizes the maximum likelihood estimates of annual cancer deaths for all scenarios. When comparing cancer deaths for the base control scenarios relative to 1990, there is a 39% reduction in 1995, a 50% reduction in 2000, and remains constant at a 50% reduction in 2010. The reduction in 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 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 fuels program reduces the number of cancer deaths by another 8% from the 1990 base control. The expanded use of reformulated fuels and the California program in the year 2000 produces another 6% reduction in cancer cases, for both scenarios, when compared to 1990. Expanded reformulated fuel use in 2010 reduces deaths due to cancer by 6% relative to 1990 and by approximately 10% for the expanded California standards scenario. Like the base case comparison, the cancer cases for the control scenarios are similar for 2000 and 2010 despite continued emission reduction, due to the projected population and VMT increase.

5.8 Non-Carcinogenic Effects of Inhalation Exposure to Benzene

EPA has no inhalation reference concentration for the noncancer effects of benzene that can be used as a basis for risk assessment. Benzene's carcinogenic effects serve as the basis for the benzene risk assessment. Since the focus of this report is on the carcinogenic potential of the various compounds, the noncancer

Table 5-10. Annual Cancer Death Projections for Benzene.[a,b]

Year-Scenario	Emission Factor g/mile	Urban Cancer Deaths	Rural Cancer Deaths	Total Cancer Deaths	Percent Reduction from 1990	
					EF	Cancer
1990 Base Control	0.0882	59	11	70	–	–
1995 Base Control	0.0472	36	7	43	46	39
1995 Expanded Reformulated Fuel Use	0.0413	31	6	37	53	47
2000 Base Control	0.0351	30	5	35	60	50
2000 Expanded Reformulated Fuel Use	0.0301	26	5	31	66	56
2000 Expanded Adoption of California Standards	0.0305	26	5	31	65	56
2010 Base Control	0.0285	30	5	35	68	50
2010 Expanded Reformulated Fuel Use	0.0248	26	5	31	72	56
2010 Expanded Adoption of California Standards	0.0228	24	4	28	74	60

[a]Projections have inherent uncertainties in emission estimates, dose-response, and exposure.
[b]The unit risk estimate for benzene is based on human data. Benzene is classified by EPA as a Group A, known human carcinogen based on sufficient human epidemiologic evidence in addition to supporting animal evidence.

information will be dealt with in a more cursory fashion. No
attempt has been made to synthesize and analyze the data
encompassed below. Also, no attempt has been made to accord more
importance to one type of noncancer effect over another. The
objective is to research all existing data, describe the
noncancer effects observed, and refrain from any subjective
analysis of the data.

The respiratory route is the major source of human exposure
to benzene, and much of this exposure is by way of gasoline
vapors and automotive emissions (EPA, 1980). Individuals
employed in industries that use or make benzene or benzene-
containing products may be exposed to the highest concentrations
of benzene, primarily by inhalation. In 1987, OSHA estimated
that approximately 238,000 workers were exposed to benzene in
seven major industry sectors, including petrochemical plants,
petroleum refineries, coke and chemicals, tire manufacturers,
bulk terminals, bulk plants, and transportation via tank trucks
(OSHA 1987). The toxic effects of benzene in humans and other
animals following inhalation exposure include central nervous
system (CNS), hematological, and immunological effects. In
humans, acute exposure to 20,000 ppm is usually fatal within 5-10
minutes (Gerarde 1960). Death is preceded by CNS effects such as
drowsiness, headache, nausea, staggering gait, delirium, vertigo,
tremors, convulsions, and unconsciousness (Cronin 1924; Gerarde
1960; Browning 1965). In humans, death has been tentatively
attributed to asphyxiation, respiratory arrest, CNS depression,
or cardiac arrhythmia (Winek and Collum 1971). Organ hemorrhage
was also reported. An inhalation LC_{50} (the concentration that is
lethal to half of the animals exposed by the inhalation route)
value for rats was calculated as 13,700 ppm for a 4-hour exposure
(Drew and Fouts 1974).

Benzene induces hematological effects in humans and animals.
Early stages of benzene toxicity may be characterized by
deficiencies in specific blood elements, resulting in anemia (a
reduction in the number of red blood cells), leukopenia (a
reduction in the number of white blood cells), or
thrombocytopenia (a reduction in the number of blood platelets).
Chronic inhalation exposure to benzene in humans results in
pancytopenia, a condition characterized by decreased numbers of
circulating erythrocytes (red blood cells), leukocytes (white
blood cells), and thrombocytes (blood platelets) (Aksoy and Erdem
1978; Aksoy et al. 1971)[8]. Individuals that develop pancytopenia

---

[8]Pancytopenia is the reduction in the number of all three major types of
blood cells (erythrocytes, or red blood cells, thrombocytes, or platelets, and
leukocytes, or white blood cells). In adults, all three major types of blood
cells are produced in the red bone marrow of the vertebra, sternum, ribs, and
pelvis. The red bone marrow contains immature cells, known as multipotent
myeloid stem cells, that later differentiate into the various mature blood cells.
Pancytopenia results from a reduction in the ability of the red bone marrow to
produce adequate numbers of these mature blood cells. Aplastic anemia is a more
severe blood disease and occurs when the bone marrow ceases to function, i.e.,
these stem cells never reach maturity. The depression in bone marrow function
occurs in two stages - hyperplasia, or increased synthesis of blood cell
elements, followed by hypoplasia, or decreased synthesis. As the disease
progresses, the bone marrow decreases functioning. This myeloplastic dysplasia

and have continued exposure to benzene may develop aplastic anemia (pancytopenia associated with fatty replacement of functional bone marrow), whereas others exhibit both pancytopenia and bone marrow hyperplasia, a condition that may indicate a preleukemic state (Aksoy et al. 1974; Aksoy and Erdem 1978). Similar hematological effects have been reproduced in animals.

Symptoms of immunotoxicity have been reported in workers chronically exposed to benzene at concentrations that ranged from 3.44-53.21 ppm for 1-21 years. Alterations in serum levels of immunoglobulin (proteins in the blood that are capable of acting as antibodies) and complement (a series of enzymatic proteins in normal serum that, in the presence of a specific stimulus, destroy bacteria and other cells) and indications of benzene-induced autoimmunity and allergy have been observed in benzene-exposed workers whose exposure has been intermediate or chronic (Lange et al. 1973a, 1973b). Eosinophilia, an indication of an allergic response, has been noted in Turkish workers (Aksoy et al. 1971). Evidence of a positive leukocyte autoagglutinnin test, associated with decreased granulocyte levels, was suggestive of allergic blood dyscrasia (disease) (Lange et al. 1973b). The autoagglutinnin test measures the clumping of one's own blood cells. A positive response indicates that one's own blood cells stimulate an allergic response in the body. In animals, lymphopenia appears to be the most consistent response to subchronic benzene exposure, and may be seen at exposures as low as 25 ppm (Cronkite et al. 1989). A dose-response study of short-term inhalation exposure to benzene in mice at levels of 10-30 ppm showed significantly depressed proliferative responses of bone-marrow-derived B cells and splenic T cells in mice (Rozen et al. 1984). Mice with *Listeria monocytogenes* (a form of bacteria) exposed to intermittent benzene concentrations of 300 ppm resulted in delayed cell-mediated immunity, causing increased bacterial numbers (730% of controls) on day 4 (Rosenthal and Snyder 1985).

The available human data on developmental effects of benzene are inconclusive. Savitz et al. (1989) conducted an epidemiological study aimed at assessing the effect of parents' occupational exposures on risk of stillbirth, preterm delivery, and small-for-gestational age infants. They used data from National Natality and Fetal Mortality surveys on the probability samples of live births and fetal deaths that occurred in the US in 1980 among married women. Savitz et al. (1989) found that a high maternal linkage to benzene was predictive of stillbirth risk. Another significant association was found for paternal exposure to lead and risk of small-for-gestational age. Despite the limitations inherent in the study design (i.e., lack of exposure data, small size of exposed populations, and possible confounding factors not accounted for), these results suggest that occupational exposure to benzene may be associated with adverse developmental and reproductive outcomes.

---

without acute leukemia is known as preleukemia. The aplastic anemia can progress to AML.

Several animal studies, involving acute inhalation exposure during pregnancy, have shown that exposure to benzene decreased body weight and increased skeletal variants such as missing sternebrae and extra ribs (Murray et al. 1979; Kimmel and Wilson 1973). Alterations in hematopoiesis (growth and development of blood elements) have been observed in the fetuses and offspring of pregnant mice exposed to benzene (Keller and Snyder 1986). Two recent reports have described adverse immunological and hematopoietic effects associated with *in utero* exposure to benzene. One of these studies, Wierda et al. (1989), produced results that suggest that *in utero* exposure of benzene may adversely alter B cell development and responsiveness, and thus, compromise the immune system after birth.

Benzene may impair fertility by causing ovarian atrophy among women occupationally exposed to high levels (levels not specified) of benzene (Vara and Kinnunen 1946). In mice, histopathological changes were observed in ovaries (bilateral cysts) and testes (atrophy/degeneration, decrease in spermatozoa, moderate increase in abnormal sperm forms) following exposure to 300 ppm benzene for 13 weeks (Ward et al. 1985). No studies were located regarding respiratory, hepatic, or renal effects in humans or animals after inhalation exposure to benzene.

Corti and Snyder (1990) reported in a recent abstract that inhalation exposure of female Swiss Webster mice to 10 ppm benzene on gestation days 6-15 resulted in a reduction in the number of erythrocyte progenitor cell colonies in bone marrow cell cultures from female offspring 6 weeks after birth. There was no effect apparent in the male offspring. These results suggest that *in utero* exposure to benzene may adversely affect normal hematopoietic development.

The inhalation reference concentration (RfC) for benzene is currently under review by the EPA RfD/RfC Workgroup (EPA, 1992c). The oral reference dose (RfD) for benzene will be reviewed by the EPA RfD/RfC Workgroup (EPA, 1992c).

5.10 References for Chapter 5

Aksoy, M., K. Dincol, T. Akgun, S. Erdem, and G. Dincol. 1971.
Haematological effects of chronic benzene poisoning in 217
workers. Br. J. Ind. Med. 28:296-302.

Aksoy, M., S. Erdem, and G. Dincol. 1974. Leukemia in shoe-
workers exposed chronically to benzene. Blood 44:837.

Aksoy, M. 1978. Benzene and leukemia. Lancet 1: 441.

Aksoy, M., and S. Erdem. 1978. Follow-up study on the mortality
and the development of leukemia in 44 pancytopenic patients with
chronic benzene exposure. Blood 52:285-292.

Aksoy, M. 1980. Different types of malignancies due to
occupational exposure to benzene: A review of recent
observations in Turkey. Environ. Res. 23: 181.

American Petroleum Institute. 1991. Letter from Terry F. Yosie
of API to Phil Lorang of EPA. December 9, 1991.

Anderson, D. and C.R. Richardson. 1979. Chromosome gaps are
associated with chemical mutagenesis (abstract No. Ec-9).
Environ. Mutat. 1: 179.

Atkinson, R. 1990. Gas-phase tropospheric chemistry of organic
compounds: a review. Atmos. Environ., 24A:1-41.

Atkinson, R., S. M. Aschmann, J. Arey, and W.P.L. Carter. 1989.
Formation of ring-retaining products from the OH radical-
initiated reactions of benzene and toluene. Int. J. Chem.
Kinet., 21:801-827.

Bailer, A.J. and D.G. Hoel. 1989. Metabolite-based internal
doses used in a risk assessment of benzene. Environ. Health
Perspect. 82:177-184.

Bartsch, H., C. Malaveille, A.M. Camus, G. Martel-Planche, G.
Brun, A. Hautfeuille, N. Sabadie, A. Barbin, T. Kuroki, C.
Drevon, C. Piccoli, and R. Montesano. 1980. Validation and
comparative studies on 180 chemicals with S. typhimurium strains
and V79 Chinese hamster cells in the presence of various
metabolizing systems. Mutat. Res. 76:1-50.

Bois, F. and R. Spear. 1991. Modelling benzene exposure.
Health and Environ. Digest 5:5.

Bond, G.G., E.A. McLaren, C. Baldwin, and R.R. Cook. 1986. An
update of mortality among chemical workers exposed to benzene.
Br. J. Ind. Med. 43:685-691.

Browning, E. 1965. Toxicity and metabolism of organic solvents.
Elsevier, Amsterdam.

California Air Pollution Control Officers Association (CAPCOA). 1991. Air Toxics "Hot Spots" Program: Risk Assessment Guidelines, January 1991.

California Air Resources Board (CARB). 1984. Report to the Scientific Review Panel on Benzene.

Carey, P.M. 1987. Air toxics emissions from motor vehicles. Ann Arbor, MI: U.S. Environmental Protection Agency, Office of Mobile Sources, EPA Report no. EPA-AA-TSS-PA-86-5.

Chan, C.C., H. Ozkaynak, J.D. Spengler, L. Sheldon, W. Nelson, and L. Wallace. 1989. Commuter's exposure to volatile organic compounds, ozone, carbon monoxide, and nitrogen dioxide. Prepared for the Air and Waste Management Association. AWMA Paper 89-34A.4.

Chen, C., D. Bayliss, and A. Chiu. 1989. Benzene public comments. Memorandum to Jack R. Farmer. May 5, 1989.

Chevron Oil Company. 1991. Communication to EPA summarizing the following studies: "Study to Determine the fate of Benzene Precursors in Gasoline", NIPER (under CARB Agreement 150128-32), 1988; "Exhaust Benzene Emissions from Late-Model Vehicles", API Publication No. 841-44700, 10/88; "Vehicle Evaporative and Exhaust Emissions as Influenced by Benzene Content of Gasoline", NIPER (Under CRC CAPE-35-83 and U.S. DOE), 4/86.

Clement Associates, Inc. 1988. Quantitative re-evaluation of the human leukemia risk associated with inhalation exposure to benzene. Supported by American Petroleum Institute, Chemical Manufacturers Association, Inc., and Western Oil and Gas Association. Final Report. October 1988.

Clement Associates, Inc. 1991. Motor vehicle air toxics health information. For U.S. EPA Office of Mobile Sources, Ann Arbor, MI. September 1991.

Cole, H. S., D. E. Lapland, G. K. Moss, and C. F. Newberry. 1983. The St. Louis Ozone Modeling Project. U. S. Environmental Protection Agency (EPA-450/4-83-021).

Corti, M. and C. Snyder. 1990. Long-term hemopoietic effects caused by *in utero* exposure to 10 ppm inhaled benzene and 5% ingested ethanol. Toxicologist 10:58.

Crebelli, R., D. Bellincampi, G. Conti, L. Conti, G. Morpurgo, and A. Carere. 1986. A comparative study on selective chemical carcinogens for chromosomal segregation, mitotic crossing-over and forward mutation induction in *Aspergillus nidulans*. Mutat. Res. 172:139-149.

Cronin, H.J. 1924. Benzol poisoning in the rubber industry. Boston Medical and Science Journal 191:1164-1166.

Cronkite, E.P., R.T. Drew, T. Inoue, Y. Hirabayashi, and J.E. Bullis. 1989. Hematotoxicity and carcinogenicity of inhaled benzene. Environ. Health Perspect. 82:97-108.

Crump K.S. and B.C. Allen. 1984. Quantitative estimates of risk of leukemia from occupational exposure to benzene. Prepared for the Occupational Safety and Health Administration.

De Flora, S., P. Zanacchi, A. Camoirano, C. Bennicelli, and G.S. Badolati. 1984. Genotoxic activity and potency for 135 compounds in the Ames reversion test and in a bacterial DNA-repair test. Mutat. Res. 133: 161-198.

DHS. 1984. California Department of Health Services. Part B. Health Effects of Benzene. Epidemiological Studies Section. November 1984.

Drew, R.T. and J.R. Fouts. 1974. The lack of effects of pretreatment with phenobarbital and chlorpromazine on the acute toxicity of benzene in rats. Toxicol. Appl. Pharmacol. 27:183-193.
Eastmond, D.A., M.T. Smith, and R.D. Irons. 1987. An interaction of benzene metabolites reproduces the myelotoxicity observed with benzene exposure. Toxicol. Appl. Pharmacol. 91:85-95.

Environ Corporation. 1987. Risk assessment issues in EPA's technical report "Air Toxics Emissions from Motor Vehicles". Prepared for the Motor Vehicle Manufacturers Association.

EPA. 1980. Ambient water quality criteria document for benzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office (Cincinnati, OH) and Carcinogen Assessment Group (Washington, DC), and the Environmental Research Labs (Corvalis, OR; Duluth, MN; Gulf Breeze, FL) for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-018.

EPA. 1985. Interim quantitative cancer unit risk estimates due to inhalation of benzene. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC for the Office of Air Quality Planning and Standards, Washington, DC.

EPA. 1986a. Mobile source benzene emissions and a preliminary estimate of their health impacts. Memo from Charles L. Gray, Jr. to Richard D. Wilson, May 15, 1986.

EPA. 1986b. The Risk Assessment Guidelines of 1986. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Office of Research and Development. Washington, DC. EPA/600/8-87/045.

EPA. 1987a. Draft Regulatory Impact Analysis: Control of Gasoline Volatility and Evaporative Hydrocarbon Emissions from New Motor Vehicles. Ann Arbor, Michigan: Office of Mobile Sources. July, 1987.

EPA. 1987b. The Total Exposure Assessment Methodology (TEAM) Study: Summary and Analysis: Volume I. Office of Research and Development, Washington, D.C. June 1987. EPA Report No. EPA/600/6-87/002a.

EPA. 1988. Locating and estimating air emissions from sources of benzene. Office of Air Quality Planning and Standards, Research Triangle Park, NC: EPA-450/4-84-007q.

EPA. 1989. AIRS user's guide volumes I-VII. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Research Triangle Park, N.C.

EPA. 1990a. VOC/PM Speciation Data System, version 1.32a (published in electronic form). Research Triangle Park, North Carolina: Office of Air Quality Planning and Standards.

EPA. 1990b. Air quality criteria for carbon monoxide. Office of Health and Environmental Assessment. EPA/600/8-90/045A. External review draft. March 1990.

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

EPA. 1991b. Nonroad Engine and Vehicle Emission Study. Office of Air and Radiation, Office of Mobile Sources, Ann Arbor, MI. November 1991. EPA Report No. 21A-2001.

EPA. 1992a. Control of Air Pollution From New Motor Vehicles and New Motor Vehicle Engines; Refueling Emission Regulations for Gasoline-Fueled Light-Duty Vehicles and Trucks and Heavy-Duty Vehicles; Proposed Rule. Federal Register 57(73):13220-13231.

EPA. 1992b. Regulation of Fuel and Fuel Additives: Standards for Reformulated and Conventional Gasoline; Proposed Rule. Federal Register 57(74):13416-13495.

EPA. 1992c. Integrated Risk Information System. U.S. Environmental Protection Agency. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

General Motors Corporation. 1991. Communication from S. R. Reddy to Chris Lindjhem, April 16, 1991.

Gerarde, H.W. 1960. Toxicology and biochemistry of aromatic hydrocarbons. London, England: Elsevier Publishing Co., 44-46.

Glatt, H. and G. Witz. 1990. Studies on the induction of gene mutations in bacterial and mammalian cells by the ring-opened

benzene metabolites *trans,trans*-muconaldehyde and *trans,trans*-muconic acid. Mutagenesis 5:263-266.

Glatt, H., R. Padykula, G.A. Berchtold, G. Ludewig, K.L. Platt, J. Klein, and F. Oesch. 1989. Multiple activation pathways of benzene leading to products with varying genotoxic characteristics. Environ. Health Perspect. 82:81-89.

Goldstein B.D., C.A. Snyder, S. Laskin, I. Bromberg, R.E. Albert, and N. Nelson. 1980. Myelogenous leukemia in rodents inhaling benzene. Unpublished data. (Cited in EPA, 1985)

Hogo, H. and M. W. Gery. 1988. "User's Guide for Executing OZIPM-4 with CBM-IV or Optional Mechanisms." U. S. Environmental Protection Agency. (EPA-600/8-88/082).

Huff, J.E. 1986. Toxicology and carcinogenesis studies of benzene (CAS No. 71-43-2) in F344/N rats and B6C3F1 mice (gavage studies). Technical report no. 289. DHHS, National Toxicology Program/National Institute of Environmental Health Sciences, Research Triangle Park, NC.

Huff, J.E., J.K. Haseman, D.M. DeMarini, S. Eustis, R.R. Maronpot, A.C. Peters, R.L. Persing, C.E. Chrisp, and A.C. Jacobs. 1989. Multiple-site carcinogenicity of benzene in Fischer 344 rats and B6C3F1 mice. Environ. Health Perspect. 82:125-163.

IARC. 1982. IARC monographs on the evaluation of carcinogenic risk of chemicals to humans. Volume 29. Some industrial chemicals and dyestuffs. International Agency for Research on Cancer. World Health Organization, Lyon, France. p. 345-389.

IARC. 1987. IARC monographs on the evaluation of carcinogenic risk of chemicals to humans. Supplement 7. Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42. International Agency for Research on Cancer. World Health Organization, Lyon, France. p. 120-122.

Infante, P.F., R.A. Rinsky, J.K. Wagoner and R.J. Young. 1977a. Benzene and leukemia. The Lancet 2(8043): 867-869.

Infante, P.F., R.A. Rinsky, J.K. Wagoner and R.J. Young. 1977b. Leukemia in benzene workers. Lancet 19: 76-78.

Ireson, R. G., E. L. Carr, J. L. Fieber, J. G. Heicken, T. C. Myers, G. M. Wilson, and G. Z. Whitten. 1990. "Urban formaldehyde and methanol concentrations for alternative methanol vehicle scenarios." Systems Applications International, San Rafael, California (SYSAPP-90/104).

Irons, R.D., W.S. Stillman, D.B. Colagiovanni, and V.A. Henry. 1992. Synergistic action of the benzene metabolite hydroquinone on myelopoietic stimulating activity of granulocyte/macrophage

colony-stimulating factor *in vitro*. Proc. Natl. Acad. Sci. 89:3691-3695.

Kale, P.G. and J.W. Baum. 1983. Genetic effects of benzene in *Drosophila melanogaster* males. Environ. Mutagen. 5:223-226.

Keller, K.A. and C.A. Snyder. 1986. Mice exposed *in utero* to low concentrations of benzene exhibit enduring changes in their colony forming hematopoietic cells. Toxicology 42:171-181.

Kimmel, C.A. and J.G. Wilson. 1973. Skeletal deviations in rats: Malformations or variations? Teratology 8:309-316.

Kissling, M. and B. Speck. 1973. Chromosome aberrations in experimental benzene intoxication. HELV. Med. Acta. 36: 59-66.

Lange, A., R. Smolik, W. Zatonski, and H. Glazman. 1973a. Leukocyte agglutinins in workers exposed to benzene, toluene, and xylene. Int. Arch. Arbeitsmed. 31:45-40.

Lange, A., R. Smolik, W. Zatonski, and J. Szymanska. 1973b. Serum immunoglobulin levels in workers exposed to benzene, toluene, and xylene. Int. Arch. Arbeitsmed. 31:37-44.

Latriano, L., B.D. Goldstein, and G. Witz G. 1986. Formation of muconaldehyde, an open-ring metabolite of benzene, in mouse liver microsomes. An additional pathway for toxic metabolites. Proc. Natl. Acad. Sci (U.S.) 83:8356-8360.

Latriano, L., G. Witz, B.D. Goldstein, and A.M. Jeffrey. 1989. Chromatographic and Spectrophotometric characterization of adducts formed during the reaction of *trans,trans*-muconaldehyde with ^{14}C-deoxyguanosine 5'-phosphate. Environ. Health Perspect. 82:249-251.

Lebowitz, H., D. Brusick, D. Matheson, D.R. Jagannath, M. Reed, S. Goode, and G. Roy. 1979. Commonly used fuels and solvents evaluated in a battery of short-term bioassays. Environ. Mutagen. 1:172-173.

Lee, E.W., C.D. Garner, and J.T. Johnson. 1988. A proposed role played by benzene itself in the induction of acute cytopenia: Inhibition of DNA synthesis. Res. Commun. Chem. Pathol. Pharmacol. 60: 27-46.

Lee, E.W., J.T. Johnson, and C.D. Garner. 1989. Inhibitory effect of benzene metabolites on nuclear DNA synthesis in bone marrow cells. J. Toxicol. Environ. Health 26:277-292.

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).

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., R.R. Schulhof, R.E. Jackson, M.M. Jimenez, G. Z. Whitten, G.M. Wilson, T.C. Meyers, and J.L. Fieber. 1992. Modeling the effects of reformulated gasolines on ozone and toxics concenetrations in the Baltimore and Houston areas. Systems Applications International, San Rafael, California (SYSAPP-92/127).

Lumley, M., H. Barker, and J.A. Murray. 1990. Benzene in petrol. Lancet 336:1318-1319.

Maltoni, C. and C. Scarnato. 1979. First experimental demonstration of the carcinogenic effects of benzene. Long-term bioassays on Sprague-Dawley Rats by oral administration. Med. Lav. 70: 352-357.

Maltoni, C., B. Conti and G. Cotti. 1983. Benzene: A multipotential carcinogen. Results of long-term bioassays performed at the Bologna Institute of Oncology. Am. J. Ind. Med. 4:589-630.

Maltoni, C., A. Ciliberti, G. Cotti, B. Conti, and F. Belpoggi. 1989. Benzene, an experimental multipotential carcinogen: Results of the long-term bioassays performed at the Bologna Institute of Oncology. Environ. Health Perspect. 82:109-124.

McAllister, R. A., W. H. Moore, J. Rice, E. Bowles, D. P. Dayton, R. F. Jongleux, R. G. Merrill, Jr., and J. T. Bursey. 1990. Urban Air Toxics Monitoring Program. U. S. Environmental Protection Agency (EPA-450/4-91-001).

McMahon, T.F. and L.S. Birnbaum. 1991. Age-related changes in disposition and metabolism of benzene in male C57BL/6N mice. In press.

Medinsky, M.A., P.J. Sabourin, R.F. Henderson, G. Lucier, and L.S. Birnbaum. 1989. Differences in the pathways for metabolism of benzene in rats and mice simulated by a physiological model. Environ. Health Perspect. 82:43-49.

Meyne, J. and M.S. Legator. 1980. Sex-related differences in cytogenic effects of benzene in the bone marrow of Swiss mice. Environ. Mutat. 2:43-50.

Morris, J.J. and E. Seifter. 1992. The role of aromatic hydrocarbons in the genesis of breast cancer. Medical Hypotheses 38:177-184.

Morris, R. E., T. C. Myers, H. Hogo, L. R. Chinkin, L. A. Gardner, R. G. Johnson. 1989. A low-cost application of the Urban Airshed Model to the New York metropolitan area and the city of St. Louis (Five Cities UAM Study Phase I). Systems Applications International, San Rafael, California (SYSAPP-89/070).

Murray, F.J., J.A. John, L.W. Rampy, R.A. Kuna, and B.A. Schwetz. 1979. Embryotoxicity of inhaled benzene in mice and rabbits. Am. Ind. Hyg. Ass. J. 40:993-998.

Nestmann, E.R., E.G. Lee, T.I. Matula, G.R. Douglas, and J.C. Mueller. 1980. Mutagenicity of constituents identified in pulp and paper mill effluents using the Salmonella/mammalian-microsome assay. Mutat. Res. 79:203-212.

Nielsen, T., T. Ramdahl, and A. Bjørseth. 1983. The fate of airborne polycyclic organic matter. Environ. Health Perspect., 47:103-114.

NTP. 1983. NTP technical report on the toxicology and carcinogenicity studies of benzene (CAS No. 71-43-2) in F344/N rats and B6C3F$_1$ mice (gavage studies), NIH Publication No. 84-2545. Research Triangle Park, NC. Board draft, July 1984.

NTP. 1986. Toxicology and carcinogenesis studies of benzene (CAS No. 71-43-2) in F344/N rats and B6C3F mice (gavage studies). NTP Technical Report Series No. 289. NIH Publication No. 86-2545.

Nylander, P.O., H. Olofsson, B. Rasmuson, and H. Svahlin. 1978. Mutagenic effects of petrol in Drosophila melanogaster. I. Effects of benzene and 1,2-dichloroethane. Mutat. Res. 57:163-167.

Oberly, T.J., B.J. Bewsey, and G.S. Probst. 1984. An evaluation of the L5178Y TK+/- mouse lymphoma forward mutation assay using 42 chemicals. Mutat. Res. 125:291-306.

OSHA. 1987. Occupational exposure to benzene. Final Rule. U.S. Department of Labor, Occupational Safety and Health Administration. Federal Register 52: 34460-34578.

Ott, M.G., J.C. Townsend, W.A. Fishbeck, and R.A. Langner. 1978. Mortality among individuals occupationally exposed to benzene. Arch. Environ. Health 33:3-10.

Peterson, H.D. 1986. Case-control analysis of Ott et al. cohort (letter). OSHA Docket H-059C, Exhibit 247, Tab D, Attachment 2.

Pezda, S.A., J.J. Vostal, and H.J. Wimette. 1991. Current and Future Research Needs in Health Effects of Air Toxics - An Automotive Industry Perspective. Presented at the Air and Waste Management Association Conference, October 16-18, 1991, Detroit, Michigan.

Possiel, N.C., D.C. Doll, K.A. Baugues, E.W. Baldridge, and R.A. Wayland. 1990. Impact of regional control strategies on ozone in the Northeastern United States. Prepared for the Air and Waste Management Association. AWMA Paper 90-93.3.

Reddy, M.V., G.R. Blackburn, C.A. Schreiner, M.A. Mehlman, and C.R. Mackerer. 1989. ^{32}P analysis of DNA adducts in tissues of benzene-treated rats. Environ. Health Perspect. 82:253-257.

Rinsky, R.A., A.B. Smith, R. Hornung, T.G. Filloon, R.J. Young, A.H. Okun, and P.J. Landrigan. 1987. Benzene and leukemia: an epidemiologic risk assessment. N. Eng. J. Med. 316:1044-1050.

Rinsky R.A., R.J. Young and A.B. Smith. 1981. Leukemia in benzene workers. Am. J. Ind. Med. 2: 217-245.

Robinson, J.P, J.A. Wiley, T. Piazza, and K. Garett. 1989. Activity Patterns of California Residents and Their Implications for Potential Exposure to Pollution. Draft final report. CARB-46-177-33. California Air Resources Board. Sacramento, CA.

Rosenthal, G.J. and C.A. Snyder. 1985. Modulation of the immune response to *Listeria monocytogenes* by benzene inhalation [Abstract]. Toxicol. Appl. Pharmacol. 80:502-510.

Rozen, M.G., C.A. Snyder, and R.E. Albert. 1984. Depression in B- and T-lymphocyte mitogen-induced blastogenesis in mice exposed to low concentrations of benzene. Toxicol. Lett. 20:343-349.

Sabourin, P.J., B.T. Chen, G. Lucier, L.S. Birnbaum, E. Fisher, and R.F. Henderson. 1987. Effect of dose on the absorption and excretion of [^{14}C]benzene administered orally or by inhalation in rats and mice. Toxicol. Appl. Pharmacol. 87:325-336.

Sabourin, P.J., W.E. Bechtold, L.S. Birnbaum, G.W. Lucier, and R.F. Henderson. 1988. Difference in the metabolism of inhaled ^{3}H-benzene by F344/N rats and B6C3F1 mice. Toxicol. Appl. Pharmacol. 94:128-140.

Savitz, D.A., E.A. Whelan, and R.C. Kleckner. 1989. Effect of parents' occupational exposures on risk of stillbirth, preterm delivery, and small-for-gestational-age infants. American Journal of Epidemiology 129:1201-1218.

Schere, K. L., and J. H. Sheffler. 1982. Final Evaluation of Urban-Scale Photochemical Air Quality Simulation Models. Environmental Sciences Research Laboratory, U. S. Environmental Protection Agency (EPA-600/3-82-094).

Shahin, M.M. and F. Fournier. 1978. Suppression of mutation induction and failure to detect mutagenic activity with Athabasca tar sand fractions. Mutat. Res. 58:29-34.

Shikiya, D.C., C.S. Liu, M.I. Kahn, J. Juarros, and W. Barcikowski. 1989. In-vehicle air toxics characterization study in the south coast air basin. South Coast Air Quality Management District, El Monte, CA. May, 1989.

Shimizu, M., Y. Yasui, and N. Matsumoto. 1983. Structural specificity of aromatic compounds with special reference to

mutagenic activity in Salmonella typhimurium: a series of chloro- or fluoro-nitrobenzene derivatives. Mutat. Res. 116:217-238.

Singh, H. B. and J. F. Kasting. 1988. Chlorine-hydrocarbon photochemistry in the marine troposphere and lower stratosphere. J. Atmos. Chem., 7:261-285.

Snyder, R., E. Dimitriadis, R. Guy, P. Hu, K. Cooper, H. Bauer, G. Witz, and B.D. Goldstein. 1989. Studies on the mechanism of benzene toxicity. Environ. Health Perspect. 82:31-35.

Tanooka, H. 1977. Development and application of *Bacillus subtilis* test systems for mutagens, involving DNA-repair deficiency and suppressible auxotrophic mutations. Mutat. Res. 42: 19-32.

Travis, C.C., J.L. Quillen, and A.D. Arms. 1990. Pharmacokinetics of benzene. Toxicol. Appl. Pharmacol. 102:400-420.

Tuazon, E. C., H. MacLeod, R. Atkinson, and W.P.L. Carter. 1986. α-Dicarbonyl yields from the NO_x-air photooxidations of a series of aromatic hydrocarbons in air. Environ. Sci. Technol., 20:383-387.

Vara, P. and O. Kinnunen. 1946. Benzene toxicity as a gynecologic problem. Acta. Obstet. Gynecol. Scand. 26:433-452.

Wallace, L.A. 1989. Major sources of benzene exposure. Environ. Health Perspect. 82:165-169.

Ward, C.O., R.A. Kuna, N.K. Snyder, et al. 1985. Subchronic inhalation toxicity of benzene in rats and mice. Am. J. Ind. Med. 7:457-473.

Whitten, G. Z. 1983. The chemistry of smog formation: A review of current knowledge. Environ. International, 9:447-463.

Wierda, D., A.G. King, R.W. Luebke, M.J. Reasor, and R.J. Smialowicz. 1989. Perinatal immunotoxicity of benzene toward mouse B cell development. Journal of the American College of Toxicology 8:981-996.

Wilson, A. et al. 1991. Air toxics microenvironment exposure and monitoring study. South Coast Air Quality Management District, El Monte, CA.

Winek, C.L. and W.D. Collom. 1971. Benzene and toluene fatalities. J. Occup. Med. 13:259-261.

Witz, G., W. Maniara, and V. Mylavarapu. 1990. Comparative metabolism of benzene and *trans,trans*-muconaldehyde to *trans,trans*-muconic acid in DBA/2N and C57BL/6 mice. Biochem. Pharmacol. 40:1275-1280.

Wong, O. 1987. An industry-wide mortality study of chemical workers occupationally exposed to benzene. II. Dose-response analyses. Br. J. Ind. Med. 44:382-395.

Wong, O., R.W. Morgan and M.D. Whorton. 1983. Comments on the NIOSH study of leukemia in benzene workers. Technical report submitted to Gulf Canada, Ltd., by Environmental Health Associates.

Yin, S-N., G-L. Li, F-D. Tain, Z-I. Fu, C. Jin, Y-J. Chen, S-J. Luo, P-Z. Ye, J-Z. Zhang, G-C. Wang, X-C. Zhan, H-N. Wu, and Q-C. Zhong. 1989. A retrospective cohort study of leukemia and other cancers in benzene workers. Environ. Health Perspect. 82:207-213.

6.0 FORMALDEHYDE

6.1 Chemical and Physical Properties (EPA, 1991a, 1992)

Formaldehyde is a colorless gas at normal temperatures with a pungent, irritating odor. It is the simplest member of the family of aldehydes and has the chemical formula HCHO. Formaldehyde gas is soluble in water, alcohols, and other polar solvents. The chemical and physical properties of pure formaldehyde are presented in Table 6-1.

In the presence of air and moisture at room temperature, formaldehyde readily polymerizes to a solid mixture known as paraformaldehyde. Another common form of formaldehyde is its cyclic trimer (three formaldehyde molecules forming a ring) known as trioxane ($C_3H_6O_3$). In aqueous solutions, formaldehyde reacts with water to form methylene glycol.

Pure, dry formaldehyde gas is stable from 25-100°C (77-212°F) and decomposes very slowly up to 300°C (572°F). Polymerization takes place slowly below room temperature but is accelerated by the presence of impurities. Decomposition of formaldehyde produces carbon monoxide and hydrogen gas. When catalyzed by certain metals (platinum, copper, or chromia and alumina), formaldehyde decomposition can produce methanol, methyl formate, formic acid, carbon dioxide, and methane.

Table 6-1. Chemical and Physical Properties of Pure Formaldehyde.

Properties	Values
Molecular weight	30.03 g/mole
Melting point	-92.0°C (-133.4°F)a
Boiling point at 1 atm.	-19.5°C (-3.1°F)
Density at -20°C (-4°F)	0.8153 g/ml
Vapor pressure at -19.5°C	1 atm.
Flash point	60°C (140°F) at a 40% solution
Solubility in water at 25°C	very soluble (up to 55%)
Conversions	1 ppm = 1.23 mg/m^3

6.2 Formation and Control Technology

Formaldehyde is the most prevalent aldehyde in 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. Formaldehyde emissions are controlled to roughly the same extent as total hydrocarbon emissions with a catalyst (Carey, 1987).

6.3 Emissions

6.3.1 Emission Fractions Used in the MOBTOX Emissions Model

Emission fractions for formaldehyde were developed using vehicle emission test data from various programs (Appendix B2). Formaldehyde emission fractions for different components included in the scenarios are included in Appendix B6.

The formaldehyde TOG emission fraction for LDGVs/LDGTs with three-way catalysts, running on baseline fuel, was based on data from 38 vehicles tested in four studies (Boekhaus et al., 1991a, 1991b, DeJovine et al., 1991, and Auto/Oil, 1990). The TOG fraction for LDGVs/LDGTs with three-way plus oxidation catalysts, running on baseline fuel, was based on data from 25 vehicles tested in eight studies (Urban, 1980a, 1980b, Sigsby et al., 1987, Stump et al., 1989, 1990, unpublished, Warner-Selph and DeVita, 1989, Boekhaus et al., 1991b, Auto/Oil, 1990). The TOG fraction for LDGVs/LDGTs with oxidation catalysts, running on baseline fuel, was based on data from 41 vehicles tested in eight studies (Urban, 1980a, Springer, 1979, Sigsby et al., 1987, Smith, 1981, Stump et al., 1989, 1990, Auto/Oil, 1990, Boekhaus et al., 1991a, Warner-Selph and Smith, 1991). The TOG fraction for LDGVs/LDGTs with no catalysts, running on baseline fuel, was based on data from 11 vehicles tested in four studies (Urban, 1981, Urban 1980a, Sigsby et al., 1987, and Warner-Selph and Smith, 1991). The LDDV fraction was based on data from 7 vehicles tested in two studies (Springer, 1977 and Springer, 1979). The HDDV and HDGV non-catalyst fractions were based on 13-mode data from two engines and one engine, respectively, tested in one study (Springer, 1979). To estimate the three-way fraction for HDGVs, the non-catalyst to three-way fraction for LDGVs/LDGTs was applied to the HDGV non-catalyst fraction.

To calculate TOG fractions for vehicles running on MTBE blends and 10% ethanol, adjustment factors were applied to the baseline emission fractions for each vehicle class/catalyst combination based on average percent change. The average percent change

numbers for vehicle class/catalyst combinations are contained in
Appendix B4.

It should be noted that percent change was calculated on a
vehicle by vehicle basis and the average of these percent changes
was then calculated for each vehicle class/catalyst combination.
When a draft memo was distributed by EPA describing the
methodology used to calculate emission fractions for this report
(EPA, 1992a), a comment was made in a review prepared by Systems
Applications International for the Motor Vehicle Manufacturers
Association (MVMA) (Ligocki, 1992) questioning this averaging
approach. The review pointed out that if a car had low total
mass emissions, but a large change in percent of a toxic, this
could result in an overestimate of the effect of this car on the
toxic level in the fleet, and an overestimate of the toxic level
in reformulated fuel relative to baseline. However, the
potential source of error resulting from this averaging technique
is diminished by a number of factors, including the fact that
data from cars exhibiting unreasonably large changes in toxic
levels were discarded. Also, the potential source of error would
be expected to affect only formaldehyde and acetaldehyde, since
benzene fractions were calculated from equations, and oxygenate
level has little effect on 1,3-butadiene. In any case, the MVMA
approach is not appreciably more accurate than the EPA approach
in predicting actual toxic fractions.

The 15% MTBE and 10% ethanol adjustment factors for
LDGVs/LDGTs with various catalyst technologies are summarized in
Table 6-2. Note that use of oxygenated fuels increases
formaldehyde emissions for all catalyst technologies. These 15%
MTBE numbers were estimated using data from Auto/Oil (1991) and
DeJovine et al. (1991) for LDGVs/LDGTs with three-way catalysts,
Auto/Oil (1991) for LDGVs/LDGTs with three-way plus oxidation and
oxidation catalysts, and Warner-Selph and Smith (1991) for
vehicles

**Table 6-2. 15% MTBE and 10% Ethanol Emission Fraction Adjustment
Factors for Formaldehyde.**

Vehicle Class	Catalyst Technology	15% MTBE Adjustment Factor	10% Ethanol Adjustment Factor
LDGV/LDGT	3-way	1.6746	1.4758
LDGV/LDGT	3-way + ox	1.2672	1.2288
LDGV/LDGT	oxidation	2.0244	1.2400
LDGV/LDGT	non-cat	1.5256	1.1034

with no catalysts. The 10% ethanol numbers were estimated using
data from Auto/Oil (1991), Warner-Selph and Smith (1991) and the
Colorado Department of Health (1987) for LDGVs/LDGTs with three-

way catalysts, the Colorado Department of Health (1987) for LDGVs/LDGTs with three-way plus oxidation catalysts, and Warner-Selph and Smith (1991) and the Colorado Department of Health (1987) for LDGVs/LDGTs with oxidation catalysts or no catalysts.

Since the average percent change was calculated for 15% MTBE (2.7% weight percent oxygen), and 11.0% MTBE (2.0% oxygen) was assumed for reformulated fuel and California standards components, average percent changes in the formaldehyde TOG fraction from 0 to 15% MTBE were multiplied by 2.0/2.7, the ratio of oxygen contents by weight. For HDGVs with three-way catalysts and with no catalysts, the same 15% MTBE and 10% ethanol adjustment factors were assumed as for LDGVs/LDGTs with the same catalyst technologies.

6.3.2 Emission Factors for Baseline and Control Scenarios

The fleet average formaldehyde emission factors as determined by the MOBTOX emissions model are presented in Table 6-3. When comparing the base control scenarios relative to 1990, the emission factor is reduced by 43% in 1995, by 61% in 2000, and by 66% in 2010. The expansion of reformulated fuel use in 1995 actually increases the emission factor, resulting in a 39% reduction relative to 1990. In 2000, the expanded control scenarios increase the emission factor slightly, when compared to the 2000 base control. In 2010, there is similarly little or no change from the 2010 base control for the expanded control scenarios.

6.3.3 Nationwide Motor Vehicle Formaldehyde Emissions

The nationwide formaldehyde metric tons are presented in Table 6-4. Total metric tons are determined by multiplying the emission factor (g/mile) by the VMT determined for the particular year. The VMT, in billion miles, was determined to be 1793.07 for 1990, 2029.74 for 1995, 2269.25 for 2000, and 2771.30 for 2010. When comparing the base control scenarios relative to 1990, the metric tons are reduced by 36% in 1995 and by 45% in 2000. Even though the emission factor continues to decrease from 2000 to 2010, this is more than offset by the large increase in VMT. As a result, metric tons in 2010 actually increase relative to 2000.

6.3.4 Other Sources of Formaldehyde

The onroad motor vehicle contribution to ambient formaldehyde levels contains both direct (primary) and secondary formaldehyde formed from photooxidation of VOC. It appears that roughly 33% of formaldehyde emissions may be attributable to motor vehicles. Section 6.5.2 contains a complete explanation of how this number is determined.

Table 6-3. Annual Emission Factor Projections for Formaldehyde.

Year-Scenario	Emission Factor g/mile	Percent Reduction from 1990
1990 Base Control	0.0412	–
1995 Base Control	0.0234	43
1995 Expanded Reformulated Fuel Use	0.0251	39
2000 Base Control	0.0162	61
2000 Expanded Reformulated Fuel Use	0.0166	60
2000 Expanded Adoption of California Standards	0.0168	59
2010 Base Control	0.0140	66
2010 Expanded Reformulated Fuel Use	0.0143	65
2010 Expanded Adoption of California Standards	0.0138	67

Table 6-4. Nationwide Metric Tons Projection for Formaldehyde.

Year-Scenario	Emission Factor g/mile	Metric Tons
1990 Base Control	0.0412	73,874
1995 Base Control	0.0234	47,496
1995 Expanded Reformulated Fuel Use	0.0251	50,946
2000 Base Control	0.0162	36,762
2000 Expanded Reformulated Fuel Use	0.0166	37,670
2000 Expanded Adoption of California Standards	0.0168	38,123
2010 Base Control	0.0140	38,798
2010 Expanded Reformulated Fuel Use	0.0143	39,630
2010 Expanded Adoption of California Standards	0.0138	38,244

Formaldehyde is produced in the U.S. by 13 chemical companies in 46 locations encompassing 18 states (EPA, 1991a). Formaldehyde is used in the manufacture of four major types of resins: urea-formaldehyde, melamine-formaldehyde, phenol-formaldehyde, and polyacetal resins. These resins are used in a wide variety of products, such as plywood, particle board, and counter tops. Formaldehyde is also used as a raw material in several synthetic organic chemical production processes, in the production of solid urea (used as a fertilizer, a protein supplement for animal feed, and in plastics), and in the production of ureaform fertilizers.

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.

In an attempt to determine the effects of actual formaldehyde exposure, an analysis was conducted by the EPA Office of Mobile Sources (EPA, 1987a) to determine the cancer risk attributable to indoor and outdoor sources of formaldehyde. The analysis consisted of three parts: (1) estimation of the U.S. population distribution and amount of time spent in each of several environments, (2) estimation of the formaldehyde concentrations in the various environments, and (3) estimation of unit risks.

This analysis determined that the largest single source of risk is the home environment, which accounted for 60 percent of the most likely number of malignant and benign tumors. The uncertainty in the formaldehyde concentrations experienced in this environment and the entire analysis is high. This is not an unexpected result as the nation, on average, spends nearly two-thirds of its time in non-mobile homes. The high exposure scenarios (mobile homes, high office exposures, high industrial exposures) which had high concentrations, accounted for just slightly more than ten percent of all tumors due to the small population involved. Mobile sources were calculated to only account for two to six percent of the total risk.

6.4 Atmospheric Reactivity and Residence Times

6.4.1 Gas-Phase Chemistry of Formaldehyde

As a result of its structure, formaldehyde has a high degree of chemical reactivity and good thermal stability. Formaldehyde is thus capable of undergoing a wide variety of chemical reactions. The major mechanisms of destruction in the atmosphere are reaction with hydroxyl radicals and photolysis.

Formaldehyde is present in emissions but is also formed by the atmospheric oxidation of virtually all reactive organic species. As a result, it is ubiquitous in the atmosphere.

The processes involved in transformation and residence times were previously discussed in Section 5.4 with the same information concerning benzene. For a more detailed explanation of the various parameters involved in these processes, please refer to Section 5.4. The information that follows on transformation and residence times has been mainly excerpted from a report produced by Systems Applications International for the EPA (Ligocki et al., 1991).

Since formaldehyde is formed by the oxidation of methane and biogenic hydrocarbons, it is ubiquitous in the atmosphere. The chemical system of NO and formaldehyde is the minimum system needed to generate urban-like photochemical ozone in air. This property has lead to the use of formaldehyde/NO smog chamber experiments for testing the inorganic reactions needed in smog mechanisms. On a per-carbon basis, formaldehyde has also been identified as the most important smog precursor in urban atmospheres (Smylie et al., 1990). Furthermore, formaldehyde is perhaps the most common secondary product from the atmospheric oxidation of all organic compounds.

6.4.1.1 Formation

Formaldehyde is formed from the atmospheric oxidation of many types of natural and anthropogenic (human produced) organic compounds. In remote areas, the slow oxidation of methane and the rapid oxidation of biogenic hydrocarbons such as isoprene produces a background concentration of about 0.6 ppb of formaldehyde during daylight hours (NRC, 1981). In urban areas, the oxidation of olefins such as ethene (C_2H_4) and propene (C_3H_6), and aromatics, such as toluene and xylene, produce formaldehyde. Dodge (1990) showed that the most important precursors for formaldehyde production are ethene, olefins, and higher aldehydes. Production of formaldehyde in the reaction of ethene with OH is particularly efficient because each mole of ethene reacts to produce 1.56 moles of formaldehyde. The atmospheric oxidation of methanol also produces formaldehyde.

6.4.1.2 Gas Phase Reactions

The reactions of formaldehyde with the OH radicals are responsible for a part of the destruction of formaldehyde in the atmosphere, while the reactions with HO_2, oxygen atoms, O_3, and Cl are not important in the ambient atmosphere.

An important destruction and radical production pathway is found in the photolysis of formaldehyde in the atmosphere. Three factors determine the rate of photolysis of a chemical species in the atmosphere (Jeffries and Sexton, 1987). The first factor is the amount of sunlight of a particular wavelength passing through the atmosphere at a given time. The second factor is the ability

of the chemical to absorb radiation. The third factor is the tendency of the molecule to form a particular set of products after it has absorbed a photon. The product of these three factors, integrated over the range of wavelengths of light present in the atmosphere, determines the photolysis rate for a given reaction.

A key property of formaldehyde photochemistry is its photolysis to form radical products. Under many conditions, the radicals from formaldehyde photolysis are the most important net source of smog generation. In addition, these radicals determine the chemical residence time of other toxic species. Formaldehyde absorbs UV radiation from below 290 nm to about 340 nm. Two pathways of photolysis are widely recognized: one pathway produces two relatively stable products, molecular hydrogen (H_2) and carbon monoxide (CO), whereas the other pathway produces two radicals, the formyl radical (HCO) and a hydrogen atom (H). Both of these radicals react quickly with atmospheric oxygen (O_2) to give hydroperoxyl radicals (HO_2) and CO.

6.4.1.3 Reaction Products

The oxidation of formaldehyde by OH proceeds primarily by H-atom abstraction, forming an HCO radical which rapidly reacts with atmospheric O_2 to form CO and HO_2 radicals. Production of formic acid (HCOOH) in the HCHO + OH reaction has been measured and found to account for only 2 percent of the product yield (Yetter et al., 1989). The HCHO + HO_2 reaction does produce formic acid; however, the rapid back-reaction precludes this from being a major formaldehyde transformation pathway. Therefore, the dominant carbon-containing product from all atmospheric formaldehyde reactions, including both photolysis pathways, is carbon monoxide.

6.4.2 Aqueous Phase Chemistry of Formaldehyde

In contrast to benzene and 1,3-butadiene, formaldehyde is quite soluble in water because it rapidly hydrates in solution to form a glycol ($CH_2(OH)_2$). Formaldehyde is readily incorporated into clouds and rain, and is an important species in cloud chemistry. The product of the aqueous-phase oxidation of formaldehyde is formic acid.

Formaldehyde is also interesting because of its participation in sulfur chemistry within clouds. Aqueous formaldehyde reacts with aqueous SO_2 (S(IV)) to form the stable adduct hydroxymethanesulfonate (HMS) (Munger et al., 1984). This reaction has been proposed to stabilize aqueous S(IV) against oxidation to sulfate (McArdle and Hoffmann, 1983).

Formaldehyde is formed in the aqueous phase by the oxidation of methanol (Jacob, 1986), and by the oxidation of HMS (Martin et al., 1989). However, the rate of in-cloud formation of formaldehyde is negligible relative to the rate of gas-phase formation.

6.4.3 Formaldehyde Residence Times

Residence times for formaldehyde were calculated by considering gas-phase chemical reactions with OH, NO_3, and HO_2, photolysis, in-cloud chemical reaction with OH, and wet and dry deposition. The reaction of aqueous formaldehyde with aqueous SO_2 was not considered. Although this reaction is fast and may be important to cloud chemistry as a whole, it does not destroy formaldehyde but merely binds it up as an adduct.

The results of the residence time calculation for formaldehyde are presented in Table 6-5. During the daytime, under clear-sky conditions, the residence time of formaldehyde is determined roughly equally by its photolysis and reaction with OH, leading to calculated residence times on the order of a few hours under summer, daytime, clear-sky conditions. The summer, daytime residence times for formaldehyde presented in Table 6-5 are comparable to a half-life of 2.6 h (equal to a residence time of 3.8 h) previously estimated for formaldehyde under polluted urban conditions (NRC, 1981). The residence time of formaldehyde in the atmosphere has also been estimated by EPA to range from 0.1 to 1.2 days (Cupitt, 1980), in good agreement with the values presented in Table 6-5.

In the presence of clouds, approximately 10 to 30 percent of the daytime chemical destruction of formaldehyde and 20 to 90 percent of the nighttime chemical destruction of formaldehyde was estimated to occur in clouds. The presence of clouds would also be expected to decrease the formation rate of formaldehyde; thus, cloud cover may actually decrease formaldehyde concentrations despite the predicted increase in residence time.

At night, formaldehyde is destroyed slowly because of its relatively slow rate of reaction with NO_3. The reaction of formaldehyde with HO_2 may be important at night under low NO_3 conditions, because the concentration of HO_2 radicals does not decrease at night as rapidly as does OH. However, since this reaction is reversible, the calculated residence time will be an upper bound. For the cases in which this reaction might be important, the residence times calculated with and without the HO_2 reaction are presented in Table 6-5 as a range of possible residence times.

Dry deposition may also be important as a removal mechanism for formaldehyde. Residence times due to dry deposition were estimated to range from 90 h under winter, nighttime conditions to

TABLE 6-5. Atmospheric residence time calculation for formaldehyde. All times are in hours unless otherwise noted.

	Los Angeles		St. Louis		Atlanta		New York	
	July	Jan	July	Jan	July	Jan	July	Jan
Clear sky – day	3	10	2	13	2	10	3	17
Clear sky – night	20-60*	90	30-250*	90	20-70*	80	20-110*	90
Clear sky – avg	4	20	3-4*	30	4	20	5	40
Cloudy – day	5	20	4	20	3	19	6	30
Cloudy – night	14-30*	70	14-30*	70	6-8*	70	18-50*	80
Cloudy – avg	7	30	6	40	4	30	9	50
Rainy – day	--**	3	3	0.8	2	1.6	3	0.8
Rainy – night	--**	1.4	3	0.3	3	0.7	3	0.5
Rainy – avg	--**	2	3	0.4	2	0.9	3	0.6
Monthly Climatological Average	5	18	4	18	4	14	7	17

*Range of values obtained with and without HCHO + HO$_2$ reaction (see text).
**Not calculated since July rainfall is zero for Los Angeles (Table 2-1).

800 h for summer, nighttime conditions. For the cases considered here, dry deposition was a minor removal mechanism except under winter, nighttime conditions. However, the deposition rate of formaldehyde to water surfaces is much greater than the deposition rate used in this calculation, and may be important to consider for urban areas located near oceans or major lakes and rivers.

Under wintertime conditions, the photolysis rate is not decreased by as large a factor as the OH radical concentration. Therefore, in the absence of precipitation, photolysis determines the winter, daytime formaldehyde residence time. Wet deposition, particularly under wintertime conditions, is an extremely effective removal mechanism for formaldehyde. Residence times for formaldehyde during winter rainy conditions range from fractions of an hour in colder climates to a few hours in warmer climates. Wet deposition accounts for roughly half of the monthly average removal of formaldehyde during the wintertime. It should be emphasized that this calculation assumes that the partitioning of formaldehyde in rain holds for all forms of precipitation. For colder climates where January precipitation is primarily in the form of snow, this assumption may not be appropriate.

As with benzene and 1,3-butadiene, the differences in formaldehyde residence time between cities within a season were not as large as the difference between seasons. The summer residence times are short in most cases, whereas the winter residence times are greater than one day in most cases. Thus, formaldehyde as well as 1,3-butadiene must be considered to be persistent in wintertime. Unlike the other two species, however, the effect of this longer winter residence time is difficult to assess for formaldehyde because of the importance of secondary formation. Rates of formation of formaldehyde will be roughly an order of magnitude slower in the wintertime. Thus, it is difficult to predict whether ambient concentrations of formaldehyde will increase or decrease in winter.

The major uncertainties in the residence time calculation for formaldehyde include the factor-of-two uncertainty in the OH radical concentration and the uncertainties in the deposition velocity. The uncertainty in the photolysis rate has only a minor effect on the overall uncertainty. The uncertainties associated with the NO_3 concentration and the NO_3 rate constant are less important for formaldehyde than for 1,3-butadiene because the NO_3 reaction with formaldehyde is much slower than the corresponding 1,3-butadiene reaction.

6.4.4 Limited Urban Airshed Modeling Results for Formaldehyde

The Urban Airshed Model (UAM) has been previously discussed in Section 5.4. Please refer to this section for details about

the model, its inputs, and modifications. Much of the
information below has been excerpted from reports conducted for
EPA by Systems Applications International (SAI) (Ligocki et al.,
1991, 1992).

Formaldehyde is an existing UAM species. The simulations
included three formaldehyde species; one each for mobile and
stationary-source primary formaldehyde and one for secondary
formaldehyde. Secondary formaldehyde is that produced by
atmospheric reactions. The full radical and product chemistry of
formaldehyde was retained, with the only change being that all
formaldehyde production was assigned to the secondary species
"FORM". Since formaldehyde is a product of the photooxidation of
virtually all atmospheric organic compounds, it was not possible
within the scope of this study to track secondary formaldehyde
formed from mobile-source precursors.

St. Louis

A time series plot of formaldehyde concentrations in the St.
Louis urban area is presented in Figure D-2 in Appendix D.
Mobile-source and stationary-source primary formaldehyde species
concentrations remain below 1 ppb throughout the simulation,
whereas secondary formaldehyde increases to more than 5 ppb in
the afternoon. The UAM simulation showed that formaldehyde
concentrations were about twice as high in the simulation with
chemistry as they were in the inert simulation, indicating that
formaldehyde is formed more rapidly than it is destroyed in urban
areas in the summertime. The concentration of formaldehyde would
be expected to decrease in the wintertime due to a decrease in
photolysis activity on formaldehyde precursors.

The contribution of mobile-source precursors to the
secondary formaldehyde concentrations can be estimated by
examining the mobile vs. stationary emissions of formaldehyde
precursors. For formaldehyde, the simulation demonstrated that
the component of the concentration due to primary formaldehyde
emissions is small (20 percent) relative to the component due to
secondary formation in the atmosphere. The fraction of this
secondary formaldehyde which formed from mobile-source precursors
is not known, but based on emissions of important formaldehyde
precursors, it appears to be 25-50 percent.

The comparison of simulated concentrations with ambient
measured concentrations showed good agreement for formaldehyde.

The formaldehyde photolysis rates used in the UAM for this
study were the higher (and currently accepted) values rather than
those used in the Carbon Bond Mechanism-IV (CBM-IV). Besides the
effect which changing the photolysis rate would have on
formaldehyde concentrations, there is the potential for secondary
effects on other species concentrations, such as 1,3-butadiene,

because the formaldehyde photolysis is a source of radicals and, ultimately, ozone. A sensitivity study was conducted in which the formaldehyde photolysis rate was increased by an additional 30%. The results from this simulation (with the base-case initial and boundary concentrations) showed, as expected, the higher photolysis rate caused a decrease in the predicted formaldehyde concentrations during the afternoon. This decrease was roughly 10% of the formaldehyde concentration. Because the higher formaldehyde photolysis rate caused increased production of reactive radicals, the 1,3-butadiene concentration decreased by about 3% in the mid-afternoon in this simulation as compared to the base case.

<u>Houston and Baltimore-Washington Area Simulations</u>

Simulations for the summer Baltimore-Washington area episode (Ligocki et al., 1992) 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. Overall, the increases and decreases in simulated ambient formaldehyde concentration approximately cancel out. 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. Maximum daily average formaldehyde concentration for the 1988 base scenario was 9.3 ppb. Motor vehicle-related formaldehyde accounted for about 35% of total formaldehyde emissions. Motor vehicle-related formaldehyde also accounted for about 10% of total simulated ambient formaldehyde on day 2 and 15% on day 3, based on the 1995 no motor vehicle scenario. 75 to 80 percent of this formaldehyde was secondary.

Summer Baltimore-Washington area simulations were in fairly good agreement with UATMP data for formaldehyde in the Baltimore part of the domain, but UAM-Tox overpredicted formaldehyde in the Washington part of the domain (although the overprediction was lower than for UAM).

In the winter 1988 base scenario, the maximum daily average formaldehyde concentration was 10.2 ppb, slightly higher than in summer. However, simulated concentrations throughout most of the domain were lower. Simulations for the winter Baltimore-Washington area episode resulted in slight increases in ambient levels of formaldehyde with the use of reformulated gasoline, on the order of 1-2 percent, with a primary formaldehyde increase and a secondary formaldehyde decrease. Motor vehicle-related formaldehyde emissions accounted for about 43% of total formaldehyde emissions. Motor vehicle primary formaldehyde emissions were about 30 percent higher with reformulated gasoline use. Motor-vehicle related formaldehyde accounted for about 12%

of the maximum simulated concentration, based on the 1995 no motor vehicle scenario. Comparison of simulated concentrations with measured concentrations in the Washington part of the modeling domain indicate that the model may underpredict winter formaldehyde concentrations.

For the summer 1987 base scenario in Houston, the maximum daily average formaldehyde concentration was 23.4 ppb. Motor vehicle-related formaldehyde accounted for about 19% of total formaldehyde emissions in the 1987 base scenario, and 6% of the maximum simulated concentration, based on the 1995 no motor vehicle scenario. Simulations for the summer Houston episode predicted slight increases in the simulated daily average concentration throughout most of the domain with use of reformulated gasoline. Comparison of simulated concentrations with measured concentrations suggest the model may overpredict formaldehyde concentrations in Houston.

6.5 Exposure Estimation

6.5.1 Annual Average Exposure Using HAPEM-MS

The data presented in Table 6-6 represent the results determined by the HAPEM-MS modeling that was described previously in Section 4.1.1. These numbers have been adjusted to represent the increase in VMT expected in future years.

The HAPEM-MS exposure estimates in Table 6-6 represent the 50th percentiles of the population distributions of exposure, i.e., half the population will be above and half below these values. High end exposures can also be estimated by using the 95th percentile of the distributions. According to the HAPEM-MS sample output for benzene, the 95th percentile is 1.8 times higher than the 50th percentile for urban areas, and 1.2 times high for rural areas. Applying these factors to the exposure estimates in Table 6-6, the 95th percentiles for urban areas range from 1.03 $\mu g/m^3$ for the 2010 expanded California standards scenario to 2.25 $\mu g/m^3$ for the 1990 base control scenario. The 95th percentiles for rural areas range from 0.37 to 0.82 $\mu g/m^3$, respectively.

6.5.2 Comparison of HAPEM-MS Exposures to Ambient Monitoring Data

As stated in section 4.1.2, four national air monitoring programs/databases contain data on formaldehyde. The Aerometric Information Retrieval System (AIRS), the Toxic Air Monitoring System (TAMS), the Urban Air Toxic Monitoring Program (UATMP), and the National Ambient Volatile Organic Compounds Data Base (NAVOC) all have a significant amount of data for formaldehyde. The urban exposure data for formaldehyde from all four databases are summarized in Table 6-7.

The AIRS data base contains data on formaldehyde for 1987 and 1988 (AIRS User's Guide Volume I-VII, 1989). The location and number of the sites varies between the two years. Referring back to the summary table in section 4.1.2, 14 sites monitored

Table 6-6.   **Annual Average HAPEM-MS Exposure Projections for Formaldehyde.**

Year-Scenario	Urban Exposure $\mu g/m^3$	Rural Exposure $\mu g/m^3$
1990 Base Control	1.25	0.68
1995 Base Control	0.78	0.42
1995 Expanded Reformulated Fuel Use	0.83	0.45
2000 Base Control	0.58	0.31
2000 Expanded Reformulated Fuel Use	0.60	0.32
2000 Expanded Adoption of California Standards	0.60	0.33
2010 Base Control	0.58	0.31
2010 Expanded Reformulated Fuel Use	0.59	0.32
2010 Expanded Adoption of California Standards	0.57	0.31

Table 6-7. Air Monitoring Results for Formaldehyde.

Program	Years	Ambient Data[a] $\mu g/m^3$	Estimated Motor Vehicle Contribution[b] $\mu g/m^3$
AIRS	1988	3.26	1.08
	1987	3.43	1.13
UATMP	1989	2.61	0.86
	1990	5.18[c]	1.71
TAMS	1987–89	2.15	0.71
NAVOC	1987	4.00	1.32

[a]Caution should be taken in comparing these numbers. The methods of averaging the data are not consistent between air monitoring databases. The sampling methodology is also inconsistent.

[b]The ambient data are adjusted to represent the motor vehicle contribution to the ambient concentration, which for formaldehyde is estimated to be 33%, based on emissions inventory apportionment and modeling.

[c]The 1990 UATMP is the only program which accounted for ozone interference in the measurement method.

formaldehyde in 1987 and 16 sites monitored it in 1988. All the
cities where the monitoring sites were located are listed below.

Birmingham, AL	Miami, FL
Jacksonville, FL	Atlanta, GA
Chicago, IL	St. Louis, MO
Louisville, KY	Baton Rouge, LA
Dearborn, MI	Detroit, MI
Port Huron, MI	Lansing/E. Lansing, MI
Cleveland, OH	Dallas, TX
Houston, TX	Burlington, VT

The average level of formaldehyde for 1987 (averaged equally by
the number of sites) was 2.79 ppb. In 1988, the average was 2.65
ppb.
Because the number of sites differs from year to year and the
number of samples taken at the various sites varies greatly, it
is misleading to make direct comparisons between these two
numbers. However, these numbers do provide a general idea of the
average amount of formaldehyde being emitted in a year.

     Looking at the AIRS data on a site by site basis for 1987,
Cleveland, Ohio had the highest average level of formaldehyde
among the 14 sites sampled (4.72 ppb) at a site located in a
central, urban, commercial area. Six samples were taken at this
site. Miami, Florida had the lowest average level of
formaldehyde in 1987 (1.43 ppb) with six samples taken in an
urban commercial area. In 1988, Louisville, Kentucky had the
highest average reading of formaldehyde (5.03 ppb) with 20
samples taken at a downtown urban commercial area. Port Huron,
Michigan had the lowest average reading (1.20 ppb) with 19
samples taken in a suburban residential area.

     Referring to the table in section 4.1.2., ten sites in the
Toxics Air Monitoring System (TAMS) monitored formaldehyde in the
following 4 cities.

> Boston (3 sites)
> Houston (3 sites)
> Chicago (3 sites)
> Seattle/Tacoma (1 site)

The period of time took place in various time periods between
1987 and 1989. The overall average for the 10 sites was 1.75
ppb. Because of the varying time intervals, it may be misleading
to make direct comparisons between the four cities involved, but
the measurements do give a general indication of the amount of
formaldehyde being emitted.

     One of the three TAMS sites in Chicago recorded the highest
amount of formaldehyde (2.27 ppb) Chicago also had the highest

measurement of formaldehyde averaged from all three of the sites located there (2.13 ppb). One of the sites in Houston had the lowest level of formaldehyde (1.23 ppb). Although it only had one site, Seattle/Tacoma had the lowest overall average of formaldehyde (1.54 ppb). Boston had the next lowest average with three sites (1.56 ppb).

The Urban Air Toxics Monitoring Program (UATMP) monitored the twelve cities listed below.

Baton Rouge, LA	Miami, FL
Chicago, IL	Pensacola, FL
Camden, NJ	St. Louis, MO
Dallas, TX	Sauget, IL
Ft. Lauderdale, FL	Washington, D.C.
Houston, TX	Wichita, KS

Washington, D.C. and Wichita, Kansas each had two monitoring sites, while the other 10 cities each had one monitoring site. At least 28 samples were collected at each site, except for Pensacola, Florida (7 samples). The comparatively larger number of samples taken in UATMP makes the data more reliable. The overall average formaldehyde level for all the samples was 2.13 ppb.

Averaged together, the two sites in Washington, D.C had the highest level of Formaldehyde ((3.77 + 3.09)/2 = 3.43 ppb). Twenty-eight samples were collected at one site and thirty were collected at the other site. Also averaged together, the two sites in Wichita, Kansas had the lowest level of formaldehyde ((1.46 + 1.40)/2 = 1.43 ppb).

In the 1990 Urban Air Toxics Monitoring Program (UATMP), 354 measurements of formaldehyde were taken at 12 sites. These sites were in the cities listed below.

Baton Rouge, LA	Chicago, IL
Camden, NJ	Houston, TX
Orlando, FL	Pensacola, FL
Port Neches, TX	Sauget, IL
Toledo, OH	Washington, D.C.
Wichita, KS	

The highest average was 6.44 $\mu g/m^3$ (7.92 ppb) at an urban commercial site in Washington, D.C.. Thirty samples were collected at this site. The lowest average was 1.83 $\mu g/m^3$ (1.49 ppb) at a suburban residential site in Houston, Texas. Twenty-six samples were collected at this site. The overall average of the averages for each site was 5.18 $\mu g/m^3$ (4.21 ppb). Ozone was removed from the ambient air collected in this program through the use of an ozone denuder. The use of an ozone denuder in the sampling system resulted in higher, but more accurate, reported

formaldehyde concentrations. Only the 1990 UATMP data will be used for the comparisons in this study.

The National Ambient Volatile Organic Compound (NAVOC) program only had one monitoring site in Philadelphia, Pennsylvania for formaldehyde. Thirty-six samples were collected and averaged equally. This resulted in an average of 3.25 ppb of formaldehyde.

HAPEM-MS assumes that the dispersion and atmospheric chemistry of formaldehyde is similar to CO. This assumption is not valid for a reactive compound like formaldehyde, which is both destroyed and formed in the atmosphere. For formaldehyde, HAPEM-MS would overestimate the primary (i.e., directly emitted) concentration in the atmosphere, since formaldehyde is more reactive than CO. On the other hand, HAPEM-MS would not account for, and thus underestimate, the secondary (i.e., atmospherically formed) formaldehyde, since HAPEM-MS does not account for atmospheric transformation. Since these two factors offset one another to some extent, it is possible that the HAPEM-MS results could still provide a reasonable estimate of the formaldehyde exposure from motor vehicles. Also, HAPEM-MS offers the advantage of being able to project future formaldehyde levels, based on emission data.

To test the reasonableness of using the HAPEM-MS modeling results, the HAPEM-MS results for 1990 are compared to ambient monitoring results for recent years. In order to make this comparison, the motor vehicle contribution to total ambient formaldehyde needs to be estimated. This requires first estimating the fractions of total ambient formaldehyde due to primary and secondary formaldehyde, and then estimating the motor vehicle contribution to primary and secondary formaldehyde.

The Five-City Study (EPA, 1989) and the UAM-Tox atmospheric modeling studies conducted by SAI (Ligocki et al., 1991, 1992) attempted to apportion the formaldehyde in the atmosphere into primary and secondary contributions. The Five City Study estimated that primary formaldehyde emissions account for about 40% of the total ambient formaldehyde. The UAM-Tox modeling studies determined that the concentration due to primary emissions is small, about 20%, relative to secondary formation of formaldehyde. The mid-point of these studies, 30%, was chosen to represent the contribution of primary formaldehyde emissions. Therefore, 70% was chosen to represent the contribution of secondary formaldehyde.

These studies also attempted to apportion a fraction of secondary formaldehyde formation in the atmosphere to motor vehicles. The Five City Study determined that motor vehicles are responsible for 35% of total VOC which contributes to secondary formaldehyde production. The St. Louis modeling study (Ligocki

et al., 1991) stated that the fraction of secondary formaldehyde formed from motor vehicle precursors is not known, but based on emissions of important formaldehyde precursors, it appears to be 25 to 50%. Based on this information, it was then estimated that motor vehicles account for approximately 35% of the secondary formaldehyde.

The 1987 EPA Air Toxics Report (Carey, 1987) attributed approximately 28% of the primary formaldehyde emissions to motor vehicles. This percentage is based on 1985 emissions data.

By using the numbers described above, the portion of formaldehyde in the ambient air that is attributable to motor vehicles was determined to be 33%. The fractions are: 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. The calculation is as follows:

.30(.28) + .70(.35) = 33% of total ambient formaldehyde from
(primary) (secondary)   motor vehicles

or: 8.4% + 24.5% = 33%

This estimate is higher than the estimates in the Houston and Baltimore-Washington Area UAM-Tox simulations (Ligocki et al., 1992). In Baltimore-Washington, motor vehicle-related formaldehyde accounted for about 10% of total simulated ambient formaldehyde on day 2 and 15% on day 3, while in Houston, motor vehicle- related formaldehyde accounted for about 19% of total simulated ambient formaldehyde. The estimate of 33% will be used in this study to represent the nationwide average percentage of ambient formaldehyde attributable to motor vehicles, while acknowledging the apparent area-to-area variations and the possibility that this may overestimate the motor vehicle contribution. Using this estimate, two approaches are used to compare the HAPEM-MS and the air monitoring results. The first approach attempted to adjust the HAPEM-MS number upward to account for secondary formaldehyde. If it is assumed that motor vehicles contribute 33% to ambient formaldehyde, 8.4% primary and 24.5% secondary (as determined in the equation above), then the ratio of secondary to primary is 2.92:1. If the primary formaldehyde is 1.25 μg/m^3 (from HAPEM-MS) then the secondary is 2.92×1.25 or 3.65 μg/m^3. When added to the primary formaldehyde result of 1.25 μg/m^3, the total is 4.90 μg/m^3 formaldehyde attributable to motor vehicles. This is inconsistent with the ambient monitoring data presented in Table 6-7 and thus was not used.

The second approach was to adjust the ambient air monitoring data to estimate the motor vehicle portion. This method applied the 33% formaldehyde from motor vehicles to each of the air monitoring results. This is presented as part of Table 6-7. Since the only program that accounted for the interference of ozone was the 1990 UATMP, only that ambient data will be used for this comparison. The resulting 1990 UATMP level is 1.71 $\mu g/m^3$. When the adjustment factor of 0.622 for the ambient motor vehicle levels, that was determined in Section 5.5.2 is applied, this exposure level becomes 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 reduce it to 1.06 $\mu g/m^3$ to agree with the ambient data. All analysis based on the HAPEM-MS ambient motor vehicle levels will have this factor applied. Adjusted urban, rural, and nationwide exposures are found in Table 6-8.

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.

6.5.3 Short-Term Microenvironment Exposures

The primary emphasis for formaldehyde exposure will be exposure in microenvironments that are enclosed, increasing the exposure to tailpipe emissions. These microenvironments include in-vehicle and parking garage exposure. The information contained in Table 6-9 is excerpted from two studies that have measured microenvironment exposures to formaldehyde. These two studies are the In-Vehicle Air Toxics Characterization Study in the South Coast Air Basin (Shikiya et al., 1989) and Air Toxics Microenvironment Exposure and Monitoring Study (Wilson et al., 1991). See the information in Section 4.2 for more details about the methodology, and Section 5.5.3 for a description of the studies.

Maximum microenvironment exposure levels of formaldehyde related to motor vehicles were determined in these studies to 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. This compares to ambient levels of 2.15 to 4.0 $\mu g/m^3$ determined through air monitoring studies and presented in Table 6-7. Since for the majority of the population these are short-term acute exposures to formaldehyde, the concern would be with non-cancer effects. No RfC has been developed by EPA though formaldehyde is a known irritant for the eyes, nose, and upper respiratory system at levels of 123 $\mu g/m^3$, and become widespread at concentrations near

3.7×10^3 µg/m^3 in humans. Exposures greater than 3.7×10^3 µg/m^3 are generally intolerable for more than short periods. Sensitive humans may detect effects at lower concentrations. Please see Section 6.8 for more information on non-cancer effects.

Due to more stringent fuel and vehicle regulations, short-term exposure to formaldehyde in these microenvironments is expected to decrease in future years.

Table 6-8.   Adjusted Annual Average HAPEM-MS Exposure Projections for Formaldehyde.

Year-Scenario	Exposure ($\mu g/m^3$)		
	Urban	Rural	Nationwide
1990 Base Control	1.06	0.57	0.95
1995 Base Control	0.66	0.35	0.58
1995 Expanded Reformulated Fuel Use	0.70	0.38	0.62
2000 Base Control	0.49	0.27	0.42
2000 Expanded Reformulated Fuel Use	0.51	0.27	0.44
2000 Expanded Adoption of California Standards	0.51	0.28	0.44
2010 Base Control	0.49	0.27	0.42
2010 Expanded Reformulated Fuel Use	0.50	0.27	0.46
2010 Expanded Adoption of California Standards	0.49	0.26	0.42

Table 6-9. Microenvironment Exposure to Formaldehyde ($\mu g/m^3$).

Scenarios	In-Vehicle		Service Station		Parking Garage		Office Building	
	Mean	Max.	Mean	Max.	Mean	Max.	Mean	Max.
SCAQMD Study[a] (Shikiya et al., 1989)	15.4	35.4	--	--	--	--	--	--
SCAQMD Study[b] (Wilson et al., 1991)	--	--	--	4.9	--	41.8	--	44.2

[a]The estimated sampling time period was 1.5 hours/round-trip.
[b]The measurements from this study are five minute levels.

6.6 Carcinogenicity of Formaldehyde and Unit Risk Estimates

6.6.1 Most Recent EPA Assessment

The information presented in Section 6.6.1 was obtained from EPA's Assessment of Health Risks to Garment Workers (EPA, 1987a), the Integrated Risk Information System (IRIS) (EPA, 1992b), the Motor Vehicle Air Toxics Health Information (Clement, 1991), as well as the primary sources cited in these documents. The carcinogenicity risk assessment for formaldehyde was last updated on IRIS in January 1992, and contains data published through 1987. The 1991 version of the formaldehyde risk assessment on IRIS does not contain any information that is not included in the 1987 risk assessment. The Office of Toxic Substances (OTS) prepared a formaldehyde risk assessment update in September 1990 (EPA 1990a, external review draft). This document is not yet final, and thus does not yet represent official Agency policy with regard to the risk assessment of formaldehyde. EPA's Science Advisory Board has reviewed this document, and has requested additional analyses. Therefore, the results presented in the OTS assessment are likely to change. Nevertheless, new issues discussed in this 1990 risk assessment update will be summarized in Section 6.6.2. Section 6.6.3 summarizes recent and ongoing research not included in the 1987 EPA risk assessment for formaldehyde. Some of this recent and ongoing research is discussed in the 1990 risk assessment, but is not yet part of the official Agency risk assessment for formaldehyde.

6.6.1.1 Description Of Available Carcinogenicity Data

Genotoxicity

Mutagenic activity of formaldehyde has been demonstrated in viruses, *Escherichia coli, Pseudomonas fluorescens, Salmonella typhimurium* (all three are bacteria), and certain strains of yeast, fungi, *Drosopohila* (fruit fly), grasshopper, and mammalian cells (Ulsamer et al., 1984). Formaldehyde has been shown to cause gene mutations, single strand breaks in DNA, DNA-protein crosslinks, sister chromatid exchanges, and chromosomal aberrations. Formaldehyde produces *in vitro* transformation in BALB/c 3T3 mouse cells, BHK21 hamster cells and C3H-10TI/2 mouse cells, enhances the transformation of Syrian hamster embryo cell by SA7 adenovirus, and inhibits DNA repair (Consensus Workshop on Formaldehyde, 1984).

Animal Data

According to EPA (1987b), the principal studies indicating formaldehyde may cause cancer in animals are Kerns et al. (1983) (the Chemical Industry Institute of Toxicology [CIIT] study), Albert et al. (1982) (the NYU study), Sellakumar et al. (1985), and Tobe et al. (1985). The carcinoma response in animals was similar for the four studies but the benign tumor response differed among the studies. EPA (1987b) concluded that there was "sufficient" evidence of carcinogenicity of formaldehyde in animals by the inhalation route based on increased incidence of a rare type of malignant cancer (i.e., squamous cell carcinoma) in rats and mice and in both sexes.

In the CIIT study, Fischer 344 rats and B6C3F1 mice (120/sex/group) inhaled 0, 2, 5.6 or 14.3 ppm formaldehyde, 6 hours/day, 5 days/week, for 24 months followed by 6 months of recovery (Kerns et al., 1983). Animals were sacrificed at 6 months, 12 months, and 18 months, while at 24 and 27 months, the number of animals sacrificed was unclear from the report. The study was terminated at 30 months. For the rats, mortality was significantly increased in the 14.3 ppm exposed animals after 12 months and in the 5.6 ppm exposed males after 17 months. At the end of the study period, squamous cell carcinomas in nasal cavities were reported in 51 of 117 male rats and 52 of 115 female rats at the high dose and in 1 of 119 male rats and 1 of 116 female rats at the intermediate dose. No tumors were observed at 0 or 2 ppm formaldehyde exposure. Polypoid adenomas (benign tumors) of the nasal mucosa were also seen in rats at all doses in a significant negative dose-related trend, although the incidence was significant only at 2 ppm formaldehyde. In the B6C3F1 mice, squamous cell carcinomas were observed in only two of the males exposed to 14.3 ppm formaldehyde. Although this increase was not significant, the occurrence of this carcinoma

type in mice was considered to be formaldehyde-related because this cancer is rare in mice.

In the Tobe study, male Fischer 344 rats who were exposed to 0 (methanol only), 0.3, 2, 3.3 or 15 ppm formaldehyde in aqueous solution methanol, 6 hours/day, 5 days/week for 28 months (Tobe et al., 1985). At the end of the 15-month exposure period, mortality in the high dose group was 88% while mortality was 60% in controls. Mortality was 32% in the low-dose group at 28 months. Squamous cell carcinomas occurred in 14 of 27 high-dose rats surviving past 12 months. No polypoid adenomas were observed but benign nasal papillomas were evident in formaldehyde-exposed animals.

Moreover, in the NYU study, after Sprague-Dawley rats inhaled 0 (air) or 14.2 ppm formaldehyde, 6 hours/day, 5 days/week, for a lifetime, there was a statistically significant elevation of the squamous cell carcinoma in 38 of 100 rats (Albert et al., 1982). Papilloma or polyps were detected in 10 of 100 exposed rats. The study was limited because only one exposure level was tested.

Sellakumar et al. (1985) exposed male Sprague-Dawley rats, 6 hours/day, 5 days/week for lifetime to 10 ppm HCl and to 14 ppm formaldehyde. The HCl and formaldehyde were administered simultaneously and separately, with an equal number of rats receiving an air control. HCl was administered to determine if tumor response was enhanced by an additional irritant effect or by the combining of formaldehyde and HCl to form bis-(chloromethyl)ether (BCME). Groups receiving formaldehyde alone or with HCl showed an increase in nasal squamous cell carcinomas; those without formaldehyde were free of carcinomas and other tumors, although rhinitis and hyperplasia were of comparable incidence.

Two other chronic inhalation studies examined the carcinogenicity of formaldehyde in upper and lower airways (Dalbey et al., 1982; Horton et al., 1963). However, nasal tissues were not systematically examined which limited the usefulness of these studies. Horton et al. (1963) exposed C3H mice to coal tar aerosol and/or formaldehyde at 40, 80, and 160 ppm for 1 hour/day, 3 days/week, for 35 weeks (4 weeks for the 160 ppm group). The study was limited because of insufficient animals surviving the first year, individual exposures were short, and complete histopathology was not reported. Dalbey (1982) exposed male Syrian golden hamsters (88-132/group) to 10 ppm formaldehyde, 5 days/week, for a lifetime. Results showed that there was no evidence of carcinogenic activity following exposure to 10 ppm formaldehyde in animals although survival was reduced relative to controls. EPA (1987a) found that the pathology evaluation in the study was less rigorous compared to

the Kerns et al. (1983) study and the study was limited because
only one dose was tested.

Rusch et al. (1983) carried out a 6-month toxicity study in
6 male cynomolgus monkeys, 40 F344 rats, and 20 Syrian golden
hamsters with 22 hours/week exposure to three levels of
formaldehyde with corresponding controls. The highest dose
tested was 2.95 ppm. The short duration of the assay, the small
sample sizes, and, possibly, the low concentrations tested,
limited the sensitivity of the assay to detect tumors. In the
highest dose group in both rats and monkeys, incidences of
squamous metaplasia/hyperplasia of the nasal turbinates were
significantly elevated.

Furthermore, several recently published drinking water
studies provide additional suggestive evidence that formaldehyde
is carcinogenic following oral exposure as well. The tumor-
promoting potential of formaldehyde in mouse skin, rat trachea,
and rat stomach has been also recently been demonstrated. The
recent carcinogenicity studies referred to above are summarized
in Section 6.6.3.3.

Human Data

EPA reviewed only cohort or case-control studies because
these studies yielded the best qualitative information for
evaluating causality. There was a total of 28 studies but many
of them had limitations that could potentially influence the
conclusions, and therefore will not be addressed in this section.
Of these studies, 11 were of chemical or industrial workers and 7
were of medically-related professionals (e.g., morticians,
pathologists). The other 10 were case-control studies examining
workers with sinonasal cavity and pharyngeal cancers. Only six
studies had enough data to evaluate exposure-response effects;
these are the studies that will be reviewed in this section. Of
these six, two cohort studies (Blair et al., 1986, 1987; Stayner
et al., 1988) and one case-control study (Vaughan et al., 1986)
were well conducted and specifically designed to detect small to
moderate formaldehyde-associated human risks. These three
studies were discussed in the IRIS cover sheet. According to EPA,
weaknesses inherent in the human studies in general included: 1)
inference of formaldehyde levels from industrial hygiene data,
2) concurrent exposures to other chemicals which prevented
determination of specific exposure levels, 3) small sample size
for cohorts, 4) small number of site-specific deaths, and 5)
insufficient follow-up.

Blair et al. (1986) conducted the largest occupational
exposure study that has been published to date (see Section
6.6.3.4 for more details on the followup). They reported a
significant increase in lung and nasopharyngeal cancer in a

cohort study at 10 industrial sites. The authors concluded that there was little evidence showing an association between lung cancer and formaldehyde exposure because the risk did not increase with exposure intensity or cumulative exposure. The observation of nasopharyngeal tumors support similar findings in animals. EPA considered the lung and nasopharyngeal cancer mortality data "meaningful" despite the lack of significant trends. EPA also believed that misclassification of exposure and categorization of deaths into 4 exposure levels (although not specified by EPA 1987a) may account for the lack of a dose-response relationship.

A cohort study by Stayner et al. (1985) found buccal cavity tumors in formaldehyde-exposed garment workers. The SMR was highest in workers with the longest duration of employment (exposure) and follow-up period (latency). There were no other details reported.

A significant association was reported between nasopharyngeal cancer and people living 10 years or more in a "mobile home" built in the 1950's to 1970's (Vaughan et al., 1986). The walls and flooring in mobile homes are generally made out of plywood or some sort of wood composite material that contains urea-formaldehyde resins or adhesives. Exposure to formaldehyde in residents of mobile homes occurs when the formaldehyde in these resins and adhesives offgas as the material ages.

The studies by Olsen et al. (1984), Hayes et al. (1986), and Hardell et al. (1982) reported significant excesses of sinonasal cancer in individuals exposed to both formaldehyde and wood-dust. However, only the first two studies controlled for wood-dust exposure. The detection limits in both studies exceeded corresponding expected excesses in the incidence of sinonasal tumor and, therefore, no significant excesses were likely to have been observed (EPA, 1987a). Acheson et al. (1984) compared excess mortalities due to lung cancer in one of six formaldehyde resin producing plants in England. Only borderline significance was observed. The authors concluded that the increases in mortality from lung cancer were not related to exposure since the elevation was not statistically significant when compared with local lung cancer rates. However, EPA (1987b) believed that the risk was sufficiently increased to enable this study to be used for corroboration. Other studies, Pattanen et al. (1985), Bertazzni et al. (1986), and Blair et al. (1986, 1987) also indicated that lung cancer also may be associated with occupational exposure to formaldehyde. The risk associated with sinonasal cancer appeared to be specific for the histologic type, squamous cell carcinoma. The relative risks observed for upper respiratory tract cancers in all the reviewed studies ranged from just above 1.0 (a risk of 1.0 implies no association between exposure and disease) to 3.0, depending on the site.

There were 19 studies that indicated the possibility that observed leukemia and neoplasms of the brain and colon may be associated with formaldehyde exposure; however, the biological support for these findings has not yet been demonstrated.

6.6.1.2 Weight-of-Evidence Judgment of Data and EPA
Classification

EPA has classified formaldehyde as a Group B1, probable
human carcinogen under its Guidelines for Carcinogen Risk
Assessment. This is based on limited epidemiological evidence
and sufficient evidence of carcinogenicity in animal studies. In
addition, this evidence is supported by mutagenic activity in
various *in vitro* test systems.

The CIIT inhalation study (Kerns et al., 1983) in rats is
considered the primary study for estimating unit risk. The study
was well designed, well conducted, multiple doses (4 exposure
levels) were included, and sufficient animals were tested. The
malignant tumor data (i.e., squamous cell carcinoma in nasal
cavity) in the Kerns et al. (1983) study were used for estimating
risk since the response in treated rats was definite and
unequivocal in both males and females and there was an increasing
dose-related trend. Furthermore, similar malignant tumor types
were evident in all rat and mouse inhalation studies with
formaldehyde exposure. EPA also believes that the appearance of
benign tumors in the Kerns et al. (1983) study contributes to the
qualitative weight-of-evidence that formaldehyde may pose a
carcinogenic hazard.

The other animal inhalation studies had limitations that
prevented their use for quantitative risk assessment. Data from
Sellakumar et al. (1985), the NYU (Albert et al. 1982) and Tobe
et al. (1985) studies were also considered by EPA for unit risk
estimates. The Tobe et al. (1985) study gave supportive evidence
in the same strain of rats but was not used for primary risk
estimation because a tumor response was observed only at the high
dose. The Albert et al. (1982) study was considered less
appropriate because it contained only one nonzero exposure
concentration. The Kerns et al. (1983) study also suggests
carcinogenicity at the high dose but the response was limited or
not significant.

A degree of uncertainty was due to the different responses
of animals to formaldehyde exposure. Only the rats showed
statistically significant numbers of neoplasms. The mice only
had two carcinomas (Tobe et al., 1985), but the response was
complicated by the fact that mice were able to reduce their
breathing rate to a greater extent than rats. According to EPA,
the mice with tumors received 14.3 ppm formaldehyde, a dose that
approximates that which rats received at 5.6 ppm. Thus, on a
"dose" received basis, the rats and mice may be equally sensitive
to formaldehyde.

The epidemiological data had limited exposure information, insufficient sample size, and concurrent exposures for risk estimate determination.

6.6.1.3   Data Sets Used For Unit Risk Estimates

The consequences of inhalation exposure to formaldehyde has been studied in rats, mice, hamsters, and monkeys. The principle evidence comes from positive studies in both sexes of two strains of rats (Kerns et al., 1983; Albert et al., 1982; Tobe et al., 1985) and males of one strain of mice (Kerns et al., 1983), all showing squamous cell carcinomas. The primary data set is the squamous cell carcinomas of the nasal turbinates in Fischer 344 rats from the CIIT study (Kerns et al., 1983). This data set used to calculate the cancer risk estimate for formaldehyde is summarized in Table 6-10.

Three epidemiological studies are also used as supporting evidence. Two cohort studies (Blair et al., 1986, 1987; Stayner et al., 1988) and one case-control study (Vaughan et al., 1986a,b) were well-conducted and specifically designed to detect small to moderate increases in formaldehyde-associated human risks. These were discussed previously in Section 6.6.1.1b. Primates and rats have been shown to respond similarly to formaldehyde exposure with
respect to the development of nasal tumors. In any case, ppm is considered equivalent across species, so a species scaling factor was needed.

6.6.1.4   Dose-Response Model Used

Since low level exposure can not be measured in animal or human studies, several models are possible for low-dose extrapolation. Data were inconsistent regarding a linear or nonlinear relationship between formaldehyde exposure and carcinogenicity. Because of the absence of biological evidence on the mechanism of action for formaldehyde, the linearized multistage procedure was chosen as the default model as specified by EPA guidelines (see Appendix F for a complete explanation), although various other models were presented for comparative purposes. They found that only the one-hit model produced higher risk estimates (about 10-fold higher).

6.6.1.5   Unit Risk Estimates

The inhalation unit cancer risk is 1.3×10^{-5} $(\mu g/m^3)^{-1}$ or 1.6×10^{-2} $(ppm)^{-1}$ based on squamous cell carcinoma in F344 rats. The unit risk should not be used if the air concentration exceeds 800 $\mu g/m^3$. The major contributor to the uncertainty in the risk estimate using the multistage model is due to the steep dose response observed in the CIIT study. There was a 50-fold increase in the number of tumors compared to a 2.5-fold increase

in the dose level; 0 tumors at 2 ppm, 2 at 5.6 ppm, and 103 at
14.5 ppm. Other uncertainties are the marked nonlinearity of the
response and the different responses observed in the tested
animals.

Table 6-10. Summary of Data Set Used to Calculate Unit Risk Estimate for Formaldehyde.

Source	Test Animal	Tumor Type	Administered Dose (ppm)	Human Equivalent Dose (mg/kg/day)	Tumor Incidence
Kerns et al. (1983)[a]	F344 rats, male and female, combined	Squamous cell carcinoma	0	0	0/156
			2	2	0/159
			5.6	5.6	2/153
			14.3	14.3	94/140
Kerns et al. (1983)[b]	F344 rats, male and female, combined	Squamous cell carcinoma	0	0[c]	0/156
			2	15.3	0/159
			5.6	70.8	2/153
			14.3	318	94/140

[a]Data set used in EPA 1987b cancer risk assessment
[b]Data set used in EPA 1990 cancer risk assessment
[c]Delivered dose expressed as pmol/mg/DNA/day calculated using rat DPX data and   adjusted for average daily dose

There is a wide range between MLE and upper bound estimates of risk (ranging from 1-5 orders of magnitude) at different exposure levels showing the statistical uncertainty of the estimates that were generated from highly non-linear data. For example, using the 1987 unit risk, at an exposure level of 3 ppm (3,685 $\mu g/m^3$) for 36 hours/week for 40 years (typical occupational exposure conditions), the upper bound estimate of lifetime cancer risk is 6×10^{-3} and the maximum likelihood estimate of risk is 6×10^{-4}, whereas a 10-year exposure to 0.07 ppm (86 $\mu g/m^3$) formaldehyde (believed to be the home/environment background upper limit in conventional homes), the upperbound estimate of risk is 1.0×10^{-4} and the maximum likelihood estimate of risk is 6.0×10^{-11}. However, the predictive power of the model is not significantly disturbed by slight perturbations of the data.

6.6.2 Other Views and Unit Risk Estimates

This section presents alternate views and/or risk assessments for formaldehyde. These are summarized in Table 6-11.

Office of Toxic Substances 1991 Formaldehyde Draft Report

The OTS (EPA, 1991b) risk assessment for formaldehyde concluded that recent animal studies confirm the previous findings of an increased incidence of squamous cell carcinomas of the nasal cavity in rats exposed by inhalation and a steep dose-response curve. 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 neoplasms. Many of the recent studies used in EPA (1991b) are discussed in Section 6.6.3.

In the OTS risk assessment update concerning the epidemiological data, it was concluded that when the risk assessment is examined in context of the previously reviewed studies, the human studies released since 1987 support the conclusions drawn by EPA in its 1987 document and do not alter the evaluation that 'limited' evidence exists for an association between formaldehyde and human cancer. Collectively, however, the data do not conclusively demonstrate a causal relationship.

The 1991 update goes on to describe that 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. The recent epidemiological studies referred to above are summarized in Section 6.6.3.4.

The OTS update also concurred with the weight-of-evidence evaluation presented in the 1987 risk assessment. Based on

Table 6-11.   Comparison of Formaldehyde Inhalation Unit Risk Estimates.

Source	Classification	Cancer Unit Risk Estimate $\mu g/m^3)^{-1}$ Upper Bound[a]
EPA (1987)	Group B1[b]	1.3×10^{-5}
EPA (1991b)	Group B1	6×10^{-7} [c]
		8×10^{-6} [d]
IARC (1987)	Group 2A[e]	$-$[f]
CARB (1992b)	Probable Human Carcinogen	6.0×10^{-6}
OSHA (1987)	Potential Occupational Carcinogen	2.64×10^{-2} [g]

[a]MLEs have not been presented because EPA does not generally compare MLEs based on animal data because of the high variability associated with these numbers. Therefore, they are of little value.
[b]Group B1 = Probable Human Carcinogen
[c]Calculated using monkey DPX data
[d]Calculated using rat DPX data
[e]Group 2A = Probable Human Carcinogen
[f]IARC did not conduct a quantitative risk assessment
[g]Upper bound estimates calculated for risk to 100,000 workers exposed to 1 ppm for 45 years. It should be noted that OSHA used the maximum likelihood estimate, and not the upper bound, for regulatory purposes.

sufficient animal evidence (mainly nasal cancers in rats and mice), limited human evidence associating nasal and nasopharyngeal cancer with formaldehyde exposure, and other key evidence including structure-activity considerations, the known genotoxic activity of formaldehyde, and the ability of formaldehyde to injure cells and affect cell division, OTS concurred that formaldehyde should be classified as a probable human carcinogen (Group B1).

The OTS updated risk assessment calculated new cancer unit risks for formaldehyde. However, the data set used as the basis for the unit risks is the same as that used by EPA in 1987; squamous cell carcinomas of the nasal turbinates in Fischer 344 rats from the CIIT study (Kerns et al., 1983). The data set used to calculate the cancer risk estimate for formaldehyde is summarized in Table 6-9.

In the OTS update, EPA chose to continue with the linearized multistage model to calculate the new unit risk estimates because there is insufficient evidence, especially with respect to mechanism, to warrant a departure from this model.

These unit risk estimates have been modified in the 1991 OTS update to reflect new information regarding dose-rate effects and the use of DNA binding data as an intracellular dosimeter for formaldehyde.

Since the 1987 risk assessment, data have become available regarding nasal DNA binding of formaldehyde in the form of DNA-protein cross-links (DPX), and the quantitation of these DPX levels (see Section 6.6.3.2 for a discussion of DPX). OTS concluded that these new data support the use of DPX as an internal measure of formaldehyde dose, and has used the data of Casanova et al. (1989) in F344 rats and Heck et al. (1989) in Rhesus monkeys to calculate internal formaldehyde doses to be used in the revised risk assessment. Specifically, the rat DPX data were input into the linearized multistage model, and the risk to humans was then calculated by applying monkey DPX data to the resulting equation because it was believed that the actual risk to humans lies somewhere between the risk estimates derived using only the rat or the monkey DPX data. The modified unit risk estimates also used a different method to adjust the calculated delivered doses to average daily doses to be input into the linearized multistage model. Generally, experimental exposure rates are multiplied by a factor of 5/7 and 6/24 (to reflect the fact that exposure only occurred for 6 hours/day, 5/days/week) to convert to continuous lifetime daily average exposure. In the case of formaldehyde, OTS considered this to be inappropriate. There is evidence to support the hypothesis that dose rate (or the concentration of formaldehyde reaching the target tissue) is more critical in determining the severity of the toxic effects, such as cell proliferation and histological

changes, than average exposure concentration. Therefore, OTS
adjusted the DPX-derived dose levels by a factor of only 5/7
because this approach acknowledges the importance of a possible
dose-rate effect by not averaging the exposure and expected DPX
over the course of 24 hours. Tumor incidences were not induced
by a continuous exposure regiment, and these tumor incidences
should be linked as nearly as possible with the exposure levels
which caused them.

Based on the results of many pharmacokinetics studies, EPA
has concluded that most of the objections expressed in the expert
panel's report have been adequately addressed and that the use of
DPX as the surrogate dose for risk estimates appears appropriate
with the following reservations. The DPX data were obtained
following a single exposure to formaldehyde (acute), whereas the
carcinogenic bioassay was a chronic (2-year) study. The
different exposure conditions may have little effect on DPX
yields at low concentrations, where the normal morphology of the
nose is unaltered by exposure, but it may have a major effect at
high concentrations. This is due to the proliferation of the
squamous cells which may have very different metabolic
abilities, formaldehyde uptake, and detoxifying mechanisms than
the epithelial cell examined in the DPX experiments. Indeed,
recent studies at CIIT on the formation of DPX in rats exposed
subchronically to 15 ppm of formaldehyde indicate that such
effects can occur (Casanova and Heck, 1991).

Another reservation regarding the use of DPX is that the
role of DPX, if any, in the induction of nasal cancer is not
completely understood. This problem is relatively insignificant
if DPX are used only as a dosimeter, and the linearized
multistage model is used to estimate risk. However, it could
become more important if the DPX were given a specific
mechanistic role in a biologically-based model.

A final reservation is that the current DPX data should not
be used to make assumptions about species differences in
sensitivity (response) since the necessary mechanistic
information is lacking.

The resulting modified unit risk factors (UCL) calculated by
OTS are 6×10^{-7} $(\mu g/m^3)^{-1}$ (7×10^{-4} $[ppm]^{-1}$) using the monkey-based DPX
data and 8×10^{-6} $(\mu g/m^3)^{-1}$ (1×10^{-2} $[ppm]^{-1}$) using the rat-based DPX
data. The MLE's are 8.1×10^{-8} $(\mu g/m^3)^{-1}$ (1×10^{-4} $[ppm]^{-1}$) and 8.1×10^{-6}
$(\mu g/m^3)^{-1}$ (1×10^{-2} $[ppm]^{-1}$), respectively. The unit risk estimate
calculated using the rat DPX data is lower than that calculated
in EPA (1987a) presented above by an approximately 25-fold
difference, 4-fold of which is due to the difference in
continuous exposure correction used and 6-fold of which is due to
the use of the rat

DPX data. The risk estimate calculated using the monkey DPX data
is approximately 9 times lower than the 1987 risk estimate.[1]

International Agency for Research on Cancer (IARC)

IARC has classified formaldehyde as a Group 2A carcinogen.
A Group 2A carcinogen is defined as an agent that is *probably*
carcinogenic to humans. This classification is based on limited
evidence for carcinogenicity in humans and sufficient evidence
for carcinogenicity in animals (IARC 1982, 1987).

IARC reviewed the available human data and concluded that
these studies do provide some evidence that occupational exposure
to formaldehyde is associated with an excess of various forms of
cancer. Cancers that occurred in excess in more than one study
are: Hodgkin's disease, leukemia, and cancer of the buccal
cavity and pharynx (particularly nasopharynx), lung, nose,
prostate, bladder, brain, colon, skin, and kidney (IARC 1982b,
1987c). However, in many of these studies, actual exposure to
formaldehyde is unknown.

IARC concluded that the available animal data provide
sufficient evidence of the carcinogenicity of formaldehyde.
These data consist of inhalation studies in one strain of mice
and two strains of rats. No unit risk estimate was determined by
IARC.

Motor Vehicle Manufacturer's Association (MVMA)

MVMA contracted with Environ Corporation to 1) describe the
means for conducting an assessment that incorporates all
scientific information pertinent to the question of risk for
formaldehyde (Environ, 1986); and 2) to evaluate the risk
assessment issues in EPA's technical report "Air Toxics Emissions
from Motor Vehicles" (Environ, 1987). It is important to note
that neither document is actually a risk assessment of
formaldehyde, i.e., no alternative unit risk estimates were
developed; rather, they are critiques of existing risk
assessments (the 1986 document critiques OSHA's 1985 risk
assessment of formaldehyde [50 FR 50412-40499], and the 1987
document critiques EPA's risk assessment of motor vehicle air
toxics (Carey, 1987) and descriptions of elements that should be
considered in a comprehensive risk assessment of formaldehyde.

Environ (1986) points out that any risk assessment of
formaldehyde must take into consideration the following issues:

---

[1]The MLEs calculated in the 1991 updated risk assessment have been presented
here, but it is explicitly stated in this document that EPA does not generally
compare MLEs based on animal data because of the high variability associated with
these numbers. Therefore, they are of little value.

(a) the mechanism of action of formaldehyde
(b) the relationship between the magnitude and duration of exposure to formaldehyde and the 'target-site dose' of the proximate carcinogen
(c) the relative importance of the genotoxic activity of formaldehyde compared to its other biological effects in determining risk
(d) the shape of the dose-response curve at dose levels below the experimental range
(e) the relationship between risk in rodents and risk in humans.

With regard to mechanism of action and its role in risk assessment, Environ (1986, 1987) describes several mechanisms that have been proposed to account for the carcinogenic effects of formaldehyde. These include:

(1) Chemistry and metabolism. This takes into account the nonlinear relationship between the concentration of formaldehyde in the air and the level of DNA adducts to establish a relationship between dose and response.

(2) Physiological effects. Animals exposed to high levels of formaldehyde reduce their rate and depth of breathing, thus resulting in a reduction of inhaled dose. In addition, formaldehyde reduces the protective flow of mucus over the surface of the nasal passages (nasal epithelium), thus resulting in the slower removal of dissolved proportions of inhaled formaldehyde. This information should be used to adjust the dose used in modeling the dose-response relationship to more accurately reflect the target-site dose.[2]
(3) Effect on proliferation of respiratory epithelium. High concentrations of formaldehyde have been shown to cause cellular degeneration and abnormal stimulation of cell replication that attempts to replace the dead cells (regenerative hyperplasia) of the nasal epithelium. This effect may contribute to the carcinogenic action of formaldehyde. This proliferation does not occur at exposure levels below 6 ppm, therefore, this mechanism would not likely contribute to carcinogenesis at low levels.

Environ agrees with EPA and OSHA that the data set that provides the best estimate of the relationship between dose and response for formaldehyde is the CIIT rat study (Kerns et al.,

---

[2]OTS, in their updated risk assessment for formaldehyde (EPA 1990), acknowledges that these two mechanisms (reduced respiration rate and mucociliary clearance), may alter the dose of formaldehyde that reaches the target tissue. However, they concluded that not enough is known to quantitate the amount that these two mechanisms may alter the actual delivered dose of formaldehyde.

1983). However, Environ (1986) cautions that all data sets
should be used to provide a range of risk estimates to provide a
better indication of the uncertainty of the estimates.

Environ (1986, 1987) contends that EPA's and OSHA's use of
the linearized multistage model for low dose extrapolation
overestimates the carcinogenic risk of formaldehyde. They
suggest that there is some evidence to indicate that formaldehyde
may be a threshold carcinogen. This, together with the fact that
the dose-response data for formaldehyde are not linear, led
Environ to conclude that linear extrapolation of responses that
occur following exposure to high doses to predict responses at
low doses may not be entirely valid. They suggest that if a
model that better fits the data is used, for example, a non-
linearized multistage model (i.e., a five-stage or six-stage
model) or a Weibull model, then the predicted risks at low dose
levels are orders-of-magnitude lower than those predicted using
the linearized multistage model.

California Air Resources Board (CARB)

CARB (1992b), like EPA and IARC, has concluded that
formaldehyde is a probable human carcinogen. CARB (1992b) has
performed an assessment of the carcinogenic risk of formaldehyde
using the CIIT rat data (Kerns et al., 1983) in the linearized
multistage model. However, their assessment differs from EPA
(1987a) in the following two ways:

(1)   The present approach uses the rate of binding of
      formaldehyde to DNA in the nasal lining of the rat, in
      order to characterize the dose rate. The EPA in its
      1987 risk assessment decided to use administered dose
      (inhalation exposure) rather than estimated tissue dose
      for risk estimation purposes because their reviewers
      did not consider the tissue data then available for
      their assessment to be adequate.

(2) The present approach uses three different scaling
     factors to extrapolate the equivalent dose rate from
     rats to humans. EPA (1987a) did not specifically
     discuss the issue of scaling to extrapolate from
     rodents to humans for formaldehyde.

The UCL for unit risk for lifetime exposure calculated by
CARB (1992b) using the methods and assumptions described above is
7.0×10^{-3} ppm^{-1} (6.0×10^{-6} [µg/m^3]$^{-1}$). The two differences in
methodology (i.e., target-site dose and scaling factor) result in
a doubling of the upper confidence limit (UCL) on the unit risk
calculated by EPA. CARB (1992b) did not calculate MLEs for
formaldehyde. CARB also calculated a range of UCL for unit risks
based on the three scaling factors and two measures of exposure

to formaldehyde by inhalation. This range is 0.3×10^{-3} ppm^{-1} to 40×10^{-3} ppm^{-1} (0.25×10^{-6} to 33×10^{-6} [μg/m^3]$^{-1}$).

Occupational Safety and Health Administration (OSHA)

OSHA published a final rule for occupational exposure to formaldehyde in 1987, in which they concluded that formaldehyde should be regarded as a "potential occupational carcinogen" (OSHA, 1987). The 1987 final rule differs only slightly from the 1985 proposed rule mentioned above. With regard to the adequacy of the available human data, in the 1985 proposed rule, OSHA has not relied on the epidemiologic results to assess risk of lifetime exposure of workers to formaldehyde. However, in 1987, OSHA stated that the evidence regarding human risk of exposure to formaldehyde has become substantial.

OSHA has also selected the CIIT rat study (Kerns et al., 1983) as the basis for its risk assessment for formaldehyde. OSHA, like EPA and CARB, selected the linearized multistage model to calculate lifetime risk of exposure to formaldehyde. Unlike CARB, OSHA chose not to use a scaling factor and also chose not to use the pharmacokinetic model relating DNA-formaldehyde adducts to external exposure dose to estimate target-site dose. OSHA (1987) concluded that the pharmacokinetic model, as it presently exists, is greatly limited by the scarcity of data identifying DNA protein- formaldehyde cross-links, and it cannot be presumed to predict overall human cancer risk resulting from long-term repeated exposures to formaldehyde.

As a result of the data set and low-dose extrapolation model chosen, and the assumptions made with regard to scaling and correct estimation of dose, OSHA (1987) calculated the following lifetime risk of cancer per 100,000 workers:

Exposure level (ppm)	Maximum Likelihood Estimate (MLE)	Upper Confidence Limit (UCL)
3	71	834
2	11.4	534
1	0.6	264
0.5	0.03	132
0.1	0.001	26

Universities Associated for Research and Education in Pathology (UAREP)

This panel reviewed the same body of literature (UAREP, 1988) as IARC (1987c) and EPA (1987) using a metanalysis approach. The UAREP panel commented only on the determination of causality. Unlike the IARC and EPA, the UAREP panel did not attempt to

categorize the epidemiological evidence other than whether causality could be established. The panel concluded that a causal relationship has not been established for cancer at any site. In addition, the panel noted that if such a causal relationship exists, the excess risk must be small. The panel noted elevated risks in nasopharyngeal cancer with formaldehyde exposure in several studies, and concluded that the evidence for causality was weak. With respect to observed excesses in nasal cavity and sinus cancers and any formaldehyde exposures, several studies suggest an approximate doubling of the risk, while other studies could not exclude an elevation of the size. Overall, the panel concluded that the presence or absence of an association could not be firmly established. With respect to lung cancer, the panel thought the evidence was not consistent and did not indicate a causal association with formaldehyde exposure.

For sites which are not directly in contact with formaldehyde, the panel stated that the rapid metabolism of formaldehyde makes it unlikely that formaldehyde is the agent responsible for increased brain tumors observed in the group that used formalin. For the excesses in leukemia observed in several studies of anatomists, embalmers, and pathologists, the panel concluded that socioeconomic factors influencing diagnosis may explain the elevations observed in these groups.

"Epidemiological Evidence on the Relationship Between Formaldehyde Exposure and Cancer" (Blair et al. 1990b)

Blair et al. (1990b) performed a metanalysis on essentially the same body of literature as reviewed by IARC (1987c), UAREP (1988), and EPA (1987) with the addition of more recent findings, either published or in press. From this analysis, the authors found excesses in deaths due to cancers of the nasal cavities, nasopharynx, lung, and brain, and due to leukemia. The investigators believed that a causal role for formaldehyde was most probable for cancers of the nasopharynx and, to a lesser extent, the nasal cavities. Blair et al. (1990b) derived their support for the conclusion from statistically significant increases in nasal cavity cancer risk, from the apparent specificity of the association with squamous cell carcinoma, and from histological changes in the nasal mucosa seen in industrial studies which correspond to those observed in the rat.

The investigators further concluded that the excesses in lung cancer were difficult to interpret due to inconsistencies among studies and lack of trends with either level or duration of exposure. In addition, the excesses of leukemia, brain, and colon cancer observed among professionals were most likely not related to formaldehyde since similar excesses were not observed among the industrial workers.

"Quantitative Cancer Risk Estimation for Formaldehyde" (Starr, 1990)

Starr (1990) calculated cancer risks based on the DPX (DNA protein cross-link) experiments of Casanova et al. (1989) in rats, and of Heck et al. (1989) in monkeys. Using the linear multistage model, Starr fit a "three-stage" model using rat DPX levels interpolated from the DPX experiment to correspond to the bioassay exposures of Kerns et al. (1983). Predicted risks corresponding to 0.1, 0.5, and 1.0 ppm formaldehyde in air, based on the DNA-binding data for both rats and monkeys are reproduced in the table above. Starr also did not address the non-nasal DPX observed in monkeys in making his calculations. Starr concluded that point estimates of human risk (also called maximum likelihood estimates, or MLEs) based on DPX in monkeys were lower than those based on airborne concentrations to rats (the basis of EPA's 1987 unit risk), by as much as 1,500,000-fold.

Comparison of Risk Estimates form Starr (1990), Upper Bounds and Point (Maximum Likelihood) Estimates[a]

Air Conc. (ppm)	Upper Bound and (MLE) Estimates of Risk From Several Formaldehyde Exposure Measures		
	Rat/1983[b]	Rat/1989[c]	Monkey/1989[d]
0.1	2 E-4[e] (3 E-7)	7 E-5 (2 E-9)	8 E-6 (2 E-12)
0.5	8 E-4 (3 E-5)	4 E-4 (3 E-7)	4 E-5 (3 E-10)
1.0	2 E-3 (3 E-4)	1 E-3 (6 E-6)	1 E-4 (1 E-8)

[a] Continuous lifetime average exposure adjustment not used.
[b] Kern et al. (1983) exposure concentrations (ppm).
[c] DNA-protein cross links (pmol/mg DNA) from Casanova et al. (1989).
[d] Using the 1989 rat DNA=binding data for the dose-response relationship, and the Heck et al (1989) DNA-protein cross-links for delivered dose at 0.1, 0.5, and 1.0 ppm.
[e] $2\ E-4 = 2 \times 10^{-4}$

Starr calculated a MLE risk of 3×10^{-7} based on the air concentration of 0.1 ppm administered to rats, while the corresponding MLE human risk based on monkey DPX was 2×10^{-12}. The differences between upper bounds on risk were less dramatic, the largest difference being 25-fold between an upper bound rat dosimetry-based risk of 2×10^{-4} and upper bound monkey dosimetry-based risk of 8×10^{-6} at an air concentration of 0.1 ppm.

6.6.3 Recent and Ongoing Research

6.6.3.1 Genotoxicity

Recent studies on the genotoxicity of formaldehyde have demonstrated the covalent binding and induction of DNA strand breaks, induction of chromosomal aberrations *in vitro*, co-mutagenesis of formaldehyde and x-rays in *Drosophila*, cytotoxicity and mutagenicity in human lymphocytes and *Salmonella* and sister chromatid exchange in anatomy students exposed to embalming solution (Bogdanffy et al., 1987; Casanova and Heck, 1987; Casanova et al., 1989; Craft et al., 1987; Crosby, 1988; Dresp and Bauchinger, 1988; Dowd et al., 1986; Ecken and Sobels, 1986; Liber et al., 1989; Heck and Casanova, 1987; Heck et al., 1989; Schmid et al., 1986; Snyder and Van Houten, 1986; Yager et al., 1986). These studies have been reviewed by EPA (1990a) and it was concluded that they added nothing new or substantially different to what was written in EPA (1987a) regarding the genotoxic effects of formaldehyde.

6.6.3.2 Pharmacokinetics

Recent work on the pharmacokinetics of formaldehyde has focused on the validation of measurement of DNA-protein adducts (DPX) as internal dosimeters of formaldehyde exposure. In other words, the binding of DNA to protein to which formaldehyde is bound to form a separate entity that can be quantified may serve as a means to measure the amount of formaldehyde that is present inside a tissue. 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 (such as the protection of underlying cells by the mucociliary apparatus) that act to limit the amount of formaldehyde that reaches cellular DNA. At issue is the rebuttal by EPA and the Science Advisory Review Board (summarized in EPA 1987a) of the assertion that DPX measurements could be used in quantitative cancer risk assessments as an indication of intracellular dose (Starr and Buck, 1984). The rebuttal was based on EPA and the Science Advisory Review Board's belief that inadequate evidence was presented demonstrating that the method used to measure DPX was valid, the measurement of DPX as an intracellular dosimetric marker was adequate, and the results obtained in acute studies that measured DPX could be extrapolated to the chronic exposure situation.

6.6.3.3 Carcinogenicity - Animal Studies

Recent studies examining the carcinogenicity of formaldehyde in animals have further studied the characteristics of nasal

tumor induction as a result of inhalation of formaldehyde. Morgan et al. (1986) mapped the specific location of the nasal squamous cell carcinomas that were observed in rats in the study by Kerns et al. (1983). The authors proposed that in addition to regional exposure, local tissue susceptibility may be an important determinant for distribution of formaldehyde-induced neoplasms.

In the CIIT study (Kerns et al., 1983), nasal tumors were induced by formaldehyde at concentrations that also induced severe degenerative, hyperplastic, and metaplastic changes in the nasal epithelium, suggesting that cytotoxicity and/or increased cell proliferation may have had a role in tumor induction. Recent studies by Woutersen et al. (1989) and Feron et al. (1988) and an ongoing study by Monticello and Morgan (1990) support the association between cytotoxicity and cell proliferation and tumor induction at exposures of 10 to 20 ppm, 6 hours/day, 5 days/week at exposures ranging from 4 weeks to 28 months. The study by Woutersen et al. (1989) more directly examined the effect of tissue damage on the tumorigenic response of formaldehyde. These authors found that external sources of damage to the nasal epithelium could enhance the tumorigenic response of Wistar rats to formaldehyde.

One explanation for the increase in tumor induction in areas of tissue damage proposes that nasal defense mechanisms may be irreparably damaged in such areas. For example, the mucociliary apparatus has been proposed to trap and remove formaldehyde in the mucus layer before it has a chance to reach underlying cells (Zwart et al., 1988). The tissue damage may prevent adequate functioning of the mucociliary apparatus. Both *in vitro* and *in vivo* studies (Morgan et al., 1983, 1986) have shown that there is a clear dose-dependent effect of formaldehyde on the mucociliary apparatus of the rats. In addition to tissue damage, exposure to high concentrations of formaldehyde has been suggested to interfere with the protective function of mucus. A recent study by Bogdanffy et al. (1987) examined [14C]-formaldehyde binding to nasal mucus from rats and a human volunteer and found the formaldehyde bound to albumin within the mucus. These authors postulated that formaldehyde binding to mucus may alter the physical characteristics of mucus and lead to mucostasis. This would allow formaldehyde to penetrate to the submucosal cell layer. In humans, nasal mucociliary function was inhibited by exposure to 0.3 ppm formaldehyde for 1 to 5 hours (Anderson and Molhave, 1983). It is also known that formaldehyde at levels below 1 ppm can be detected in the olfactory region of the human nose, indicating that formaldehyde is not completely removed by the mucus layer, even at low concentrations.

Another explanation for the increase in tumor induction in areas of tissue damage is that cytotoxicity may increase cell proliferation thereby increasing the amount of single-stranded

DNA available for damage by formaldehyde. Alternatively, cytotoxicity may in some way promote the carcinogenic response in formaldehyde-initiated cells.

Cell proliferation in response to formaldehyde has been observed in human tissues and the monkey, as well as in the rat. In studies by Klein-Szanto et al. (1989) and Ura et al. (1989) human tracheobronchial epithelia were transplanted into deepithelialized rat tracheas. A concentration-dependent proliferative response similar to that observed in the rat was observed when the tracheas were exposed *in vivo* to devices that slowly released formaldehyde. Exposure of monkeys to 0 or 6 ppm of formaldehyde for 1 or 6 weeks resulted in an 18-fold increase in cell proliferation in formaldehyde exposed animals (Monticello et al., 1989).

Although increased cell proliferation has been observed in a number of studies in which nasal tumors have been induced, stimulation of cell proliferation does not appear to be sufficient to cause tumors. For example, in the study by Monticello and Morgan (1990), although proliferation and inflammation were observed at the same doses at which carcinogenicity was observed, proliferation and inflammation were not observed only at those sites at which tumors developed. Also, Zwart et al. (1988) found that exposure of formaldehyde produced patterns of cell proliferation that were not consistent with carcinogenic patterns. For example, after 3 days of exposure to 3 ppm, increases in cell proliferation were observed in regions with a high tumorigenic response; but, after 13 weeks the proliferation in these areas was slightly less than in controls. These acute (Swenberg et al., 1983; Zwart et al., 1988) and chronic (Monticello and Morgan, 1990) studies have demonstrated that there is a correlation between cytotoxicity and cell proliferation induced by formaldehyde in the rat nasal epithelium and that the cell proliferation rate is concentration-dependent.

The role of concentration versus total dose (i.e., the total dose that an animal receives is the exposure concentration multiplied by the duration of exposure) in the response of respiratory tissue to formaldehyde was examined in two studies by Wilmer et al. (1987, 1989). In both studies the Wistar rats were exposed to formaldehyde on a continuous and intermittent basis and the response appeared to be more dependent on concentration than on total dose.

A number of recent studies have also examined the carcinogenic potential of formaldehyde by the oral route. Exposure of rats to 0.2% formaldehyde (0.001 to 0.25%) in the drinking water produced squamous cell papillomas in the forestomach (Takahashi et al., 1986) and an increase in gastric neoplasms (squamous cell carcinomas, adenocarcinomas, and

leiomyosarcomas [a tumor of the smooth muscle tissue]) (Soffritti
et al., 1989). A slight increase in leukemia was also observed
in treated animals, but the significance of this finding was not
addressed. In contrast, no increase in tumors was observed in
Wistar rats exposed to formaldehyde (0.002 to 0.5%) in the
drinking water (Til et al., 1989; Tobe et al., 1989) but
hyperplasia and inflammation of the forestomach and glandular
stomach were reported. These results provide suggestive evidence
of carcinogenicity of formaldehyde by the oral route.

A number of recent studies have also examined the tumor
promotion potential of formaldehyde. Using rat tracheal
explants, Cosma and Marchok (1987) examined the effects of
formaldehyde, benzo[a]pyrene, and the combination of these agents
on the induction of carcinogenesis. Tracheal explants are
tracheal cells taken from a rat and grown in tissue culture
outside of the animal. Carcinogenicity was quantified as the
number of growth altered populations observed per tracheal
explant. Formaldehyde treatment (0.2%) twice weekly for 4.5
months by itself produced only 0.25 altered populations per
explant, benzo[a]pyrene produced 2.37 altered populations per
explant, and pretreatment with benzo[a]pyrene followed by
formaldehyde treatment produced 7.83 populations per explant,
indicating the tumor promotion potential of formaldehyde. Also,
in a skin painting experiment by Iversen (1986), hr/hr Oslo
strain mice were treated with 51.2 µg of the tumor initiator
dimethylbenz[a]anthracene (DMBA). Nine days later, a group of
these mice was treated with 200 µl of 10% formaldehyde twice a
week for 60 weeks. Although the incidence of tumors was similar
in DMBA treated animals both with and without formaldehyde
treatment (approximately 38%), the time of appearance of the
tumors was significantly reduced in those mice treated with both
the formaldehyde and DMBA. However, a later experiment using
SENCAR mice (bred for maximal sensitivity to carcinogens) found
no change in tumor induction when mice that had been pretreated
with 51.2 µg of DMBA were treated twice weekly with 4%
formaldehyde (Iversen, 1988). Thus, in some tissues formaldehyde
may have tumor promoting potential.

6.6.3.4 Carcinogenicity - Epidemiological Studies

Since the 1987 EPA carcinogenicity assessment, a limited
number of new epidemiologic studies and reanalyses of previous
studies have been published. Many of the reanalyses have
examined the results of the largest study that has been published
to date (Blair et al., 1986, 1987). This study examined the
mortality experience of 26,561 workers employed in a total of 10
plants known to use formaldehyde. The estimated 8-hour time-
weighted-average exposure to formaldehyde fell into five
categories: trace, <0.1 ppm, 0.1-0.5 ppm, 0.5-<2.0 ppm, and >2.0
ppm based on job category. Blair et al. (1986, 1987) reported
that workers exposed to >0.1 ppm formaldehyde had an elevated

rate of lung cancer; but, that no increase in lung cancer incidence could be correlated with increases in exposure. Also, these authors reported that workers with exposure to both formaldehyde and particulates had a dose-related elevated rate of nasopharyngeal cancer.

The increase in nasopharyngeal cancer was reanalyzed by Collins et al. (1988) and was reported to be confined to only one of the 10 plants studied. Also, the dose-related increase in nasopharyngeal cancer originally reported by Blair et al. (1986, 1987) was not seen if only those workers with simultaneous exposure to particulates and formaldehyde were considered. Blair et al. (1986, 1987) had grouped exposure to formaldehyde and particulates irrespective of whether the exposures had occurred simultaneously. Although Collins et al. (1988) indicated that these data showed a lack of an association between formaldehyde exposure and increased incidence of nasopharyngeal cancer, EPA (1990a) reevaluated the data using a Poisson trend statistic and found a significant trend for increased nasopharyngeal cancer with increasing formaldehyde exposure.

Robins et al. (1988) reanalyzed the lung cancer data using a method developed to correct for the existence of a healthy worker effect. These authors confirmed the lack of an association between lung cancer and increased formaldehyde exposure. Sterling and Weinkam (1988 1989) also reanalyzed the lung cancer data from Blair et al. (1986 1987) using methods that would reduce the influence of a healthy worker effect. This included using a time-integrated exposure score and comparison of internal high and low exposure groups. The report published in 1988 contained calculation errors and was amended in 1989. The 1989 paper reported a significant increase in the odds ratio (OR)[3] for lung cancer in those over age 40 (40-55 yr, OR = 11.10, 95% CI[4] = 6.45 to 1926; 55+ yr, OR = 67.44, 95% CI = 35.59 to 127.59). A significant increase in the odds ratio for lung cancer for hourly workers was also observed (OR = 1.61, 95% CI = 1.12 to 2.31). Also, a significant trend for increased lung cancer incidence was reported with increased cumulative exposure, although none of the cumulative exposure levels was associated with a significant increase in lung cancer incidence (<0.1 ppm-yr, OR = 1.0; 0.1-0.5 ppm-yr, OR = 1.21, 95% CI = 0.84 to 1,74; 0.5-2 ppm-yr, OR = 1.19, 95% CI = 0.78 to 1.83; 2+ ppm-yr, OR = 1.56, 95% CI = 0.95 to 2.56).

---

[3]The odds ratio (OR) is an estimate of the relative risk (RR). It is a measure of association between the characteristic and disease in a case-control study. Relative risks (i.e., odds ratios) that are >1 imply an association between the characteristic and the disease.

[4]The confidence interval (CI) is the investigator's assurance that the sample selected is one of 95% (for a 95% confidence interval) of all samples that will provide a correct statement based on the interval.

Blair et al. (1990) disputed the results reported by Sterling and Weinkam (1989) based on the observation that Sterling and Weinkam had grouped all respiratory cancer deaths rather than examining only lung cancer deaths. However, reanalysis using only lung cancer deaths lowered the calculated risks, but did not affect the overall conclusions of Sterling and Weinkam. Further analysis of the association between lung cancer and formaldehyde exposure by Blair et al. (1990) revealed that lung cancer mortality was elevated in workers with formaldehyde and particulate exposure from the production of resin and molding compounds and that exposure to melamine, urea, phenol, or wood dust in these operations may have accounted for the increases in lung cancer that were attributed to formaldehyde exposure.

Two new case control studies reported the cancer mortality of persons occupationally exposed to formaldehyde. In the study by Gerin et al. (1989), an elevated odds ratio of 2.3 (95% CI = 0.9 to 6.0) was determined for persons with adenocarcinoma of the lung and long-duration, high exposure to formaldehyde. The odds ratio appeared to increase between those with long-duration low level exposure (OR = 0.8, 95% CI = 0.3 to 1.3), those with long-duration, medium level exposure (OR = 0.8, 95% CI = 0.4 to 1.6), and those with long-duration high level exposure, but this was not statistically analyzed. In the study by Roush et al. (1987), an odds ratio of 2.3 (95% CI = 0.9 to 6.0) was determined for persons with nasopharyngeal cancer and occupational exposure to formaldehyde at high levels 20 years prior to death. The odds ratio was statistically significant for those persons over 68 years of age (OR = 4.0, 95% CI = 1.3 to 12.0). No such increase was observed for persons with sinonasal cancer and occupational exposure to high levels of formaldehyde 20 years prior to death (OR = 1.5, 95% CI = 0.6 to 3.1).

Another case-control study (Partanen et al., 1990) examined possible associations between formaldehyde and respiratory cancer of 136 respiratory cancers among 7307 male Finnish woodworkers. These men were employed in jobs in particleboard, plywood, construction carpentry, furniture manufacturing, and glue manufacturing plants, and in sawmills. After accounting for a minimum latency period of 10 years, smoking, and vital status at the time of data collection, an elevated odds ratio for respiratory cancer (OR = 1.4, 90% CI = 0.4 to 4.1) was found with exposure to cumulative formaldehyde (either dustborne or as gas) (≥3 ppm-months). When further analyzing upper respiratory cancer and lung cancer separately, the odds ration for upper respiratory cancer becomes OR = 2.4 and that of lung cancer OR = 0.9. Partanen et al. (1990) believed these results are compatible either with chance or with a weak elevated risk mainly due to cancers of the upper respiratory organs.

Hayes et al. (1990) conducted a proportional mortality ratio (PMR) study of embalmers and funeral directors in the U.S.

Statistically significantly elevated proportions of deaths were found from a variety of causes, specifically cancers of the nasalpharyngeal, colon and lymphatic and hematopoietic systems. There was also a significant increase in ischemic heart disease. The authors believed the apparent elevated proportions of death due to nasalpharyngeal cancer and leukemia were consistent with previous observations in formaldehyde-exposed industrial cohorts and other studies of professionals.

Other related studies examining cancer mortality among workers exposed to formaldehyde include a population-based case control study by Linos et al. (1990) that observed an increase in follicular non-Hodgkin's lymphoma and acute myeloid leukemia among embalmers and funeral directors. Also, Malker et al. (1990) found a significant increase in nasopharyngeal cancer in workers in fiberboard plants and among book binders (both are subject to formaldehyde exposure). A study of 9.365 leather tannery workers reported 1 death due to squamous cell carcinoma of the nasal cavity (0.4 expected) and attributed the death to 18 years of exposure to a variety of chemicals, including chrome and formaldehyde (Stern et al. 1987).

Histochemical analyses of biopsies taken from nasal tissues of workers exposed to formaldehyde revealed precancerous lesions. Holmstrom et al. (1989) observed significant changes in the middle turbinate of workers exposed to well-defined levels of formaldehyde. However, similar changes were not observed in nasal tissues of workers exposed to formaldehyde and wood dust. Boysen et al. (1990) also found a significant increase in the degree of metaplasia in the nasal cavity of workers exposed to formaldehyde.

These new studies support the previous conclusion by EPA (1987a) that limited evidence of an association between formaldehyde exposure and nasopharyngeal and, possibly lung, cancer in humans exists. No definitive causal relationships are demonstrated in the new studies.

6.7 Carcinogenic Risk for Baseline and Control Scenarios

Table 6-12 summarizes the annual cancer incidences for all the scenarios. 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 50% in 2010, which is actually an increase when compared to 2000. The reduction in emissions are 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 vehicles modifications.

From Table 6-12 it can also be observed that the expanded use
scenarios provide no decrease in the cancer cases and, in one
scenario, the cancer cases increase slightly. This is generally
due to the fact that increased use of oxygenates in gasoline will
increase formaldehyde emissions. The HAPEM-MS exposure model
estimates exposure based on direct emissions of formaldehyde. As
discussed in Section 6.5.2, however, the use of oxygenates in
gasoline is expected to change the reactivity of the emissions.
It is probable that secondary (i.e., atmospherically formed)
formaldehyde could be reduced with the use of oxygenates. As a
result, the cancer risk estimates given in Table 6-11 should be
considered conservative estimates.

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.

Table 6-12. Annual Cancer Incidence Projections for Formaldehyde.[a,b]

Year-Scenario		Emission Factor g/mile	Urban Cancer Cases	Rural Cancer Cases	Total Cancer Cases	Percent Reduction from 1990	
						EF	Cancer
1990	Base Control	0.0412	37	7	44	–	–
1995	Base Control	0.0234	24	4	28	43	36
1995	Expanded Reformulated Fuel Use	0.0251	25	5	30	39	32
2000	Base Control	0.0162	18	3	21	61	52
2000	Expanded Reformulated Fuel Use	0.0166	19	3	22	60	50
2000	Expanded Adoption of California Standards	0.0168	19	3	22	59	50
2010	Base Control	0.0140	19	3	22	66	50
2010	Expanded Reformulated Fuel Use	0.0143	20	4	24	65	45
2010	Expanded Adoption of California Standards	0.0138	19	3	22	67	50

[a]Projections have inherent uncertainties in emission estimates, dose-response, and exposure.
[b]Cancer incidence estimates are based on upper bound estimates of unit risk, determined from animal studies. EPA has classified formaldehyde as a Group B1, probable human carcinogen based on limited epidemiological evidence and sufficient evidence in animal studies.

6.8 Non-carcinogenic Effects of Inhalation Exposure to Formaldehyde

Since the focus of this report is on the carcinogenic potential of the various compounds, the noncancer information will be dealt with in a more cursory fashion. No attempt has been made to synthesize and analyze the data encompassed below. Also, no attempt has been made to accord more importance to one type of noncancer effect over another. The objective is to research all existing data, describe the noncancer effects observed, and refrain from any subjective analysis of the data.

Irritation of the eyes (lacrimation and increased blinking) and mucous membranes is the principal effect of exposure to low concentrations (0.05-2.0 ppm) of formaldehyde observed in humans (NRC, 1981). Other human upper respiratory effects associated with acute formaldehyde exposure include a dry or sore throat, and a tingling sensation of the nose. These effects are frequently seen following exposure to 1-11 ppm (NRC, 1981). Sensitive humans may detect effects at lower concentrations (CARB, 1991b). Tolerance to eye and upper airway irritation may develop after 1-2 hours exposure, but symptoms may return if exposure is resumed following an interruption (NRC, 1981). Nasal mucocillary clearance system effects (loss of cilia, keratosis, mild dysplasia) have been reported in humans at concentrations of 0.1 ppm (Edling et al., 1985), and following chronic exposure to undetermined concentrations (NRC, 1981). Forty percent of formaldehyde-producing factory workers reported nasal symptoms such as rhinitis, nasal obstruction, and nasal discharge following chronic exposure (Wilhelmsson and Holmstrom, 1987). In persons with bronchial asthma, the upper respiratory irritation caused by formaldehyde can precipitate an acute asthmatic attack, sometimes at concentrations below 5 ppm (Burge et al., 1985); formaldehyde exposure may also cause bronchial asthma-like symptoms in nonasthmatics (Hendrick et al., 1982; Nordman et al., 1985). However, it is unclear whether asthmatics are more sensitive than nonasthmatics to formaldehyde's effects (EPA, 1990a). Lower airway irritation, characterized by cough, wheezing, and chest tightness, has been reported often in people chronically exposed to 5-30 ppm formaldehyde, and has been observed in concentrations below 1 ppm (EPA, 1987a). However, acute exposure did not cause lower airway symptoms in medical students in an anatomy laboratory (Uba et al., 1989). Neither lower airway irritation (cough, chest symptoms, and dyspnea [labored or difficult breathing]) nor decrements in pulmonary functioning were more frequently reported among asthmatics than among nonasthmatics (Uba et al., 1989). Formaldehyde concentrations exceeding 50 ppm may cause severe lower respiratory tract reactions, in which not only the airways, but also the alveolar tissue is involved. This acute injury includes pneumonia, bronchial inflammation, pulmonary edema, and, at concentrations exceeding 100 ppm, death may occur in sensitive individuals (NRC, 1981). Pulmonary effects, as measured by pulmonary function tests, have not been reported consistently across studies. Overall, chronic decrements in lung function do not appear to be associated with formaldehyde exposure (Witek et al., 1987; Sauder et al., 1987), although small transient

decreases have been noted (Sauder et al., 1986; Horvath et al., 1988).

Immune stimulation may occur following formaldehyde exposure, although conclusive evidence is not available. Patterson et al. (1986) demonstrated the presence of IgE antibodies against formaldehyde-human serum albumin conjugates and human serum albumin (HSA). IgE (immunoglobulin gamma E) is a protein antibody produced by cells of the lining of the respiratory and intestinal tract. It appears that formaldehyde is capable of inducing respiratory tract allergy, but data are lacking on induction concentrations (Burge et al., 1985; Nordman et al., 1985). Central nervous system effects such as dizziness, apathy, inability to concentrate, and sleep disturbances have been reported in a variety of studies following inhalation exposure in humans (EPA, 1987a). However, in general, formaldehyde's effect on the CNS is not clearly defined (Consensus Workshop, 1984).

With regard to the developmental toxicity of formaldehyde, menstrual disorders were reported among 47.5% of women occupationally exposed to formaldehyde vapors from urea-formaldehyde resins, with dysmenorrhea (pain in association with menstruation) being the most common disorder (Shumilina, 1975). There have been several animal inhalation studies conducted to assess the developmental toxicity of formaldehyde. The only exposure-related effect noted in a study conducted by Martin (1990) observed a decrease in maternal body weight gain at the high-exposure level but no adverse effects on reproductive outcome or the fetuses that could be attributed to treatment were noted. In another study conducted by Sallenfait et al. (1989), reduced fetal weight was noted following exposure of pregnant Sprague Dawley rats to 20 or 40 ppm formaldehyde on gestations days 6-20. No effects on embryonic or fetal lethality, or in the external, visceral, or skeletal appearance of the fetuses were noted. In Ulsamer et al. 1984, other effects such as increased duration of gestation and body weight of offspring, microscopic changes in the liver, kidneys, and other organs of fetuses from exposed dams, and decreased levels of nucleic acid in the testes of exposed males have been reported.

Acute and subchronic inhalation exposure of various laboratory animals to low (<1 ppm) or moderate (10-50 ppm) concentrations of formaldehyde vapor is known to cause increased airway resistance, decreased sensitivity of the nasopalatine nerve (a nerve that innervates both the nose and the palate), irritation of the eyes and respiratory system, and changes in the hypothalamus (a part of the brain that is important in controlling certain metabolic activities such as maintenance of water balance, sugar and fat metabolism regulation of body temperature and secretion of hormones). Exposure to high concentrations (>100 ppm) of formaldehyde vapor can cause salivation, acute dyspnea, vomiting, cramps, and death (CIR, 1984). Subchronic and chronic inhalation exposure in experimental animals has resulted in a variety of nasal cavity lesions, including dysplasia (abnormal development of tissue) and squamous metaplasia (conversion of one kind of tissue into a form that is not normal for that tissue) of respiratory epithelium,

purulent or seropurulent rhinitis (an inflammation of the nasal tissue characterized by discharges that contain pus or serum and pus), interstitial inflammation of the lungs (CIR, 1984), reduced weight gain, reduced liver weights, and lesions of the kidney, liver, cerebral cortex, and respiratory tract (EPA, 1985c). Rusch et al., (1983) determined a NOAEL for squamous metaplasia and rhinitis of 1.0 ppm for rats and monkeys, although the study duration was not specified. Effects on the liver (decreased liver weights, histological changes) and kidney (vasodilation in a part of the renal cortex that is near the renal medulla) effects were also seen in animals following subchronic inhalation exposure (Rusch et al., 1983; Feldman and Bonashevskaya, 1971). In animals, formaldehyde has been shown to affect the firing rate of certain nerves in the nasal sensory system (EPA, 1987a). At high concentrations, formaldehyde has been reported to cause cerebral acid proteinase activity in rats in one study and decrease in cerebral RNA concentration, together with decreases in the succinate dehydrogenase and acid proteinase activities, in another (Consensus Workshop, 1984).

A range of predicted responses for upper respiratory and eye irritation risk, for a given formaldehyde concentration is obtained when seven studies are examined comparatively (Bender et al., 1983; Hanrahan et al., 1984; Horvath et al., 1988; Kulle, 1985; Liu et al., in press; Anderson and Molhave, 1984; Ritchie and Lehnen, 1987). Caution must be taken in inferring the results in EPA (1987) and in this data to the general population. Limitations in these studies at the present prevent the inference of eye and upper respiratory risks. None of the studies reviewed in this document and in EPA (1987) provide adequate data to precisely quantify general population risks for eye and upper respiratory effect associated with a specific formaldehyde concentration. Nevertheless, these studies document eye and upper respiratory tract effects at levels previously identified , 0.1 ppm to 3.0 ppm. Even though the prevalence of exposure can not be precisely estimated for a given formaldehyde concentration, these studies support the conclusion that the number of individuals responding in a population will increase with increasing formaldehyde concentration.

An inhalation reference concentration (RfC) is not available for formaldehyde at this time. EPA (1992b) has derived an oral reference dose (RfD) of 2×10^{-1} mg/kg/day, based on reduced weight gain and histopathology in rats following a 2-year drinking water study (Til et al., 1989). An uncertainty factor of 100 and a no-observed-adverse-effect level (NOAEL) of 15 mg/kg/day in male rats were used to derive the RfD.

A recent study by Krzyzanowski et al. (1990) analyzed the relation of chronic respiratory symptoms and pulmonary function to indoor formaldehyde exposure in a sample of children and adults in Tucson, Arizona. The average concentration of formaldehyde, measured in 202 households, was 26 ppb (32 $\mu g/m^3$). In only a few cases did the formaldehyde exceed 90 ppb (111 $\mu g/m^3$), with a maximum value of 140 ppb (172 $\mu g/m^3$). The data were collected from 298 children and 613 adults.

In children, the prevalence rates of chronic respiratory symptoms were not related to the formaldehyde exposure (considered in three categories: below 40 ppb, 41-60 ppb, and over 60 ppb). However, the diseases diagnosed by a doctor, asthma and chronic bronchitis in children 6-15 years of age, were more prevalent in houses with formaldehyde levels of 60-120 ppb (74-148 $\mu g/m^3$) than in those children less exposed. This is especially evident in children also exposed to environmental tobacco smoke. The effects in asthmatic children exposed to formaldehyde below 50 ppb (62 $\mu g/m^3$) were greater than in healthy ones. The effects in adults were less evident: decrements in expiratory flow rates due to formaldehyde over 40 ppb (49 $\mu g/m^3$) were seen only in the morning, and mainly in smokers. This childhood data are considered signs of developmental toxicity since the definition of developmental toxicity includes children up to the time of puberty.

6.9 References for Chapter 6

Acheson, E.D., M.J. Gardner, B. Pannett, H. Barnes, C. Osmond, and C. Taylor. 1984. The Lancet 1:611.

Albert, R.E., A.R. Sellakumar, S. Laskin, M. Kuschner, N. Nelson, and D.A. Snyder. 1982. Gaseous formaldehyde and hydrogen chloride induction of nasal cancer in the rat. J. Natl. Cancer Inst. 68:597-603.

Anderson, I. and L. Molhave. 1984. Controlled human studies with formaldehyde. In: Gibson, J.E. (ed.) "Formaldehyde Toxicity", Hemisphere Publishing Corp., New York. pp. 154-165.

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.

Bender, J.R., L.S. Mullin, G.J. Graepel, and W.E. Wilson. 1983. Eye irritation response of humans to formaldehyde. Am. Ind. Hyg. Assoc. J. 44: 463-465.

Bertazzi, P.A., C. Zochetti, A. Pesatori, L. Radice, and T. Vai. 1986. Exposure to formaldehyde and cancer mortality in a cohort of workers producing resins. Scand. J. Work Environ. Health 12: 461-468.

Blair, A., P. Stewart, M. O'Berg, W. Gaffey, J. Walrath, J. Ward, R. Bales, S. Kaplan, and D. Cubit. 1986. Mortality among industrial workers exposed to formaldehyde. JNCI 76:1071-1084.

Blair, A., P. Stewart, R.N. Hoover, J.F. Fraumeni, J. Walrath, M. O'Berg, and W. Gaffey. 1987. Cancers of the nasopharynx and oropharynx and formaldehyde exposure. JNCI 78:191-192.

Blair, A., P.A. Stewart, and R.N. Hoover. 1990a. Mortality from lung cancer among workers employed in formaldehyde industries. Am. J. Indust. Med. 17:683-699.

Blair, A., R. Saracci, P.A. Stewart, R.B. Hayes, and C. Shy. 1990b. Epidemiologic evidence on the relationship between formaldehyde exposure and cancer. Scand. J. Work Environ. Health 16: 381-393.

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.

Bogdanffy, M.S., P.H. Morgan, T.B. Starr, and E.T. Morgan. 1987.
Binding of formaldehyde to human and rat nasal mucus and bovine
serum albumin. Toxicol. Lett. 38:145-154.

Boysen, M., E. Zadig, V. Digernes, V. Abeler, A. and Reith.
1990. Nasal mucosa in workers exposed to formaldehyde: a pilot
study. Br. J. Indust. Med. 47:116-121.

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

CARB. 1992a. Proposed identification of formaldehyde as a toxic
air contaminant. Part A Exposure assessment. California Air
Resources Board, Stationary Source Division. January, 1992.

CARB. 1992b. Proposed identification of formaldehyde as a toxic
air contaminant. Part B Health assessment. California Air
Resources Board, Stationary Source Division. January, 1992.

Carey, P.M. 1987. Air toxics emissions from motor vehicles.
Ann Arbor, MI: U.S. Environmental Protection Agency, Office of
Mobile Sources, EPA Report no. EPA-AA-TSS-PA-86-5.

Casanova, M., D.F. Deyo, and Hd'A. Heck. 1989. Covalent binding
of inhaled formaldehyde to DNA in the nasal mucosa of Fischer-344
rats: Analysis of formaldehyde and DNA by high-performance
liquid chromatography and provisional pharmacokinetic
interpretation. Fund. Appl. Toxicol. 12:397-417.

Casanova, M. and Hd'A. Heck. 1987. Further studies of the
metabolic incorporation and covalent binding of inhaled 3H and
14C formaldehyde in Fischer-344 rats: Effects of glutathione
depletion. Toxicol. Appl. Pharmacol. 89:105-121.

Casanova, M. and Hd'A. Heck. 1991. The impact of DNA-protein
cross-linking studies on quantitative risk assessments of
formaldehyde. CIIT Activities, Vol. 11, No. 8.

Casanova-Schmitz, M., T.B. Starr, and Hd'A. Heck. 1984.
Differentiation between metabolic incorporation and covalent
binding in the labeling of macromolecules in the rat nasal mucosa
and bone marrow by inhaled ^{14}C and ^{3}H formaldehyde. Toxicol.
Appl. Pharmacol. 76:26-44.

Casanova, M., K.T. Morgan, W.H. Steinhagen, J.I. Everitt, J.A. Popp, and Hd'A. Heck. 1991. Covalent binding of inhaled formaldehyde to DNA in the respiratory tract of Rhesus monkeys: Pharmacokinetics, rat-to-monkey interspecies scaling, and extrapolation to man. Fundam. Appl. Toxicol. (in press).

CIR. 1984. Cosmetic ingredient review. Final report on the safety assessment of formaldehyde. J. Am. Coll. Toxicol. 3:157-184.

Clement International Corporation. 1991. Motor vehicle air toxics health information. For U.S. EPA Office of Mobile Sources, Ann Arbor, MI: September 1991.

Collins, J.J., J.C. Caporossi, and H.M.D. Utidjian. 1988. Formaldehyde exposure and nasopharyngeal cancer:re-examination of the National Cancer Institute study and an update of one plant. JNCI 80:376-377.

Colorado Department of Health. 1987. Unpublished data from a motor vehicle emissions toxics study of regulated and non-regulated pollutants. Aurora Emission Technical Center, Aurora, Colorado.

Consensus Workshop on Formaldehyde. 1984. Final report: Deliberations of the consensus workshop on formaldehyde, October 3-6, 1983, Little Rock, AK.

Cosma, G.N. and A.C. Marchok. 1987. The induction of growth altered cell population (tumor-initiation sites) in rat tracheal implants exposed to benzo(a)pyrene and formaldehyde. Carcinogenesis 8:1951-1953.

Craft, T.R., E. Bermudez, and T.R. Skopek. 1987. Formaldehyde mutagenesis and formation if DNA-protein crosslinks in human lymphoblasts in vitro. Mutat. Res. 176:147-155.

Crosby, R.M., K.K. Richardson, T.R. Craft, K.B. Benforado, H.L. Liber, and T.R. Skopek. 1988. Environ. Mol. Mutagenesis 12:155-166.

Cupitt, L. T. 1980. "Fate of toxic and hazardous materials in the air environment." U.S. Environmental Protection Agency (EPA-600/3-80-084).

Dalbey, W.E. 1982. Formaldehyde and tumors in hamster respiratory tract. Toxicology 24:9-14.

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.

Dodge, M. C. 1990. Formaldehyde production in photochemical smog as predicted by three state-of-the-science chemical oxidant mechanisms. J. Geophys. Res., 95:3635-3648.

Dowd, M.A., M.E. Gaulden, B.L. Proctor, and G.B. Seiber. 1986. Formaldehyde-induced acentric chromosome fragments and chromosome stickiness in *Chortophaga* neuroblasts. Environ. Mutagenesis 8:401-411.

Dresp, J. and M. Bauchinger. 1988. Direct analysis of the clastogenic effect of formaldehyde in unstimulated human lymphocytes by means of the premature chromosome condensation technique. Mutat. Res. 204:349-352.

Ecken, J.C.J. and F.H. Sobels. 1986. The effect of X-irradiation and formaldehyde treatment on spermatogonia on the reversion of an unstable, P-element insertion mutation in *Drosophila melanogaster*. Mutat. Res. 1765:61-65.

Edling, C., L. Odkvist, H. Hellquist. 1985. Formaldehyde and the nasal mucosa. Br. J. Ind. Med. 42:570-571.

Environ Corporation. 1986. Improving the assessment of risk of exposure to formaldehyde. Prepared for the Motor Vehicle Manufacturers Association.

Environ Corporation. 1987. Risk assessment issues in EPA's technical report "Air Toxics Emissions from Motor Vehicles". Prepared for the Motor Vehicle Manufacturer Association.

EPA. 1985. Health and Environmental Effects Profile for Formaldehyde. Cincinnati OH: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office. October 1985. ECAO-CIN-P142. Final Draft.

EPA. 1987a. Cancer Risk Due to Indoor and Outdoor Sources of Formaldehyde. Memo from Charles L. Gray to Richard D. Wilson. July 16, 1987.

EPA. 1987b. Assessment of health risks to garment workers and certain home residents from exposure to formaldehyde. April 1987. Office of Pesticides and Toxic Substances.

EPA. 1991a. Locating and estimating air emissions from sources of formaldehyde (revised). Office of Air Quality Planning and Standards, Research Triangle Park, NC. Report no. EPA-450/4-91-012.c

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

EPA. 1992a. Calculation of exhaust and evaporative toxic emissions mass fractions for the motor vehicle related air toxics report. Draft memo from Richard Cook to Phil Lorang. March 9, 1992.

EPA. 1992b. Integrated Risk Information System. U.S. Environmental Protection Agency. Office of Health and

Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

Feldman, Y.G. and T.I. Bonashevskaya. 1971. On the effects of low concentrations of formaldehyde. Hyg. Sanit. 36:174-180.

Feron, V.J., J.P. Bruijntjes, P.A. Woutersen, H.R. Immell, and L.M. Appelman. 1988. Nasal tumors in rats after short-term exposure to a cytotoxic concentration of formaldehyde. Cancer Lett. 39:101-111.

Gerin, M., J. Siemiatycki, L. Nadon, R. Dewar, and D. Krewski. 1989. Cancer risks due to occupational exposure to formaldehyde: Results of a multi-site case-control study in Montreal. Int. J. Cancer 44:53-58.

Hanrahan, L.P., K.A. Dally, H.A. Anderson, M.S. Kanarak, and J. Rankin. 1984. Formaldehyde vapor in mobile homes: A cross sectional survey of concentrations and irritant effects. Am. J. Public Health 74: 1026-1027.

Hardell, L., B. Johansson, and D. Axelson. 1982. Amer. J. Ind. Med. 3: 247-257.

Hayes, R.B., A. Blair, P.A. Stewart, R.F. Herrick, and H. Mahar. 1990. Mortality of US embalmers and funeral directors. Am. J. Ind. Med. 18: 641-652.

Hayes, R.B., J.W. Raatgever, and M. Gerin. 1986. Int. J. Cancer 37: 487-492.

Heck, Hd'A. and M. Casanova. 1987. Isotope effects and their implications for the covalent binding of inhales ^{3}H and ^{14}C formaldehyde in the rats nasal mucosa. Toxicol. Appl. Pharmacol. 89:122-134.

Heck, Hd'A., M. Casanova, W.H. Steinhagen, J.I. Everitt, E.T. Morgan, and J.A. Popp. 1989. Formaldehyde toxicity. DNA_protein crosslinking studies in rats and nonhuman primates. In: Feron VJ and Bosland MC (eds.), "Nasal Carcinogenesis in Rodents: Relevance to Human Health Risk", Pudoc Wagenigen, The Netherlands, pp. 159-164.

Hendrick, D.J., R.J. Rando, D.J. Lane, and M.J. Morris. 1982. Formaldehyde asthma: Challenge exposure levels and fate after five years. J. Occup. Med. 24:893-897.

Holmstrom, M., B. Wilhelmsson, H. Hellquist, and G. Rosen. 1989. Histological changes in the nasal mucosa in persons occupationally exposed to formaldehyde alone and in combination with wood dust. Acta. Otolaryngol. (Stockh) 107:102-129.

Horton, A.W., R. Tye, and K.L. Stemmer. 1963. Experimental carcinogenesis of the lung. Inhalation of gaseous formaldehyde or an aerosol of coal tar by CeH mice. J. Natl. Cancer Inst. 30: 31-43.

Horvath, E.P., H. Anderson W.E. Pierce, L. Hanrahan, and J.D. Wendlick. 1988. Effects of formaldehyde on the mucous membranes and lungs. J. Am. Med. Assoc. 259: 701-707.

IARC. 1982. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Volume 29. Some Industrial Chemicals and Dyestuffs. International Agency for Research on Cancer. World Health Organization, Lyon, France. p. 345-389.

IARC. 1987. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Supplement 7. Overall Evaluations of Carcinogenicity: An updating of IARC monographs volumes 1 to 42. International Agency for Research on Cancer. World Health Organization, Lyon, France. p. 211-216.

Iversen, O.H. 1986. Formaldehyde and skin carcinogenesis. Environ. Internat. 12:541-544.

Iversen, O.H. 1988. Formaldehyde and skin tumorigenesis in SENCAR mice. Environ. Internat. 14:23-27.

Jacob, D. J. 1986. Chemistry of OH in remote clouds and its role in the production of formic acid and peroxymonosulfate. J. Geophys. Res., 91(D9):9807-9826.

Jeffries, H. E. and K. G. Sexton. 1987. "Technical Discussion Related to the Choice of Photolytic Rates for Carbon Bond Mechanisms in OZIPM4/EKMA." U. S. Environmental Protection Agency (EPA-450/4-87-003).

Kerns, W.D., K.L. Pavkov, D.J. Donofrio, E.J. Gralla, and J.A. Swenberg. 1983. Carcinogenicity of formaldehyde in rats and mice after long-term inhalation exposure. Cancer Res. 43:4382-4392.

Klein-Szanto, A.J.P., H. Ura, and J. Resau. 1989. Formaldehyde induced lesions of xenotransplanted human nasal respiratory epithelium. Toxicol. Pathol. 17:33-37.

Krzyzanowski, M., J.J. Quackenboss, and M.D. Lebowitz. 1990. Chronic respiratory effects of indoor formaldehyde exposure. Environ. Res. 52:117-125.

Kulle, T.J. Letter to C.S. Scott, U.S. EPA, August 28, 1985.

Liber, H.L., K. Benforado, R.M. Crosby, D. Simpson, and T.R. Skopek. 1989. Formaldehyde-induced and spontaneous alterations in human hprt DNA sequence and mRNA expression. Mutat. Res. 226:31-37.

Ligocki, M.P. 1992. Review of EPA memorandum on calculation of toxic emission mass fractions. Prepared for Motor Vehicle Manufacturers Association. Systems Applications International, San Rafael, California (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., R.R. Schulhof, R.E. Jackson, M.M. Jimenez, G. Z. Whitten, G.M. Wilson, T.C. Meyers, and J.L. Fieber. 1992. Modeling the effects of reformulated gasolines on ozone and toxics concenetrations in the Baltimore and Houston areas. Systems Applications International, San Rafael, California (SYSAPP-92/127).

Linos, A., A. Blair, K.P. Cantor, L. Burmeister, S. VanLier, R.W. Gibson, L. Schuman, and G. Everett. 1990. Leukemia and non-Hodgkin's lymphoma among embalmers and funeral directors. JNCI 82:66.

Liu, K.S., F.Y. Huang, S.B. Hayward, J. Wesolowski, and K. Sexton. Irritant effects of formaldehyde exposure in mobile homes. Environ. Hlth. Perspec. (in press).

Malker, H.S.R., J.K. McLaughlin, J.A. Weiner, D.T. Silverman, W.J. Blot, J.L.E. Ericsson, and J.F. Fraumeni. 1990. Occupational risk factors for nasopharyngeal cancer in Sweden. Br. J. Indust. Med. 47:213-214.

Martin, L.R., M.P. Easton, J.W. Foster, and M.W. Hill. 1989. Oxidation of hydroxymethanesulfonic acid by Fenton's reagent. Atmos. Environ., 23:563-568.

Martin, W.J. 1990. A teratology study of inhaled formaldehyde in the rat. Reproductive Toxicology 4:237-239.

McArdle, J. V., and M. R. Hoffmann. 1983. Kinetics and mechanism of the oxidation of aquated sulfur dioxide by hydrogen peroxide at low pH. J. Phys. Chem., 87:5425-5429.

Monticello, T.N., E.T. Morgan, J.I. Everitt, and J.A. Popp. 1989. Effects of formaldehyde gas on the respiratory tract of Rhesus monkeys. Am. J. Pathol. 134:515-527.

Monticello, T.N. and K.T. Morgan. 1990. Correlation of cell proliferation and inflammation with nasal tumors in F-344 rats following chronic formaldehyde exposure. Am. Assoc. Cancer Res. 31:139.

Morgan, E.T., X-Z. Jiang, T.B. Starr, and W.D. Kerns. 1986. More precise localization of nasal tumors associated with chronic exposure of F-344 rats to formaldehyde gas. Toxicol. Appl. Pharmacol. 82:264-271.

Munger, J.W., D.J. Jacob, and M.R. Hoffmann. 1984. The occurrence of bisulfite-aldehyde addition products in fog- and cloudwater. J. Atmos. Chem., 1:335-350.

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

National Research Council (NRC). 1981. Formaldehyde and other aldehydes. Washington, DC: National Research Council, Committee on Aldehydes, Board on Toxicology and Environmental Health Hazards. National Academy Press.

Olsen, J.H., S.P. Jensen, M. Hink, K. Faurbo, N. Breum, and O. Jensen. 1984. Occupational formaldehyde exposure and increased nasal cancer risk in man. Int. J. Cancer 34: 639-644.

OSHA. 1987. Occupational exposure to formaldehyde. Final Rule. U.S. Department of Labor, Occupational Safety and Health Administration. Fed. Reg. 50: 46168-46312.

Partanen, T., T. Kauppinen, S. Hernberg, J. Nickels, R. Luukkoner, T. Hakulinen, and M.A. Pukkala. 1990. Formaldehyde exposure and respiratory cancer among woodworkers - an update. Scan. J. Work Environ. Health 16: 394-400.

Partanen, T., T. Kauppinen, M. Nurminen, J. Nickels, S. Hernberg, T. Hakulinen, E. Pukkala, and E. Savonen. 1985. Formaldehyde exposure and respiratory and related cancers: A case-referent study among Finnish workers. Scand. J. Work Environ. Health 11: 409-411.

Patterson, R., V. Pateras, L.C. Grammer, et al. 1986. Human antibodies against formaldehyde human serum albumin conjugates or human serum albumin in individuals exposed to formaldehyde. Int. Archs. Allergy Appl. Immun. 79:53-59.

Perry, R.H. and Chilton, C.H. 1973. Chemical Engineer's Handbook, Fifth Edition. McGraw-Hill Book Company.

Ritchie, I.N. and R.G. Lehnen. 1987. Formaldehyde-related health complaints of residents living in mobile and conventional homes. Am. J. Public Health 77: 323-328.

Robins, J., N. Pambrun, C. Chute, and D. Blevins. 1988. Estimating the effect of formaldehyde exposure on lung cancer and non-malignant respiratory disease (NNRD) mortality using a new method to control for the healthy worker effect. In: Progress in Occupational Methodology. Hogstedt C and Reuterwall C. (eds.). Excerpta Medica, Amsterdam, pp. 75-78.

Roush, G., J. Walrath, L. Sayner, S. Kaplan, J.T. Flannery, and A. Blair. 1987. Nasopharyngeal cancer, sinonasal cancer, and occupations related to formaldehyde: A case-control study. JNCI 79:1221-1224.

Rusch, G.M., J.J. Clary, W.E. Rinehart, and H.F. Bolte. 1983. A 26-week inhalation toxicity study with formaldehyde in the monkey, rat, and hamster. Toxicol. Appl. Pharmacol. 68: 329-343.

Saillenfait, A.M, P. Bonnet, and J. De Ceaurriz. 1989. The effects of maternally inhaled formaldehyde on embryonal and foetal development in rats. Fd. Chem. Toxic. 27:545-548.

Sauder, L.R., M.D. Chatham, D.J. Green, and T.J. Kulle. 1986.
Acute pulmonary response to formaldehyde exposure in healthy
nonsmokers. J. Occup. Med. 28:420-424.

Sauder, L.R., D.J. Green, N.D. Chatham, et al. 1987. Acute
pulmonary response of asthmatics to 3.0 ppm formaldehyde.
Toxicol. Industr. Health 3:569-578.

Schmidt, U. and E. Loeser. 1986. Epoxidation of 1,3-butadiene
in liver and lung tissue of mouse, rat, monkey and man. Adv.
Exp. Med. Biol. 197:951-957.

Sellakumar, A.R., C.A. Snyder, J.J. Solomon, and R.E. Albert.
1985. Carcinogenicity of formaldehyde and hydrogen chloride in
rats. Toxicol. Appl. Pharmacol. 81: 401-406.

Shikiya, D.C., C.S. Liu, M.I. Kahn, J. Juarros, and W.
Barcikowski. 1989. In-vehicle air toxics characterization study
in the south coast air basin. South Coast Air Quality Management
District, El Monte, CA. May, 1989.

Shumilina, A.V. 1975. Menstrual and child-bearing functions of
female workers occupationally exposed to the effects of
formaldehyde. Gig. Truda. I. Professional Nye. Zabolevaniya
12:18-21.

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. 1981. Characterization of Exhaust Emissions from
High Mileage Catalyst-Equipped Automobiles. Ann Arbor, Michigan:
U.S. Environmental Protection Agency, Office of Mobile Sources.
Publication no. EPA-460/3-81-024.

Snyder, R.D. and B. Van Houten. 1986. Genotoxicity of
formaldehyde and an evaluation of its effect on the DNA repair
process in human diploid fibroblasts. Mutat. Res. 165:21-30.

Soffritti, M., C. Maltoni, F. Maffei, and R. Biagi. 1989.
Formaldehyde: An experimental multipotent carcinogen. Toxicol.
Indust. Health 5:699-730.

Springer, K. J. 1977. Investigation of Diesel-Powered Vehicle
Emissions VII. Ann Arbor, Michigan: U.S. Environmental Agency,
Office of Mobile Sources. Publication no. EPA-460/3-76-034.

Springer, K. J. 1979. Characterization of Sulfates, Odor,
Smoke, POM and Particulates from Light and Heavy-Duty Engines --
Part IX. Ann Arbor, Michigan: U.S. Environmental Protection
Agency, Office of Mobile Sources. Publication no. EPA-460/3-79-
007.

Starr T.B. 1990. Quantitative cancer risk estimation for
formaldehyde. Risk Anal. 10: 85-91.

Stayner, L., A.B. Smith, G. Reeve, et al. 1985. Proportionate mortality study of workers exposed to formaldehyde in the garment industry. Am. J. Ind. Med. 7:229-240.

Stayner, L. T., L. Elliot, L. Blade, R. Keenlyside, and W. Halperin. 1988. A retrospective cohort mortality study of workers exposed to formaldehyde in the garment industry. Am. J. Ind. Med. 13:667-682.

Sterling, T.D. and J.J. Weinkam. 1989. Reanalysis of lung cancer mortality in a National Cancer Institute study on mortality among industrial workers exposed to formaldehyde. Additional discussion. J. Occup. Med. 31:881-883.

Stern, F.B., I.I. Beaumont, W.E. Halperin, L.I. Nurthy, B.W. Hills, and J.M. Fajen. 1987. Mortality of chrome leather tannery workers and chemical exposures and tanneries. Scand. J. Work Environ. Health 13:108-117.

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.

Swenberg, J.A., C.S. Barrow, C.J. Boreikol, Hd'A Heck, R.J. Levine, E.T. Morgan, and T.B. Starr. 1983. Non-linear biological responses to formaldehyde and their implications for carcinogenic risk assessment. Carcinogenesis 4: 945-952.

Smylie, G.M., G.Z. Whitten, R.E. Morris, J.L. Fieber, and K. O'Connor. 1990. "Technical analysis of the ARB staff proposal for hydrocarbon reactivity adjustment factors for clean fuel/low emission vehicle regulations." Systems Applications International, San Rafael, California (SYSAPP-90/092).

Takahashi, M., R. Hasegawa, F. Furukawa, K. Toyoda, H. Sato, and Y. Hayashi. 1986. Effects of ethanol, potassium metabisulfite, formaldehyde, and hydrogen peroxide on gastric carcinogenesis in rats after initiation with N-methyl-N-nitrosoguanidine. Jpn. J. Cancer Res. (Gann) 77:118-124.

Til, H.P., P.A. Woutersen, V.J. Feron, V.H.M. Hollanders, and H.E. Falke. 1989. Two-year drinking-water study of formaldehyde in rats. Fd. Chem. Toxicol. 27:77-87.

Tobe, M., T. Kaneko, Y. Uchida, et al. 1985. Studies of the inhalation toxicity of formaldehyde. National Sanitary and Medical Laboratory Service (Japan). pp 1294.

Tobe, M., K. Naito, and Y. Kurokawa. 1989. Chronic toxicity study on formaldehyde administered orally to rats. Toxicology 56:79-86.

Uba, G., D. Pachorek, J. Bernstein, et al. 1989. Prospective study of respiratory effects of formaldehyde among healthy and asthmatic medical students. Am. Jour. Ind. Med. 15:91-101.

Ulsamer, A.G., K.C. Gupta, M.S. Cohen, and P.W. Preuss. 1982. Formaldehyde in indoor air: Toxicity and risk. In: Proceed. 45th(2) Ann. Meet. Air Pollut. Control Assoc. 16 p.

Ulsamer, A.G., J.R. Beall, H.K. Kang, et al. 1984. Overview of health effects of formaldehyde. In: Saxsena J. (ed.), Hazard Assessment of chemicals -- Current Developments. NY: Academic Press, Inc. 3:337-400.

Universities Associated for Research and Education in Pathology, Inc. 1988. Epidemiology of chronic occupational exposure to formaldehyde: report of the ad hoc panel on health aspects of formaldehyde. Toxicol. Indus. Health 4: 77-90.

Ura. H., P. Nowak, S. Litwin, P. Watts, R.D. Bonfil, and A.J.P. Klein-Szanto. 1989. Effects of formaldehyde on normal xenotransplanted human tracheobronchial epithelium. Am. J. Pathol. 134:99-106.

Urban, C. M. 1980a. 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. 1980b. 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-460/3-8-005.

Urban, C. M. 1980c. 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. 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.

Vaughan, T.L., C. Strader, S. Davis, and J.R. Daling. 1986a. Formaldehyde and cancers of the pharynx, sinus, and nasal cavity: I. Occupational exposures. Int. J. Cancer. 38: 677-683.

Vaughan, T.L., C. Strader, S. Davis, and J.R. Daling. 1986b. Formaldehyde and cancers of the pharynx, sinus, and nasal cavity. II. Residential exposures. Int. J. Cancer. 38: 685-688.

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.

Warner-Selph, M. A., and Joseph DeVita. 1989. Measurements of toxic exhaust emissions from gasoline-powered light duty vehicles. SAE Paper 892075.

Wilmer, J.W., P.A. Woutersen, L.M. Appelman, W.R. Leeman, and V.J. Feron. 1989. Subchronic (13-week) inhalation toxicity study of formaldehyde in male rats: 8-hour intermittent versus 8-hour continuous exposure. Toxicol. Lett. 47:287-293.

Wilmer, J.W., P.A. Woutersen, L.M. Appelman, W.R. Leeman, and V.J. Feron. 1987. Subacute (4-week) inhalation toxicity study of formaldehyde in male rats: 8-hour intermittent versus 8-hour continuous exposure. J. Appl. Toxicol. 7:15-16.

Wilson, A. et al. 1991. Air toxics microenvironment exposure and monitoring study. South Coast Air Quality Management District, El Monte, CA.

Witek, T.J., E.N. Schachter, T. Tosun, et al. 1987. An evaluation of respiratory effects following exposure to 2.0 ppm formaldehyde in asthmatics: Lung function, symptoms, and airway reactivity. Arch. Environ. Health 42:230-237.

Woutersen, P.A., A. van Garderen-Hoetmer, J.P. Bruijntjes, A. Zwart, and V.J. Feron. 1989. Nasal tumors in rats after severe injury to the nasal mucosa and prolonged exposure to 10 ppm formaldehyde. J. Appl. Toxicol. 9:39-46.

Yager, J.W., K.L. Cohn, R.C. Spear, J.N. Fisher, and L. Morse. 1986. Sister-chromatid exchanges in lymphocytes of anatomy students exposed to formaldehyde-embalming solution. Mutat. Res. 17:135-139.

Yetter, R.A., H. Rabitz, F.L. Dreyer, R.G. Maki, and R.B. Klemm. 1989. Evaluation of the rate constant for the reaction OH + H_2CO: Application of modeling and sensitivity analysis techniques for determination of the product branching ratio. J. Chem. Phys., 91:4088-4097.

Zwart, A., P.A. Woutersen, L.M. Appelman, J.W. Wilmer, and B.J. Spit. 1988. Cytotoxic and adaptive effects in rats nasal epithelium after 3-day and 13-week exposure to low concentrations of formaldehyde vapour. Toxicology 51:87-99.

7.0 1,3-BUTADIENE

7.1 Chemical and Physical Properties (EPA, 1989; 1992)

1,3-Butadiene is a colorless, flammable gas at room temperature with a pungent, aromatic odor, and a chemical formula C_4H_6 (CH_2:CHCH:CH_2). Table 7-1 summarizes 1,3-butadiene's chemical and physical properties. 1,3-Butadiene is insoluble in water, slightly soluble in methanol and ethanol, and soluble in organic solvents such as benzene and ether. 1,3-Butadiene is also structurally related to known carcinogens.

Because of its reactivity, 1,3-butadiene is estimated to have a short atmospheric lifetime. The actual lifetime depends upon the conditions at the time of release. The primary removal mechanisms are through chemical reactions with hydroxyl radicals and ozone. Therefore, factors influencing 1,3-butadiene's atmospheric lifetime, such as the time of day, sunlight intensity, temperature, etc., also include those affecting the availability of hydroxyl radicals and ozone.

Table 7-1. Chemical and Physical Properties of 1,3-Butadiene.

Properties	Values
Molecular weight	54.09 g/mole
Melting point	$-108.91°C$ ($-164.04°F$)
Boiling point	$-4.41°C$ ($24.06°F$)
Density at 20°C (68°F)	0.6211 g/cm^3
Vapor pressure at 20°C	1.2 atm.
Flash point	$-105.0°C$ ($-157.0°F$)
Solubility in water at 20°C	0.735 g/L
Conversions at 25°C (77°F)	1 ppm (by volume) = 2.21 mg/m^3 1 mg/m^3 = 0.45 ppm (by volume)

7.2 Formation and Control Technology

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. As a rule, refiners try to minimize the level of 1,3-butadiene in gasoline and diesel fuel because it tends to readily form a varnish which can be harmful to engines (EPA, 1989). Therefore, the majority of gasoline and diesel fuel should have no significant 1,3-butadiene content.

1,3-Butadiene emissions appear to increase roughly in proportion to 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.

7.3 Emissions

7.3.1 Emission Fractions Used in the MOBTOX Emissions Model

Actual vehicle emissions were used to develop 1,3-butadiene emission fractions. Because 1,3-butadiene decays rapidly in the Tedlar bags used to collect emissions samples, exhaust speciation analyses often underestimate 1,3-butadiene emissions. This is especially true of older studies. Thus, although 1,3-butadiene emissions data from many studies were available (Appendix B2), it was decided to use data from one study with a very large number of vehicles, recently conducted by CARB (1991), in which deterioration of 1,3-butadiene was strictly controlled, and emission fractions were adjusted to account for time lag between sample collection and sample analysis. This study also tested more typical in-use vehicles, rather than low mileage vehicles as in other studies. 1,3-Butadiene emission fractions for different programs included in modeling components are included in Appendix B6. For vehicles with three-way catalysts, 1,3-butadiene emission fractions from the Auto/Oil study were somewhat lower than emission fractions from the CARB data (overall, about 15%). This may indicate that later model vehicles, with more efficient catalysts, have lower 1,3-butadiene fractions. Thus, with fleet turnover, 1,3-butadiene fractions in motor vehicle emissions may drop. Also, it should be noted that most 1,3-butadiene emissions occur during cold starts, and use of heated catalysts in future years will reduce these cold start emissions (see Ford Motor Company comments in Appendix I).

CARB measured 1,3-butadiene mass emissions for 55 LDGVs/LDGTs with three-way catalysts or three-way plus oxidation catalysts, 7 LDGVs/LDGTs with oxidation catalysts, 16 LDGVs/LDGTs with no catalysts, 2 LDDVs, and 1 HDDV. CARB then calculated THC fractions and converted these fractions to TOG fractions using conversion factors. CARB's THC fraction was converted to a TOG fraction using the conversion factors in Table 3-7, rather than the CARB conversion factors. The resultant TOG fractions for vehicles running on baseline fuel are listed in Appendix B5. CARB calculated an average emission fraction for three-way catalyst and three-way plus oxidation catalyst LDGV/LDGT combined. Since only 7 of the 55 vehicles from the combined category had three-way plus oxidation catalysts, the average emission fraction was applied to the three-way catalyst category. For LDGVs/LDGTs with three-way plus oxidation catalysts, it was assumed that TOG fractions for this category would be the same as for oxidation catalysts. CARB also did not measure 1,3-butadiene emissions for HDGVs. It was assumed the fraction for HDGVs with

three-way catalysts was the same as for LDGVs with three-way catalysts, and that the fraction for HDGVs with no catalysts was the same as for LDGVs with no catalysts.

No 1,3-butadiene data were available for oxygenated fuels from the CARB study. To calculate TOG fractions for vehicles running on MTBE blends and 10% ethanol, adjustment factors were applied to the baseline emission fractions for each vehicle class/catalyst combination. To calculate an appropriate adjustment factor, percent of 1,3-butadiene in exhaust was compared for baseline and oxygenated blends (Appendix B4). This comparison was performed for 15% MTBE and 10% ethanol. The average percent change (expressed as a fraction) was added to 1, representing baseline emissions with gasoline, and the baseline 1,3-butadiene fractions then multiplied by the resultant factor. The 15% MTBE and 10% ethanol adjustment factors for LDGVs/LDGTs with various catalyst technologies are summarized in Table 7-2. The 15% MTBE numbers were estimated using data from Auto/Oil (1990) and DeJovine et al. (1991) for LDGVs/LDGTs with three-way catalysts, Auto/Oil (1991) for LDGVs/LDGTs with three-way plus oxidation and oxidation catalysts, and Warner-Selph and Smith (1991) for LDGVs/LDGTs with no catalysts. The 10% ethanol numbers were estimated using data from Auto/Oil (1990) and Warner-Selph and Smith (1991) for LDGVs/LDGTs with three-way catalysts, and Warner-Selph and Smith (1991) for LDGVs and LDGTs with oxidation catalysts or no catalysts. Due to a lack of data, the adjustment factor for LDGVs/LDGTs with three-way plus oxidation catalysts was assumed to be equal to the one for LDGVs/LDGTs with oxidation catalysts.

Table 7-2. 15% MTBE and 10% Ethanol Emission Fraction Adjustment Factors for 1,3-Butadiene.

Vehicle Class	Catalyst Technology	15% MTBE Adjustment Factor	10% Ethanol Adjustment Factor
LDGV/LDGT	3-way	0.9798	0.8812
LDGV/LDGT	3-way + ox	0.9873	0.9375
LDGV/LDGT	oxidation	1.1790	0.9375
LDGV/LDGT	non-cat	1.2382	1.1233

Since the average percent change was calculated for 15% MTBE (2.7% weight percent oxygen), and 11.0% MTBE (2.0% oxygen) was assumed for reformulated fuel and California standards components, average percent changes in the 1,3-butadiene TOG fraction from 0 to 15% MTBE were multiplied by 2.0/2.7, the

ratio of oxygen contents by weight for reformulated gasoline and 15% MTBE. For HDGVs with three-way catalysts and with no catalysts, the same 15% MTBE and 10% ethanol adjustment factors were assumed as for LDGVs/LDGTs with the same catalyst technologies.

7.3.2 Emission Factors for Baseline and Control Scenarios

The fleet average 1,3-butadiene emission factors as determined by the MOBTOX emissions model are presented in Table 7-3. When comparing the base control scenarios relative to 1990, the emission factor is reduced by 40% in 1995, by 54% in 2000, and by 57% in 2010. The expansion of reformulated fuel use in 1995 provides no net reduction in the emission factor, relative to 1990. In 2000 and in 2010, the expansion of reformulated fuel usage and the California standards reduces the emission factor by another 2 to 3% over the base control from the respective year.

7.3.3 Nationwide Motor Vehicle 1,3-Butadiene Emissions

The nationwide 1,3-butadiene metric tons are presented in Table 7-4. Total metric tons are determined by multiplying the emission factor (g/mile) by the VMT determined for the particular year. The VMT, in billion miles, was determined to be 1793.07 for 1990, 2029.74 for 1995, 2269.25 for 2000, and 2771.30 for 2010. When comparing the base control scenarios relative to 1990, the metric tons are reduced by 32% in 1995 and by 42% in 2000. Even though the emission factor continues to decrease from 2000 to 2010, this is more than offset by the large increase in VMT. As a result, metric tons in 2010 actually increase relative to 2000.

7.3.4 Other Sources of 1,3-Butadiene

Mobile sources account for approximately 94% of the total 1,3-butadiene emissions (EPA, 1989).   The remaining 1,3-butadiene emissions (6%) come from stationary sources related to industries producing 1,3-butadiene and those industries that use 1,3-butadiene to produce other compounds.

Of the 6% attributable to stationary 1,3-butadiene sources, 73.8% is produced by the styrene-butadiene copolymer (rubber and latex) industry. Another 25.8% of the 1,3-butadiene emissions are produced by the industries manufacturing polybutadiene, neoprene rubber, acrylonitrile-butadiene-styrene resin, nitrile rubber, and adiponitrile, the raw material for nylon 6,6 production. There are also miscellaneous producers/users that account for only a small percentage of the total stationary source emissions. The final 0.4% is produced by the manufacturing of 1,3-butadiene itself. Of the 11 producers of 1,3-butadiene, 9 are located in Texas, and 2 in Louisiana (EPA, 1989).

Table 7-3. Annual Emission Factor Projections for 1,3-Butadiene.

Year-Scenario	Emission Factor g/mile	Percent Reduction from 1990
1990 Base Control	0.0156	–
1995 Base Control	0.0094	40
1995 Expanded Reformulated Fuel Use	0.0093	40
2000 Base Control	0.0071	54
2000 Expanded Reformulated Fuel Use	0.0069	56
2000 Expanded Adoption of California Standards	0.0069	56
2010 Base Control	0.0067	57
2010 Expanded Reformulated Fuel Use	0.0064	59
2010 Expanded Adoption of California Standards	0.0062	60

Table 7-4. Nationwide Metric Tons Projection for 1,3-Butadiene.

Year-Scenario	Emission Factor g/mile	Metric Tons
1990 Base Control	0.0156	27,972
1995 Base Control	0.0094	19,080
1995 Expanded Reformulated Fuel Use	0.0093	18,877
2000 Base Control	0.0071	16,112
2000 Expanded Reformulated Fuel Use	0.0069	15,658
2000 Expanded Adoption of California Standards	0.0069	15,658
2010 Base Control	0.0067	18,568
2010 Expanded Reformulated Fuel Use	0.0064	17,736
2010 Expanded Adoption of California Standards	0.0062	17,182

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. This figure is based on the average of an EPA estimate and a California Air Resources Board estimate (CARB, 1991).

Analysis of EPA data indicated that about 46% of mobile source 1,3-butadiene emissions (43% of total 1,3-butadiene emissions) can be attributed to onroad motor vehicles, with the remainder attributable to nonroad mobile sources. This figure is based on a number of crude estimates and assumptions. First, it was estimated that about 70% of mobile source VOCs are attributable to onroad vehicles (Section 5.3.4). This VOC split was adjusted by onroad and nonroad 1,3-butadiene fractions to come up with the estimate of 46% of mobile source 1,3-butadiene from on-road motor vehicles. For onroad vehicles, 1,3-butadiene was estimated to be 0.61% of exhaust. This is a 1990 fleet average toxic fraction, with fractions in Appendix B2, weighted using 1990 VMT fractions. For nonroad vehicles, 1,3-butadiene was estimated to be 1.3% of exhaust, based on the NEVES report (EPA, 1991).

The CARB study cited above indicated that, in California, of about 96% of 1,3-butadiene estimated to come from mobile sources, 71% could be attributed to onroad motor vehicles, and the remainder to other mobile sources. Thus, by averaging the EPA estimate and the CARB estimate, an estimated contribution of 59% of mobile source 1,3-butadiene emissions from onroad motor vehicles was derived.

7.4 Atmospheric Reactivity and Residence Times

7.4.1 Gas Phase Chemistry of 1,3-Butadiene

The processes involved in transformation and residence times were previously discussed in Section 5.4. For a more detailed explanation of the various parameters involved in these processes please refer to Section 5.4. The information that follows on transformation and residence times has been mainly excerpted from a report produced by Systems Applications International for the EPA (Ligocki et al., 1991).

The structure of 1,3-butadiene (C_4H_6) is a straight-chain molecule with two conjugated double bonds ($H_2C=CH-CH=CH_2$). Species containing double bonds are referred to as "alkenes" or "olefins". These double bonds represent extremely active sites for atmospheric oxidation. In contrast to the slow rate of reaction of benzene in the atmosphere, 1,3-butadiene reacts quite rapidly with the hydroxyl radical, ozone and nitrate radical. Furthermore, the oxidation of 1,3-butadiene produces two species which are themselves toxic, formaldehyde and acrolein (C_3H_4O).

Acrolein and
its oxidation products are powerful lacrimators (compounds which
cause eye irritation). Concern for the high atmospheric
reactivity of compounds such as 1,3-butadiene and their
undesirable products such as acrolein lead to early regulations
limiting the olefin content of gasoline (e.g., Rule 66 by the Los
Angeles Air Pollution Control District in 1966).

7.4.1.1 Gas Phase Reactions

     There are three chemical reactions of 1,3-butadiene which
are important in the ambient atmosphere: reaction with OH,
reaction with O_3, and reaction with NO_3. All of these reactions
are rapid, indicating that 1,3-butadiene will be transformed
rapidly in the atmosphere. The reaction of 1,3-butadiene with
the oxygen radical and with NO_2 do occur in the atmosphere, but
because their concentrations are much lower than OH and O_3, these
reactions are not important in the ambient atmosphere.

7.4.1.2 Reaction Products

     The atmospheric oxidation of 1,3-butadiene by OH proceeds
primarily by addition across the double bonds. Olefins generally
react to form two aldehyde products, one from each side of the
original double bond. Therefore, the major products from
1,3-butadiene are formaldehyde (from the terminal carbon) and
acrolein (from the internal carbon). These products would be
expected, at least to some extent, from any of the atmospheric
reactions of this diolefin, although the mechanism and products
from the O_3 reaction are not completely understood. An exception
is the reaction of 1,3-butadiene with NO_3. This reaction
apparently proceeds primarily by addition, producing
approximately 60% nitrates, with the yield of formaldehyde and
acrolein only 12% (Barnes et al., 1990).

     Formaldehyde has many sources in the atmosphere. The
production of formaldehyde from the oxidation of 1,3-butadiene
would not be expected to be a significant portion of the total
secondary formaldehyde production. However, 1,3-butadiene can be
considered to be the major precursor species for atmospheric
acrolein production.

7.4.2 Aqueous Phase Chemistry of 1,3-Butadiene

     The aqueous solubility of 1,3-butadiene is very low, an
order of magnitude smaller than that of benzene. Incorporation
of 1,3-butadiene into clouds and rain will not be an important
process due to the low solubility of 1,3-butadiene despite the
relatively rapid reaction with OH radical.

7.4.3 1,3-Butadiene Residence Times

     Residence times for 1,3-butadiene were calculated by

considering only gas-phase chemical reactions with OH, NO_3, and O_3. Due to the low solubility of 1,3-butadiene, wet deposition and in-cloud chemical destruction are not important processes for 1,3-butadiene, and were not considered in the calculations. The importance of dry deposition for 1,3-butadiene depends upon the value assumed for the reactivity parameter. For these calculations, 1,3-butadiene was assumed to be unreactive on surfaces; if this assumption is valid then its deposition velocity will be negligibly small.

The results of the residence time calculations for 1,3-butadiene are presented in Table 7-5. During the daytime, the residence time of 1,3-butadiene is determined primarily by its reaction with OH radical, with a small contribution from the reaction with O_3. The residence time of 1,3-butadiene under summer, daytime, clear-sky conditions is one hour or less for all four cities. The residence time of 1,3-butadiene has previously been estimated at 4 hours under clean, background atmospheric conditions (Cupitt, 1987). The shorter residence times estimated here are a result of the higher oxidant concentrations predicted for urban areas.

At night, the rapid reaction of 1,3-butadiene with NO_3, and to a lesser extent O_3, leads to residence times of 0.5-6 h under clear-sky conditions. In fact, for Los Angeles, the summer, clear-sky residence time for 1,3-butadiene is estimated to be shorter at night than it is during the daytime. These relatively short residence times for 1,3-butadiene even at night indicate that there is very little possibility of carryover of 1,3-butadiene concentrations from one day to the next during the summertime under clear-sky conditions. Under cloudy-sky conditions, summer nighttime residence times were estimated to be 6-15 h. These are long enough to allow for the possibility of day-to-day carryover.

Daytime residence times for different cities within a given season varied by factors of 2-3, whereas nighttime residence times varied by much larger factors. As with benzene, the difference
between summer and winter conditions was large at all sites, with winter residence times 10-30 times greater than summer residence times.

Under wintertime conditions, the residence time of 1,3-butadiene was estimated to be in the range of 12-2000 h. Although daytime residence times during the winter are still relatively short, residence times at night can be very long because of the extremely low NO_3 concentrations. Behavior of 1,3-butadiene is therefore very different in the winter than it is during the summer. Significant day-to-day carryover of 1,3-butadiene concentrations is possible in the winter, particularly under cloudy-sky conditions.

The major uncertainties in the residence time calculations for 1,3-butadiene are most likely the nighttime NO_3 concentrations and
the NO_3 reaction rate. Concentrations of NO_3 are highly variable,

TABLE 7-5.   Atmospheric residence time calculation for 1,3-butadiene. All times are in hours unless otherwise noted.

	Los Angeles		St. Louis		Atlanta		New York	
	July	Jan	July	Jan	July	Jan	July	Jan
Clear sky – day	0.8	5	0.5	7	0.6	7	1.0	14
Clear sky – night	0.4	16	6	200	0.6	5	1.1	1600
Clear sky – avg	0.6	8	2	17	0.6	6	1.0	40
Cloudy – day	1.7	10	1.2	16	1.2	16	2	30
Cloudy – night	6	90	15	400	7	90	11	2000
Cloudy – avg	2	20	2	40	1.8	30	3	80
Monthly Climatological Average	0.8	11	2	30	1.0	12	1.7	50

often peaking shortly after sunset and then decreasing rapidly by midnight (Platt et al., 1980). In addition, the NO$_3$ reaction rate is only known to within a factor of two. Therefore, although the daytime residence times are accurate to about a factor of two, nighttime residence times are certain only to within an order of magnitude.

7.4.4 Limited Urban Airshed Modeling Results for 1,3-Butadiene

The Urban Airshed Model (UAM) has been previously discussed in Section 5.4. Please refer to this section for details about the model, its inputs, and modifications. Much of the information below has been excerpted from reports conducted for EPA by Systems Applications International (SAI) (Ligocki et al., 1991, 1992).

1,3-Butadiene was treated explicitly in the UAM-Tox. Mobile and stationary emissions of 1,3-butadiene were tagged separately and carried through the simulation as distinct species. The gas phase reactions discussed previously were added to the chemistry subroutines. Because the focus of the study was on destruction of the toxic species rather than on the subsequent chemistry of their reaction products, no products were included in the UAM modifications for 1,3-butadiene.

St. Louis Simulation

In the St. Louis simulation, the high reactivity of 1,3-butadiene is demonstrated by the large deviation of the reactive and inert 1,3-butadiene curves in Figure D-3 located in Appendix D. By mid-afternoon, the concentration predicted in the absence of chemistry would be 0.4 ppb, versus 0.05 ppb for the simulation which included chemistry. Thus, the afternoon concentration of 1,3-butadiene was reduced by 90 percent due to atmospheric reactions. The two curves approach each other again after sunset, when the 1,3-butadiene concentration reached its highest value of the simulation. These results suggest that human exposure to 1,3-butadiene during the summertime will be limited to areas near sources. According to Table 7-5, 1,3-butadiene is destroyed relatively rapidly even at night, so little or no carryover of 1,3-butadiene concentration to the following day would be expected. The concentration of 1,3-butadiene would be expected to be greater in the wintertime due to the less active photochemistry.

The comparison of simulated concentrations with ambient measured concentrations showed good agreement for 1,3-butadiene.

Baltimore-Washington and Houston Area Simulations

Simulations for the summer Baltimore-Washington area episode (Ligocki et al., 1992) 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. Maximum daily average 1,3-butadiene concentration for the 1988 base scenario was 0.95 ppb. Motor vehicle-related 1,3-butadiene emissions accounted for about 23% of total 1,3-butadiene emissions, based on the 1995 no motor vehicle scenario. This motor vehicle emission estimate is lower than the 56% estimate obtained in Section 7.3.4 for motor vehicles. The Ligocki et al. (1992) study suggests that a reason for this discrepancy could be the inclusion of more types of area source toxic emissions in the UAM-Tox inventory than had been considered previously. Also, the nonroad contribution to mobile source 1,3-butadiene could be underestimated. Summer Baltimore-Washington area simulations were in very good agreement with UATMP data throughout the domain.

In the winter 1988 base scenario, the maximum daily average 1,3-butadiene concentration was 2.57 ppb, about 3 times higher than in summer. A major reason for this is slower reactive decay of 1,3-butadiene in winter. Motor vehicle-related 1,3-butadiene accounted for 29% of total 1,3-butadiene emissions. Reformulated gasoline use had very little effect on winter 1,3-butadiene ambient concentrations.

For the summer 1987 base scenario in Houston, the maximum daily average 1,3-butadiene concentration was 33.2 ppb. This high level was due to the single largest point source of 1,3-butadiene emissions in the United States. However, the model may have significantly overestimated the magnitude of this concentration. Throughout the rest of the modeling domain, concentrations were around 2 ppb. Motor vehicle-related 1,3-butadiene accounted for 16% of total 1,3-butadiene emissions, based on the 1995 no motor vehicle scenario. Motor vehicle-related 1,3-butadiene contributed less to overall 1,3-butadiene in Houston than in the Baltimore-Washington area, due to the large impact of point sources in Houston. Simulations for the summer Houston episode predicted little effect on maximum daily average concentration of 1,3-butadiene with reformulated gasoline.

7.5 Exposure Estimation

7.5.1 Annual Average Exposure Using HAPEM-MS

The data presented in Table 7-6 represent the results determined by the HAPEM-MS modeling that was described previously in Section 4.1.1. These numbers have been adjusted to represent the increase in VMT expected in future years.

The HAPEM-MS exposure estimates in Table 7-6 represent the 50th percentiles of the population distributions of exposure, i.e., half the population will be above and half below these

values. High end exposures can also be estimated by using the
95th

Table 7-6. **Annual Average HAPEM-MS Exposure Projections for 1,3-Butadiene.**

Year-Scenario	Exposure ($\mu g/m^3$)		
	Urban	Rural	Nationwide
1990 Base Control	0.48	0.26	0.42
1995 Base Control	0.31	0.17	0.28
1995 Expanded Reformulated Fuel Use	0.31	0.17	0.27
2000 Base Control	0.26	0.14	0.23
2000 Expanded Reformulated Fuel Use	0.25	0.13	0.22
2000 Expanded Adoption of California Standards	0.25	0.13	0.22
2010 Base Control	0.28	0.15	0.25
2010 Expanded Reformulated Fuel Use	0.27	0.14	0.24
2010 Expanded Adoption of California Standards	0.26	0.14	0.23

percentile of the distributions. According to the HAPEM-MS sample output for benzene, the 95th percentile is 1.8 times higher than the 50th percentile for urban areas, and 1.2 times high for rural areas. Applying these factors to the exposure estimates in Table 7-6, the 95th percentiles for urban areas range from 0.45 µg/m^3 for the 2000 expanded reformulated gasoline and California standards use scenarios to 0.86 µg/m^3 for the 1990 base control scenario. The 95th percentiles for rural areas range from 0.16 to 0.31 µg/m^3, respectively.

7.5.2 Comparison of HAPEM-MS Exposures to Ambient Monitoring Data

As stated in section 4.1.2, four national air monitoring programs/databases contain data on air toxics and the data for 1,3-butadiene is found in only three. The Aerometric Information Retrieval System (AIRS), the Urban Air Toxic Monitoring Program (UATMP), and the National Ambient Volatile Organic Compounds Data Base (NAVOC) all have data for 1,3-butadiene. The urban exposure data for 1,3-butadiene from the three databases are found in Appendix C and summarized in Table 7-7.

In the 1988 Aerometric Information Retrieval System (AIRS), 18 measurements of 1,3-butadiene were taken at 3 sites. These sites were in the cities listed below.

        Louisville, KY   Houston, TX
        Burlington, VT

The highest average was 2.45 µg/m^3 (1.11 ppb) at an suburban residential site in Houston, Texas. Six samples were collected at this site. Houston, 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 6 samples from Houston from the final average ppb for the entire program. The second highest average was 1.04 µg/m^3 (0.47 ppb) at a urban commercial site in Burlington, Vermont. Six samples were collected at this site. The lowest average was 0.97 µg/m^3 (0.44 ppb) at a urban industrial site in Louisville, Kentucky. Six samples were also collected at this site. The overall average of the averages for each site was 1.48 µg/m^3 (0.67 ppb). The removal of the 6 Houston samples changes the ambient mean level from 1.48 µg/m^3 (0.67 ppb) to 1.01 µg/m^3 (0.46 ppb).

In the 1990 Aerometric Information Retrieval System (AIRS), 101 measurements of 1,3-butadiene were taken at 7 sites. These sites were in the cities listed below.

        Detroit, MI   Houston, TX
        Arlington County, VA   Henrico County, VA
        Hampton, VA   Hopewell, VA
        Roanoke, VA

Table 7-7. Air Monitoring Results for 1,3-Butadiene.

Program	Years	Ambient Data[a] µg/m^3	Estimated Motor Vehicle Contribution[b] µg/m^3
AIRS	1988	1.01	0.56
	1990	0.47	0.26
	1991	0.22	0.12
UATMP	1989	0.46	0.26
	1990	0.31	0.17
NAVOC	1987	0.75	0.42

[a]Caution should be taken in comparing these numbers. The methods of averaging the data are not consistent between air monitoring databases. The sampling methodology is also inconsistent.

[b]The ambient data are adjusted to represent the motor vehicle contribution to the ambient concentration, which for 1,3-butadiene is estimated to be 56%, based on emissions inventory apportionment.

The highest average was 1.58 µg/m^3 (0.72 ppb) at an suburban residential site in Houston, Texas. Four samples were collected at this site. Due to the reasons cited above, the four samples from Houston, Texas were excluded from the final average ppb for the entire program. The second highest average was 0.73 µg/m^3 (0.33 ppb) at an urban commercial site in Detroit, Michigan. Nineteen samples were collected at this site. The lowest average was 0.29 µg/m^3 (0.13 ppb) at a suburban residential site in Hopewell, Virginia. Sixteen samples were also collected at this site. The overall average of the averages for each site (minus Houston, Texas) was 0.47 µg/m^3 (0.21 ppb).

In the 1991 Aerometric Information Retrieval System (AIRS), 117 measurements of 1,3-butadiene were taken at 6 sites. These sites were in the cities listed below.

Detroit, MI	Arlington County, VA
Henrico County, VA	Hampton, VA
Hopewell, VA	Roanoke, VA

The highest average was 0.27 µg/m^3 (0.12 ppb) at suburban residential sites in Henrico County and Roanoke, Virginia. Twenty-one and fourteen samples were collected, respectively, at each site. The lowest average was 0.13 µg/m^3 (0.06 ppb) at a suburban residential site in Hopewell, Virginia. Sixteen samples were collected at this site. The overall average of the averages for each site was 0.22 µg/m^3 (0.10 ppb).

In the 1989 Urban Air Toxics Monitoring Program (UATMP), 160 measurements of 1,3-butadiene were taken at 14 sites. These sites were in the cities listed below.

Baton Rouge, LA	Chicago, IL
Camden, NJ	Dallas, TX
Fort Lauderdale, FL	Houston, TX
Miami, FL	Pensacola, FL
St. Louis, MO	New Sauget, IL
Washington, D.C.	Wichita, KS

The highest average was 1.33 µg/m^3 (0.60 ppb) at a suburban residential site in Houston, Texas. Thirty-four samples were collected at this site. The lowest average was 0.11 µg/m^3 (0.05 ppb) at a suburban industrial site in Pensacola, Florida. Only seven samples were collected at this site. The overall average of the averages for each site was 0.46 µg/m^3 (0.21 ppb).

In the 1990 Urban Air Toxics Monitoring Program (UATMP), 349 measurements of 1,3-butadiene were taken at 12 sites. 1,3-Butadiene was identified in 106 of the samples. These sites were in the cities listed below.

Baton Rouge, LA   Chicago, IL
Camden, NJ   Houston, TX
Orlando, FL   Pensacola, FL
Port Neches, TX   Sauget, IL
Toledo, OH   Washington, D.C.
Wichita, KS

The highest average was 24.51 µg/m^3 (11.09 ppb) at a suburban
residential site in Port Neches, Texas. Twenty-eight samples
were collected at this site. 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. The second highest average was 1.04
µg/m^3 (0.47 ppb) at a suburban residential site in Houston,
Texas. Twenty-eight samples were collected at this site. The
lowest average was 2.73 µg/m^3 (0.06 ppb) that was measured at
five different sites. The overall average of the averages for
each site was 2.25 µg/m^3 (1.02 ppb). The removal of the 28 Port
Neches samples changes the ambient mean level from 2.25 µg/m^3
(1.02 ppb) to 0.31 µg/m^3 (0.14 ppb).

In the 1987 National Ambient Volatile Organic Compound
Database (NAVOC), 9 measurements of 1,3-butadiene were taken at 6
sites. These sites were in the cities listed below.

Bakersfield, CA   Concord, CA
Fremont, CA   Richmond, CA
San Jose, CA   Stockton, CA

The highest average was 1.33 µg/m^3 (0.60 ppb) at an urban site in
Fremont, California. Two samples were used for the average at
this site. The lowest average was 0.55 µg/m^3 (0.25 ppb) also at
an urban site in San Jose, California. Two samples were also
used for the average for this site. The overall average of the
averages for each site was 0.75 µg/m^3 (0.34 ppb).

HAPEM-MS assumes that the dispersion and atmospheric
chemistry of 1,3-butadiene is similar to CO. This assumption
would appear not to be valid for a reactive compound like 1,3-
butadiene, which is transformed in the atmosphere. To test the
reasonableness of the HAPEM-MS modeling results, the HAPEM-MS
results for 1990 are compared to ambient monitoring results for
recent years. Before comparing the HAPEM-MS results to the
ambient data, the ambient monitoring data must be adjusted to
represent the amount that is attributed to mobile sources. The
data derived from emission inventories estimate that 56% of the
ambient 1,3-butadiene can be apportioned to motor vehicles. The
numbers in the second column of Table 7.7 are 56% of the ambient
levels and thus represent estimated motor vehicle levels.

The motor vehicle apportionment of the ambient monitoring

data, presented in Table 7-7, ranges from 0.12 to 0.56 µg/m^3.
When the adjustment factor of 0.622 that was determined in
Section 5.5.2 is applied, this range becomes 0.08 to 0.35 µg/m^3.
The HAPEM-MS 1990 base control level of 0.48 µg/m^3 lies above
this range. Since the unit risk estimate for 1,3-butadiene is an
upper bound estimate, the upper end of the ambient range is used
to calculate cancer incidences. The HAPEM-MS 1990 base control
level must be multiplied by a factor of 0.73 to agree with the
upper end of the ambient data. All analysis based on the HAPEM-
MS ambient motor vehicle levels will have this factor applied.
Adjusted urban, rural, and nationwide exposures are found in
Table 7-8.

In an ambient monitoring study conducted by the California
Air Resources Board (CARB, 1992a) 20 monitoring sites were
established throughout the State of California to assess 1,3-
butadiene levels. The range of the averages of the six basins
detailed in this study was 0.49 to 0.93 µg/m^3 (0.22 to 0.42 ppb).
When this range is adjusted for the motor vehicle contribution
and integrated exposure it becomes 0.17 to 0.32 µg/m^3 (0.08 to
0.15 ppb). The upper end of this range compares favorably with
the adjusted HAPEM-MS exposure number.

The degree of confidence in the air monitoring programs,
especially the Urban Air Toxics Monitoring Program (UATMP),
appears to be high. The UATMP analyzed 1,3-butadiene using gas
chromatography/ multiple detector (GC/MD). The GC/MD compound
identifications were confirmed by analyzing about 15% of the 1989
UATMP samples by gas chromatography/mass spectrometer (GC/MS).
The GC/MS samples confirmed 94.1% of all the compound
identifications resulting from the initial analysis. UATMP also
determined the level of confidence in its 1,3-butadiene
identification analysis. The precision (percent coefficient of
variation, % CV) was calculated for the compound response ratio
in the sample and the compound response ratio in the standard.
It was determined that approximately 22% of the samples were
within ±20% CV, while 60% were below the 0.10 ppbv detection
limit (EPA, 1990).

As demonstrated in the section above, it is very clear that
there is a need for better ambient data and exposure methodology
for all the pollutants examined in this study. An individual's
annual exposure could be very different then the one number
presented in this study due to geographic and temporal variation
inherent in exposures. Actual exposure estimates need to take
this into account.

7.5.3 Short-Term Microenvironment Exposures

The primary emphasis for 1,3-butadiene exposure will be
exposure in microenvironments that are enclosed, increasing the
exposure to tailpipe emissions. These microenvironments include

Table 7-8.   Adjusted Annual Average HAPEM-MS Exposure Projections for 1,3-Butadiene.

Year-Scenario	Exposure ($\mu g/m^3$)		
	Urban	Rural	Nationwide
1990 Base Control	0.35	0.19	0.30
1995 Base Control	0.23	0.12	0.20
1995 Expanded Reformulated Fuel Use	0.23	0.12	0.20
2000 Base Control	0.19	0.10	0.16
2000 Expanded Reformulated Fuel Use	0.18	0.09	0.16
2000 Expanded Adoption of California Standards	0.18	0.09	0.16
2010 Base Control	0.20	0.11	0.18
2010 Expanded Reformulated Fuel Use	0.20	0.10	0.17
2010 Expanded Adoption of California Standards	0.19	0.10	0.16

in-vehicle and parking garage exposure, though, actual exposure information is only available for in-vehicle exposure. This information is taken from the Commuter's Exposure to Volatile Organic Compounds, Ozone, Carbon Monoxide, and Nitrogen Dioxide (Chan et al., 1989), which focused on the driver's exposure to VOC's in the Raleigh, NC area. See the information in Section 4.2 for more details about the methodology, and Section 5.5.3 for a description of the study.

The in-vehicle exposure level of 1,3-butadiene was determined in this study to have a mean of 3.0 $\mu g/m^3$ and a maximum measured level of 17.2 $\mu g/m^3$. Exterior to the vehicle, the mean was determined to also be 3.0 $\mu g/m^3$ with a maximum level of 6.9 $\mu g/m^3$. This compares to ambient levels of 0.31 to 1.48 $\mu g/m^3$ determined through air monitoring studies and presented in Table 7-7. Since for the majority of the population these are short-term acute exposures to 1,3-butadiene, the concern would be with non-cancer effects. Health information for non-cancer effects is very limited and no RfC has been developed by EPA. Inhalation of 1,3-butadiene is mildly toxic in humans at low concentrations (data on actual levels are not conclusive) and may result in a feeling of lethargy and drowsiness. At very high concentrations, 1,3-butadiene causes narcosis leading to respiratory paralysis and death. Please see Section 7.8 for more information on non-cancer effects.

Due to more stringent fuel and vehicle regulations, short-term exposure to 1,3-butadiene in microenvironments is expected to decrease in future years.

7.6 Carcinogenicity of 1,3-Butadiene and Unit Risk Estimates

7.6.1   Most Recent EPA Assessment

The information presented in Section 7.6.1 was obtained from the EPA document Mutagenicity and Carcinogenicity Assessment of 1,3-Butadiene (EPA, 1985), EPA's Integrated Risk Information System (IRIS) (EPA, 1992), the Motor Vehicle Air Toxics Health Information (Clement, 1991), as well as the primary sources cited in these documents. The carcinogenicity risk assessment for 1,3-butadiene was last updated on IRIS in January 1992, and contains data published through 1991. However, with the exception of a change in absorption factor (used to calculate the target dose) based on new pharmacokinetic data, the 1992 version of the 1,3-butadiene risk assessment on IRIS is based on the same study as the 1985 risk assessment. EPA's Office of Research and Development has just recently started the process to review the 1,3-butadiene risk assessment. Section 7.6.3 summarizes recent and ongoing research not included in the 1985 EPA risk assessment for 1,3-butadiene.

7.6.1.1   Description of Available Carcinogenicity Data

Genotoxicity

Three studies have shown 1,3-butadiene to be mutagenic for *Salmonella typhimurium* upon addition of mammalian hepatic (liver) homogenates for metabolism (de Meester et al., 1978, 1980; Poncelet et al., 1980). The weight of evidence available suggests that 1,3-butadiene is a promutagen in bacteria; its mutagenicity depends on metabolic activation by hepatic homogenates prepared from chemically induced animals. No whole-animal mutagenicity studies have been reported.

Pharmacokinetic and various types of toxicity studies indicate that the carcinogenic effects of 1,3-butadiene can be attributed to the metabolites 3,4-epoxybutene and/or 1,2,3,4-diepoxybutane. These metabolites, which are potent alkylating agents (chemically react with DNA), have been shown to be mutagenic and carcinogenic (Lawley and Brookes, 1967; Ehrenberg and Hussain, 1981). The metabolite, 3,4-epoxybutene, is a direct-acting mutagen in bacteria, and induces sister chromatid exchanges and chromosomal aberrations in mice (de Meester et al., 1978; Voogd et al. 1981, Hemminki et al., 1980).

1,2:3,4-Diepoxybutadiene is a bifunctional alkylating agent, and as such it can form cross-links between two strands of DNA. It is mutagenic in bacteria (Voogd et al., 1981, Wade et al., 1979), fungi (Olszewska and Kilbey, 1975; Luker and Kilbey, 1982), and the germ cells of *Drosophila* (Sankaranarayanan, 1983; Sankaranarayanan et al., 1983). It also induces DNA damage in cultured hamster cells and in mice (Perry and Evans, 1975; Conner et al., 1983), is clastogenic in fungi and cultured rat cells (Zaborowski et al., 1983; Dean and Hodson-Walker, 1979), and produces chromosome damage/breakage in *Drosophila* germ cells (Zimmering, 1983).

Under certain conditions, such as during rubber curing, 1,3-butadiene can dimerize (two molecules bonding together). The dimer was not mutagenic in the Salmonella preincubation assay in the presence of liver homogenates from chemically induced rats or hamsters (NTP, 1985). In contrast, the metabolites of the dimer are mutagenic or clastogenic in various in vitro bacterial and animal cell systems as a base-pair substitution mutagen (Murray and Cummins, 1979; Simmon and Baden, 1980; Truchi et al., 1981; Voogd et al., 1981). Therefore, the evidence indicates that 3,4-epoxybutene, diepoxybutane, and other mono- and diepoxide metabolites are mutagens/clastogens in microbes and animals.

Animal Data

In a chronic study conducted by the National Toxicology Program (NTP, 1984), B6C3F1 mice (50/sex/group) were exposed via inhalation to 0, 625, and 1,250 ppm 1,3-butadiene, 6 hours/day, 5

days/week. Because of excessive deaths, primarily due to
lymphoma, among treated animals, mice were sacrificed after 60-61
weeks instead of the planned lifetime exposure (2 years).
Histopathologic examination revealed an increased frequency of
primary tumors at both exposure levels. These tumors included
hemangiosarcomas (malignant tumors in the blood vessels) that
were found primarily in the heart, alveolar/bronchiolar adenomas
and carcinomas, and lymphomas. The incidence of acinar cell
carcinoma (mammary) and granulosa cell tumor (layer cells located
in the ovary) or ovarian carcinomas were increased in females in
the high-dose group. According to EPA (1985), NTP conducted an
audit of the study and reported that some genetic variation was
observed during 1981 in the male C3H parents of the mice used in
this study. The effect of genetic nonuniformity in the hybrid
mice on the study was unknown, but the results were considered
valid because of the use of matched concurrent controls. More
recent work by NTP is discussed in Section 7.6.3.

In a two-year study conducted by Hazleton Laboratories
Europe, Ltd. (1981) (later published as Owen et al., 1987, see
Section 7.6.3.2) Charles River CD rats (110/sex/group) inhaled 0,
1,000, and 8,000 ppm 1,3-butadiene, 6 hours/day, 5 days/week, for
111 weeks (males) and 105 weeks (females). The authors reported
significant increases in both common and uncommon tumors. There
was an increase in multiple mammary gland tumors in females for
all treatment groups, thyroid follicular adenoma and carcinoma in
the high-dose females, and Leydig cell (cells located in the
testicles believed to be responsible for secreting testosterone)
adenoma and carcinoma in the high-dose males. The report did not
include detailed histopathological evaluations and did not
perform independent data quality evaluation (EPA, 1985).
Therefore, uncertainty about the number of tissues examined
limited the usefulness of animal-to-human extrapolation. In
addition, the higher butadiene dimer (the combination of two
butadiene molecules) content of the material for rats might
contribute to the difference in the effective dose, although its
effect on the study results is unknown (EPA, 1985). The
incidence of tumors in hormonal-dependent tissues was greater in
rats, although it may have been masked by early deaths in mice.

Human Data

There were several epidemiological studies evaluating
mortality due to cancer in workers exposed to 1,3-butadiene.
These study results were inconsistent and limited because of
concurrent occupational exposures to other contaminants, usually
styrene (potential carcinogen and leukemogen), and the lack of
adequate exposure data on 1,3-butadiene concentrations.

Excess mortalities were reported in 6,678 male workers in a
rubber tire manufacturing plant in Akron, Ohio (McMichael et al.,
1974; 1976). During a 9-year follow-up period from 1964-1972,
statistically significant increases in deaths were due to stomach

and prostate cancer, lymphosarcoma, and leukemia, as well as diabetes mellitus and arteriosclerosis. The 1968 U.S. male population was used as the standard population. An age-standardized risk ratio of 6.2 for lymphatic and hematopoietic cancer was calculated for workers with at least 5 years of exposure. To further evaluate the cancer-specific deaths, McMichael et al. (1976) conducted a case-control study which indicated that certain cancers were significantly elevated in certain job classifications and with at least 5 years of exposure. Levels of exposure to 1,3-butadiene were not quantified.

In a historic prospective cohort study, increased incidence of lymphatic and hematopoietic cancers were reported in 8,938 males in the rubber manufacturing plant during 1964-1973 (Andjelkovich et al., 1976). Data were collected from company records, life insurance death claims, and bureaus of vital statistics. The increased mortality ratios were evaluated in relation to work areas by using the entire cohort as a reference group. EPA (1985) concluded that the study was limited because of the uncertainty regarding the duration that a subject worked in a specific job department (i.e., estimated duration ranged from 10% to 100% of employment) and the use of 1968 mortality data which may have underestimated expected deaths. Furthermore, the levels of exposure were not quantified.

Checkoway and Williams (1982) evaluated the same group of rubber manufacturing workers as the McMichael et al. (1976) case-control study. The objective of this study was to quantify exposure and to relate it to hematologic measurements. Air sampling of the plant and blood samples from the subjects were taken in May 1979. Time-weighted averages of 20.03 and 13.67 ppm were determined for 1,3-butadiene and styrene, respectively. No association was found between hematologic values and 1,3-butadiene exposure. Because the study was cross-sectional, excess cancer risk was not expected to be identified since subjects who may have developed cancers and left the job force were not available for evaluation. The study was also limited because air sampling could not be used to generalize past exposure levels. Furthermore, concurrent exposure to more than one potentially toxic chemical renders it impossible to associate any adverse health effects that may be seen with exposure to a particular chemical.

A retrospective cohort mortality study was conducted on two rubber plants in Texas by Meinhardt et al. (1982). The time-weighted average exposures of butadiene were 1.24 and 13.5 ppm for the two plants. Subjects were also concurrently exposed to styrene and benzene. Deaths due to lymphatic and hematopoietic cancers and lymphatic leukemia were exhibited, although they were not statistically significant. Results showed borderline significance for the subcohort employed during the batch process of the

production process. This finding may have been biased by
uncertainty in the number of deaths and/or factors in choosing
the study group (EPA, 1985).

There were no excess mortalities in a retrospective cohort
mortality study involving 8 styrene-butadiene rubber plants
(Matanoski et al., 1982). None of the SMRs were statistically
significant. According to EPA, information on the employees was
gathered from company records; however, this study evaluated less
than 50% of the population of the 8 plants and may have
underestimated the number of deaths.

7.6.1.2 Weight-of-Evidence Judgment of Data and EPA
Classification

1,3-Butadiene is classified by EPA as a Group B2, probable
human carcinogen using EPA's Proposed Guidelines for Carcinogen
Risk Assessment (EPA, 1984). This classification was based on
sufficient evidence from two species of rodents and inadequate
epidemiologic evidence, as described in Section 7.6.1.1 above.

The mouse inhalation study by NTP (1984) was considered the
primary study for calculating the cancer risk estimate of 1,3-
butadiene (EPA, 1985). It was the most appropriate choice
because the study was well-conducted and tumors were observed in
animals of both treatment groups. The rat bioassay (Hazleton
Laboratories, 1981) had deficiencies that limited its use as the
primary data set for animal-to-human extrapolation. According to
EPA, the quality of the study and its results have not been peer-
reviewed or published (this study has since been published as
Owen et al., 1987, see Section 7.6.3.2), the histopathology
report was not available, and the calculated slope factors for
male and female rats were limited from a modeling standpoint
since it had only one effective dose. In spite of the fact that
this study has been published, EPA still considers it inadequate
for risk assessment because of reporting problems, and because
the pharmacokinetic analysis in Owens et al. (1987) is considered
by EPA to indicate that the effective doses were the same for
both treatment groups.

The human studies were not used for determining unit risk
because there were inadequate data on the carcinogenicity of 1,3-
butadiene, a lack of exposure information, and concurrent
exposures to several other possible carcinogens (i.e., styrene)
to the workers. However, EPA did conduct quantitative estimates
based on mouse-to-man extrapolation to predict human responses in
several epidemiologic studies. Comparisons were hampered by
scarcity of information concerning actual exposures, age
distributions, and work histories. Considering the uncertainties
in the human exposure data, the estimate based on animal
extrapolation is consistent and the best that can be achieved.

7.6.1.3   Data Sets Used For Unit Risk Estimates

A summary of the data set used to calculate the cancer unit risk estimate for 1,3-butadiene (EPA, 1985) is presented in Table 7-9. EPA used both male and female mouse data sets from the NTP (1984) study in determining the unit cancer risk estimates. Animals with at least one of the statistically significant increased tumors or tumors considered unusual were included in the data set. The data set had an adequate number of animals per treatment group and the estimates across species for females were relatively close which supports the confidence of the slope factor.
However, only high doses were tested so the true shape of the dose-response curve at low environmental levels is not known.

7.6.1.4   Dose-Response Model Used

The low-dose linear multistage extrapolation model was used for calculating the unit risk estimate (see Appendix F for a description of the linearized multistage model), although alternative models were discussed but found inappropriate by EPA. This model gave a conservative estimate while the other models result in a lower risk estimate.

Because 1,3-butadiene is considered a partially soluble vapor, the average dose/day is proportional to the O_2 consumption and is proportional to two-thirds of the weight and also to gas solubility in body fluids (expressed as absorption coefficient). All three factors listed above must be utilized when determining average dose/day. In the absence of experimental information, the absorption fraction is assumed to be the same for all species. In
order to convert to internal dose in animals, EPA used the absorption study by Bond et al. (1986) which reported 20% absorption of 1,3-butadiene following inhalation exposure in rats and mice.

Because mice were exposed to 1,3-butadiene for a less-than-lifetime duration, an adjustment was made for extrapolation from the 60-61 weeks in the NTP mouse study to two full years (lifetime exposure).

7.6.1.5   Unit Risk Estimates (UCL and MLE)

The upper-limit unit risks were 3.4×10^{-1} ppm^{-1} ($3.8\times10^{-4}[\mu g/m^3]^{-1}$) for male mice and 1.9×10^{-1} ppm^{-1} (2.1×10^{-4} $[\mu g/m^3]^{-1}$) for female mice using a 20% absorption rate at low exposures. The geometric mean of unit risks was 2.5×10^{-1} ppm^{-1} (2.8×10^{-4} $[\mu g/m^3]^{-1}$) for the two mouse unit risks (EPA, 1992). Calculating geometric means of several unit risk estimates is standard EPA policy to derive a single unit risk estimate. However, the unit risk should not be used if air concentrations exceed 16 $\mu g/m^3$. The maximum likelihood estimate

(MLE) of unit risk based on several tumor types observed in male

Table 7-9.   Summary of Data Set Used to Calculate Unit Risk Estimate for 1,3-Butadiene.

Source	Test Animal	Tumor Type	Administered Dose (ppm)	Internal Dose (mg/kg/day)	Tumor Incidence
NTP (1984)	B6C3F1 mice	Hemangiosarcomas of the heart, lymphomas, and alveolar/ bronchiolar adenomas/ carcinomas	0	0	2/50 (male) 4/48 (female)
			625	18.4	43/49 (male) 31/48 (female)
			1250	27.8	40/45 (male) 45/49 (female)

and female mouse NTP data for a 1 ppm continuous lifetime exposure in the air is 2.5×10^{-2} (2.8×10^{-5} $[\mu g/m^3]^{-1}$).

The unit risk estimates in EPA (1985) were different from those reported in EPA (1992) because the values were calculated using the absorption data from the 1985 NTP absorption study which reported an absorption rate of 54% in mice and rats. Therefore, according to EPA (1985), calculations from the NTP (1985) study resulted in an inhalation unit risk estimate of 9.1×10^{-1} $(ppm)^{-1}$ (1.0×10^{-3} $[\mu g/m^3]^{-1}$) for males and 4.5×10^{-1} $(ppm)^{-1}$ (5.0×10^{-4} $[\mu g/m^3]^{-1}$) for females with a geometric mean of 6.4×10^{-1} $(ppm)^{-1}$ (7.2×10^{-4} $[\mu g/m^3]^{-1}$). In this case, the unit risk was not to be used if the air concentration exceeded 40 $\mu g/m^3$ since the slope factor may differ from that stated at higher concentrations.

7.6.2   Other Views and Risk Estimates

This section presents alternate views and/or risk assessments for 1,3-butadiene. These alternate risk assessments are summarized in Table 7-10. All alternate risk assessments are expressed as UCLs; no MLEs are presented.

International Agency for Research on Cancer (IARC)

IARC has classified 1,3-butadiene as a Group 2A carcinogen. A Group 2A carcinogen is defined as an agent that is *probably* carcinogenic to humans. This classification is based on limited evidence for carcinogenicity in humans and sufficient evidence for carcinogenicity in animals (IARC, 1992).

IARC reviewed the available human data and concluded that these studies do provide some evidence that occupational exposure to 1,3-butadiene is associated with an excess of leukemias and lymphomas. However, these data are considered by IARC to be limited because concomitant exposure to other potentially carcinogenic agents (e.g., styrene and benzene) preclude any definitive causative link to be drawn between exposure to 1,3-butadiene and cancer.

IARC concluded that the available animal data provide sufficient evidence of the carcinogenicity of 1,3-butadiene. These data consist of inhalation studies in mice and rats conducted by NTP (1984) and Hazleton Laboratories Europe, Ltd. (Owen et al., 1987). Details of these studies were mentioned previously in Section 7.6.1.1. No unit risk was determined by IARC.

California Air Resources Board (CARB)

CARB (1992a,b) has performed an assessment of the carcinogenic risk of 1,3-butadiene using both the mouse (NTP, 1984; Melnick et al., 1990) and rat (Owen et al., 1987) data in

the linearized multistage model. As EPA did, total significant
tumor incidences

Table 7-10.   **Comparison of 1,3-Butadiene Unit Risk Estimates and Calculated Doses for an Extra Lifetime Cancer Risk of 1×10^{-6}.**

Source	Tumor Types	Classification	Cancer Unit Risk Estimate $(\mu g/m^3)^{-1}$	Dose $(\mu g/m^3)$ For a Cancer Risk of 1×10^{-6}
OSHA (1990)[b]	Pooled female mouse tumors, multiple types[c]	Human Carcinogen	5.8×10^{-6}	1.7×10^{-1}
	Pooled female mouse hemangiosarcomas		2.7×10^{-6}	3.7×10^{-1}
	Pooled female rat tumors[d]		7.5×10^{-6}	1.3×10^{-1}
EPA (1985)	Pooled male and female mouse tumors	Group B2[e]	7.2×10^{-4f}	1.4×10^{-3}
	Pooled male rat tumors		4.7×10^{-4}	2.1×10^{-3}
	Pooled female rat tumors		6.2×10^{-4}	1.6×10^{-3}
ICF (1986)	Male mouse lymphomas	–	3.4×10^{-3}	2.9×10^{-4}
	Female mouse liver tumors		2.6×10^{-4}	3.8×10^{-3}
Turnbull et al. (1990) (Environ)	Pooled male rat tumors[g]	–	5.9×10^{-7}	$1.7 \times 10^{+0}$
	Pooled female rat tumors[h]		5.1×10^{-7}	$2.0 \times 10^{+0}$
EPA (1992)	Pooled female mouse data	Group B2	2.8×10^{-4}	3.5×10^{-3}

Table 7-10. Continued.

Source	Tumor Type	Classification	Cancer Unit Risk Estimate $(\mu g/m^3)^{-1}$	Dose $(\mu g/m^3)$ For a Cancer Risk of 1×10^{-6}
Hattis and Watson (1987)	Male rat-total tumors	-	1.1×10^{-7}	$9.3\times10^{+0}$
	Female rat-total tumors		1.3×10^{-6}	7.6×10^{-1}
	Male mice-total tumors		2.3×10^{-5}	4.5×10^{-2}
	Female mice-total tumors		1.7×10^{-5}	6.0×10^{-2}
IARC (1992)	Multiple tumors	Group 2A[i]	-	-
CARB (1992b)	Total rat tumors (less mammary fibroadenomas and uterine tumors)	-	4.4×10^{-6}	2.3×10^{-1}
CARB (1992b)	Total mouse tumors	-	1.7×10^{-4}	6.0×10^{-3}

[a]MLEs are not presented because they were not always calculated by the various organizations. Furthermore, EPA dose not generally compare MLEs based on animal data because of the high variability associated with these numbers. Therefore, they are of little value.
[b]Source: Grossman and Martonik, 1990. Based on estimates of extra risk per 10,000 for a lifetime occupational exposure. The following assumptions were made: absorption at low doses is 54%, adult body weight is 70 kg, adult breathing rate is 10 m^3/8-hour day, exposure is for 250 days/year for 45 years of a 74 year lifetime.
[c]Incidence of lymphoma excluded from pooled tumor incidence.
[d]High-dose group dropped from the analysis.
[e]Group B2 = Probable Human Carcinogen
[f]UCLs is the geometric mean of the UCLs estimated from the male mouse data and the female mouse data.
[g]Incidence of Zymbal gland carcinoma excluded from pooled tumor incidence.
[h]Incidence of mammary fibroadenoma excluded from pooled tumor incidence.
[i]Group 2A = Probable human carcinogen.

for each species and sex were used instead of individual site-specific tumor incidences because CARB believed that tumors which rapidly resulted in animal mortality may have masked the development of tumors at other, possibly more sensitive sites.

CARB concluded that, for use in risk assessment, the quality of the Melnick et al. (1990) is superior to that of the rat data. The primary reasons for this conclusion are: 1) the use of lower, more relevant dose levels in the Melnick et al. (1990) study; 2) the use of five dose levels in the Melnick et al. (1990) study, compared to two in the rat study; 3) the presence of two mouse studies; 4) the fact that the rat study has not been replicated; 5) the consistency in sites of carcinogenicity between the two mouse
studies; 6) the greater detail in the available mouse data which allows in-depth analysis; and 7) suggestions from limited epidemiological observations that 1,3-butadiene exposure may be associated in humans with lymphatic and hematopoietic cancers, effects that were seen in mice. The continuous internal dose (i.e., the dose of butadiene that is retained in the animal) was considered by CARB to be the best estimate of delivered dose (i.e., the dose of butadiene that is actually available at target tissue sites) available. The continuous internal doses were derived from the applied external doses using the data of Bond et al. (1986).

CARB calculated the theoretical human risk associated with a continuous lifetime exposure to butadiene (q_1^*, 95% UCL) as 1.7×10^{-4} [$\mu g/m^3$]$^{-1}$ based on the mouse inhalation study of Melnick et al. (1990). The risk based on the mouse data is comparable to EPA's current unit risk of 2.8×10^{-4} [$\mu g/m^3$]$^{-1}$, which is a geometric mean of the unit risks derived from the male and female mouse data sets (EPA, 1992, see Section 7.6.1.5).

CARB also fit the data to various other models. They concluded that the data gave better fits to the linearized multistage model or the GLOBAL 86 version of the linearized

multistage model than to the other models. They concluded that the mouse provides the best estimate for the upper bound for plausible excess cancer risk to humans.

Based on the findings of 1,3-butadiene-induced carcinogenicity and the results of the risk assessment, CARB finds that, at ambient concentrations, 1,3-butadiene is an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health.

Occupational Safety and Health Administration (OSHA)

OSHA contracted with ICF/Clement (ICF/Clement, 1986) to conduct a risk assessment on 1,3-butadiene to be used as rule-

making support in setting a revised occupational exposure
standard for this chemical. ICF/Clement's risk assessment
differed from EPA (1985) only by the data set used, and the final
adjusted doses used in the risk assessment. ICF/Clement
expressed dose in parts per million, employed the linearized
multistage model for low-dose extrapolation, adjusted for less-
than-lifetime exposure, and adjusted the experimental dose for
absorption. This adjustment differed from that used by EPA
(1985) and was done using a line generated by plotting a log-log
scale based on data reported in EPA (1985) that indicated
retention of 1,3-butadiene is inversely related to dose. As in
the EPA (1985) risk assessment, the data from the NTP (1984)
mouse bioassay were used to calculate the unit risk for 1,3-
butadiene. However, EPA used pooled tumor incidence data for
mice, whereas ICF/Clement used site-specific individual tumor
data. ICF/Clement claimed that the multistage model is based on
the observation that cancer is a progressive disease that
develops in stages. Data are available suggesting that the
number of stages or the stage at which a particular carcinogen
acts may vary among different organ systems in the body.
Therefore, ICF/Clement concluded that the use of pooled tumor
data is not well-justified on theoretical grounds, even though
EPA felt differently when it developed a potency factor for 1,3-
butadiene. The results of these analyses for the worst case
(male mouse lymphoma) and best case (female mouse liver tumors),
as compared to those calculated by EPA are presented in Table 7-
10.

OSHA also conducted its own risk assessment of 1,3-butadiene
(Grossman and Martonik, 1990). In this risk assessment,
experimental dose was measured in milligrams per kilogram per day
and adjusted for absorption (method not specified). The risks
were derived using both pooled tumor and site-specific tumor
incidence data for both mice (NTP, 1984) and rats (Owen et al.,
1987), using the multistage model. The results of these
analyses, as compared to those performed by EPA (1985),
ICF/Clement (1986), CARB (1991), and Environ (see section below)
are presented in Table 7.10.

Chemical Manufacturer's Association (CMA)

CMA contracted with Environ Corporation to perform an
independent assessment of the potential risk to workers from
exposure to 1,3-butadiene (Turnbull et al., 1990). Environ's
risk assessment departed from that of EPA (EPA, 1985) with regard
to the data set used and the low dose extrapolation models
employed. However, like EPA, (1985), they measured dose in
milligrams per kilogram per day and adjusted the experimental
dose of 1,3-butadiene for retained dose, assuming an absorption
of 54%, regardless of dose. EPA (1992) has since revised their
calculation of the unit risk by assuming a 20% absorption rate at
low exposures as per Cote and Bayard (1990) (see discussion in
Section 7.6.1.5). Environ disagreed with the choice of the mouse

data from the NTP (1984) study as the basis for the unit risk for a number of reasons (Environ, 1987).

In an attempt to address some of the perceived uncertainties with the NTP (1984) mouse data listed in Environ 1987, Environ employed the following procedures. Separate extrapolations were conducted based on tumor-bearing animals having any of the tumors that showed a significant increase in incidence in one or both of the treated groups, and on the same animals except those that developed lymphoma. To account for the less-than-lifetime exposure duration in the mouse study, the Hartley-Sielken general product model was used. The results of these calculations are summarized in Table 7-10.

Environ also calculated unit risks on the rat data from the Hazleton Laboratories Europe, Ltd. study (Owen et al., 1987) using three different low-dose extrapolation models. However, the tumor incidences from this study that were used by Environ differed from those used by EPA for unexplained reasons. The results of the unit risk estimates resulting from the use of the multistage model only are presented in Table 7-10.

The results of these analyses generally predicted lower risks than those predicted by EPA, and indicate that mice appear to be at a greater risk (by a factor of 5-fold to 40-fold) than rats. Environ noted that some of this species difference (3-fold to 5-fold) may be due to differences in metabolism, and that mice metabolize 1,3-butadiene to the carcinogenic epoxide derivatives at a higher rate than rats (though not mentioned by Environ in its analysis, this species difference could reflect decreased elimination of reactive intermediates in mice as compared to rats). In addition, Environ predicted the lifetime risk to humans using several exposure levels based on all of the risk estimates derived above. This risk was then used to calculate the expected number of extra deaths from lymphopoietic cancer and compared them to the actual number of deaths observed in the Matanoski et al. (1982) cohort. This exercise led to the conclusion that these risks were inconsistent with the observations made in occupational studies, i.e., the animal risks (particularly those based on the mouse data) overpredicted the risk to humans. However, it should be noted that the revised risk estimate for butadiene cited in EPA (1992) that incorporates a new absorption factor results in the prediction of 40% less excess cancer cases (Cote and Bayard, 1990). Therefore, the conclusions of Turnbull et al. (1990) with regard to overpredicting the risk to humans exposed to butadiene may no longer be valid.

Hattis and Wasson (1987)

Hattis and Wasson (1987) developed a pharmacokinetic/mechanism-based model for butadiene in an attempt to further refine the estimate of the "effective" dose for 1,3-

butadiene to be used in risk assessment. This model estimates
the effective dose "as the total amount of butadiene that
eventually undergoes at least the first step of metabolic
activation to 3,4-epoxy-1-butene". Development of this model
required an estimation of the octanol/water partition coefficient
from chemical structural information and a water/air partition
coefficient from aqueous solubility information as a function of
temperature so that tissue/blood and blood/air partition
coefficients could be estimated. Contrary to previous
assumptions that butadiene was metabolized only in the liver, the
model was structured to allow butadiene metabolism throughout the
"vessel-rich group" (kidneys, viscera, and brain) in addition to
the liver. Finally, maximal metabolic rates for humans were
scaled using general metabolic rates -- (body weight)$^{.75}$. Using
their model, Hattis and Wasson (1987) calculated rodent
metabolized doses that were 2-4.5 times the absorbed doses used
in earlier (i.e., EPA [1985] and Environ [1987]) risk
assessments. This larger metabolized dose effectively reduces
the apparent carcinogenic potency of butadiene as compared to the
earlier risk assessments. Another factor that reduces the
carcinogenic risk of butadiene is the fact that this model
predicts that net absorption will represent only about 11-15% of
the butadiene reaching the alveoli over an 8-hour period, and
only 8-10.5% of total inhaled butadiene.

Risk assessments conducted prior to Hattis and Wasson's work
assumed 50% of total inhaled butadiene would be absorbed by
humans at low doses. (However, in the most current EPA
assessment [1992], an absorption factor of 20% is used, see
discussions above). This difference results in a further
reduction of human delivered dose, and therefore, risk. Hattis
and Wasson (1987) also differed from the EPA (1985) and Environ
(1987) risk assessments in the manner in which they treated tumor
incidence. Rather than add up the tumor-bearing animals at all
sites with statistically significant tumor increases before
calculating risk, they calculated separate risks from each
individual site and then added up the overall expected risks from
all of the sites at the end.[1] The effect of such an approach is
most likely to overestimate the risk. The UCLs calculated using
the effective doses estimated with their model as compared to the
UCLs calculated by EPA, CARB, ICF/Clement, OHSA, and Environ are
summarized in Table 7-10.

<u>National Institute for Occupational Safety and Health (NIOSH)</u>

NIOSH (Dankovic et al., 1991) developed a quantitative risk

---

[1]While it is appropriate and acceptable to consider each tumor separately,
model each response, and then combine the probabilities at the end to arrive at
an overall expected risk, it is not correct to simply add the individual tumor
risks. It is correct to add the individual risks together and then subtract out
the product of the risks to arrive at the overall risk, and this is most likely
what Hattis and Wasson (1987) actually did, as evidenced by the numbers presented
in their table.

assessment of 1,3-butadiene based on the NTP bioassay in B6C3F$_1$ mice of Melnick et al. (1990). The risk assessment utilized the data from the published report as well as the data on the time of death and tumor status of each individual mouse in the study. The NIOSH study also chose to use exposure concentration instead of internal dose and the Weibull time-to-tumor model to determine their risk estimate.

Excess risk estimates were derived from fitting the one-stage, two-stage, and three-stage Weibull time-to-tumor models to the seven individual tumor types observed in the male mice, and to the nine individual tumor types observed in the female mice. Overall, the estimate yielding the largest extrapolated human risks at low exposure concentrations, that is, the most sensitive site, was the female mouse lung. Based on this site, the projected excess risk for a person occupationally exposed to 2 ppm 1,3-butadiene, for an entire working lifetime, is estimated to be 597 cases of cancer per 10,000 (5.97×10^{-2}), or approximately 6 per 100.

Caution must be taken in comparing this number to the previously stated risk estimates summarized in Table 7-10. The risk estimates in Table 7-10 are based on a 70 year lifetime exposure to 1 μg/m^3 of 1,3-butadiene whereas, the NIOSH risk estimate is based on a working lifetime exposure of 2 ppm (4.42×10^3 μg/m^3) 1,3-butadiene.

7.6.3   Recent and Ongoing Research

7.6.3.1   Genotoxicity

Two new studies were published after the EPA assessment that supported the observation that metabolites of 1,3-butadiene are genotoxic (Gervasi et al., 1985; Sharief et al., 1986). The study by Gervasi et al. (1985) observed that 1,2:3,4-diepoxybutane is a potent mutagen in the *S. typhimurium* mammalian microsome assay. Gervasi et al. (1985) also demonstrated that the potency of 1,2:3,4-diepoxybutane in this assay correlated well with the alkylating ability of this compound using nicotinamide as a substrate. Sharief et al. (1986) examined the *in vivo* genotoxicity of another 1,3-butadiene metabolite, 1,2-epoxybutene-3. A single intraperitoneal injection of 1,2-epoxybutene-3, at doses as low as 25 mg/kg, produced a significantly increased frequency of sister chromatid exchange (SCE) and chromosomal aberrations in bone marrow cells of C57B1/6 mice.

At the time of the EPA assessment (EPA, 1985), no *in vivo* studies of the genotoxicity of 1,3-butadiene were available for review. A number of inhalation studies have since been completed that examine the genotoxic effects of 1,3-butadiene exposure. For example, exposure of rats and mice to concentrations of 1,3-butadiene ranging from 10 to 10,000 ppm for 6 hr/day for 2 days

produced no increase in the frequency of micronucleus (MN) induction or SCE in bone marrow of Sprague-Dawley rats, but significantly increased the frequency of MN and SCE in bone marrow of B6C3F1 mice at doses as low as 100 ppm (Choy et al., 1986; Cunningham et al., 1986).[2] No increase in MN or SCE was observed in the B6C3F1 mice at 50 ppm. Exposure of B6C3F1 mice to concentrations of 1,3-butadiene ranging from 6.25 to 625 ppm for a somewhat longer period (6 hr/day, 5 days/week, for 2 weeks) revealed significant increases in SCE at 6.25 ppm, MN at 62.5 ppm, and chromosomal aberrations at 625 ppm in bone marrow (Tice et al., 1987). Chromosomal aberrations were predominantly chromatid-type breaks and exchanges. Increases in MN in peripheral blood were observed at doses of 1,3-butadiene as low as 6.25 ppm following longer-term exposure (for 6 hr/day, 5 days/week, for 13 weeks) (Jauhar et al., 1988). The potent genotoxicity of 1,3-butadiene in the mouse compared with the absence or low level of such effects in rats are consistent with the relative carcinogenic effects in these two species.

The strain specificity of the genotoxic effects in the mouse was tested by comparing chromosomal damage in B6C3F1 mice with that seen in NIH Swiss mice (Irons et al. 1987a). After a single 6-hour exposure to 1,250 ppm 1,3-butadiene, a high frequency of chromosomal aberrations, chromatid breaks, and chromatid and isochromatid gaps were seen in both strains of mice.[3] The NIH Swiss mouse does not possess murine leukemia virus, indicating that the genotoxicity of 1,3-butadiene is not dependent on the presence of this virus. However, the virus may play a role in the expression of murine leukemogenesis in the B6C3F1 strain.

Exposure of B6C3F1 mice and Wistar rats to (^{14}C)-1,3-butadiene (approximately 700 ppm for 4-7 hours) resulted in covalent binding of the radioactivity to liver nucleoproteins (a combination of a nucleic acid and a protein that is found in cell nuclei) and DNA in both species (Kreiling et al., 1986a). The alkylation of nucleoproteins was approximately twice as high in mice as in rats. The degree of alkylation was proportional to the different rates of metabolism of 1,3-butadiene in these two species. In contrast, the incorporation of radioactivity into DNA was approximately equal in both mice and rats. It is unclear to what extent the incorporation of radioactivity in DNA represented alkylation of nucleosides or metabolic incorporation into nucleosides. However, an alkylation product of guanine, 7-

---

[2]Micronuclei are formed after cell division when pieces of chromosomes do not get included within either nucleus of the newly formed cells. Sister chromatid exchange occurs when pieces of DNA break off and reattach to another piece of DNA. An increased frequency of micronuclei or SCE indicates chromosome breakage.

[3]A chromatid gap is a short missing region of DNA in one strand of a dividing chromosome. An isochromatid gap is a short missing region of DNA in one strand of an abnormally dividing (i.e., two strands break instead of each of the strands separating intact) chromosome.

(1-hydroxy-3-buten-2-yl) guanine was identified in mouse liver DNA after inhalation exposure to 1,3-butadiene (Laib and Kreiling, 1987).

A dominant lethal study in CD-1 mice was performed following inhalation exposure of males to concentrations of 1,3-butadiene ranging from 200 to 5,000 ppm for 6 hr/day for 5 days (Hackett et al., 1988b). A significant increase in intrauterine deaths was observed in females bred with males exposed to 1,000 ppm but not in females bred with males exposed to 5,000 ppm.

Cytogenetic monitoring of 1,3-butadiene rubber workers was reported in an abstract by Zhou et al. (1986). No significant increase in chromosomal aberrations or SCE in peripheral lymphocytes was observed when a group of 30 styrene-butadiene workers were compared with matched controls. Sex, age, and smoking status were considered in the analysis. However, 1,3-butadiene exposure levels were not measured and workers may have been exposed to toluene.

7.6.3.2   Pharmacokinetics

1,3-Butadiene is a carcinogen in both rats and mice, with mice being substantially more sensitive than rats (Csanády and Bond, 1991a). In the development of the pharmacokinetic model by CIIT, both *in vitro* and *in vivo* studies have demonstrated that 1,3-butadiene is metabolized by cytochrome P-450 to 1,2-epoxy-3-butene (butadiene monoepoxide, BMO). Further metabolic activity may transform BMO to two other metabolites, 1,2-epoxy-3,4-butanediol and diepozybutane (DEB). All three epoxides can potentially interact with DNA (Bryant and Osterman-Golkar, 1991). Not all of the metabolites of 1,3-butadiene have been identified yet, and those that have been identified, there has been limited pharmacokinetic testing.

Differences in the pharmacokinetics of 1,3-butadiene in mice and rats have been more closely examined in recent studies in an effort to explain the differences in the carcinogenic potency of 1,3-butadiene in these two species.

Many studies (Bond et al., 1986, 1987, 1988; Deutschmann and Laib, 1989; Kreiling et al., 1986b, 1987, 1988; Schmidt and Loeser, 1985, 1986; Jelitto et al., 1989) suggest that differences in species carcinogenicity susceptibility may be related to differences in 1,3-butadiene metabolism. When compared to the rat, mice have both a higher rate of 1,2-epoxybutene-3 synthesis and presence of DNA adducts, as well as a limited ability to detoxify this metabolite.

A physiologically-based, pharmacokinetics model for 1,3-butadiene exposure in rats and mice, based on the conversion of 1,3-butadiene to 1,2-epoxybutene-3, was developed by Hattis and Wasson (1987) utilizing blood butadiene concentrations as

described by Bond et al. (1986) and metabolic rates as described by Kreiling et al. (1986b). For this model, blood/air and tissue/air partition coefficients were estimated from structural and solubility information. According to this model, however, differences in pharmacokinetics failed to account for the differences in carcinogenicity of 1,3-butadiene in these two species.

Recent data from Csanády and Bond (1991b) indicate that the maximum rates of 1,3-butadiene metabolism in liver microsome isolated from humans, B6C3F$_1$ mice, and Sprague-Dawley rats (all male) are in the ratio of approximately 3:3:1. However, the investigators found that the key ratio that governs metabolic rates at low concentrations was 5-fold greater in mouse liver than in human liver. This means that the mouse produces more in the way of potential adduct forming metabolite (BMO), and thus is considered a more susceptible species. Csanády and Bond (1991b) also report that B6C3F$_1$ mouse lung microsomes are much more active than human lung microsomes in metabolizing 1,3-butadiene to BMO, with the key metabolic ratio in the mouse lung being approximately 6-fold greater than in the human lung.

In a recent presentation by CIIT (Recio et al., 1991) the *in vivo* mutagenicity of 1,3-butadiene was assessed in lung, liver, and bone marrow using a transgenic mutagenicity assay. It was found that the overall activation (by oxidation) of 1,3-butadiene to BMO was significantly higher for mice, especially in the lung. The detoxification of BMO (by hydrolysis) is slower in the mouse than in the rat or the human; thus, this correlates with the higher carcinogenicity sensitivity of mice than rats to 1,3-butadiene.

7.6.3.3   Carcinogenicity - Animal Studies

Additional information regarding the carcinogenicity of 1,3-butadiene in animals has been presented since the EPA mutagenicity and carcinogenicity assessment of 1,3-butadiene was performed in 1985. For example, another long-term inhalation study of 1,3-butadiene in B6C3F1 mice was initiated. The need for another mouse carcinogenicity study arose because the study that was originally evaluated in the EPA assessment demonstrated a strong multiple-organ carcinogenic response to 1,3-butadiene at exposure concentrations of 625 and 1,250 ppm (Huff et al., 1985), but clear dose-response relationships were not established and the study was terminated after 60 weeks of exposure because of reduced survival due to fatal tumors. Therefore, a study that examined lower exposure concentrations (6.25-625 ppm) was initiated. Preliminary results from that study through week 65 were reported (Melnick et al., 1988, 1989a, 1989b). After 65 weeks of exposure, 73/90 males and 80/90 females exposed to 625 ppm have died. The primary lesion observed in these animals was lymphocytic lymphoma. This was more prevalent in the males than in females. Other types of cancer observed in the high-dose

animals included hemangiosarcoma of the heart, squamous cell
neoplasms in the forestomach, alveolar-bronchiolar neoplasms,
adenoma of the Harderian gland, mammary gland adenocarcinoma, and
granulosa cell neoplasms of the ovary. Elevated incidences of
these neoplasms were also seen at 200 ppm 1,3-butadiene.
Alveolar-bronchiolar neoplasms of the lung in females were
increased above the incidence in controls at all concentrations
of 1,3-butadiene tested.

As part of this study, three groups of animals were also
exposed for limited periods of time to study the relationship
between exposure levels and duration of exposures on butadiene-
induced carcinogenicity. The groups consisted of male mice
exposed to 625 ppm for either 13 weeks or 26 weeks and mice
exposed to 312 ppm for 52 weeks. The animals were then held
until 65 weeks from the start of exposure. By week 65 of the
study, the incidence of lymphocytic lymphoma in animals exposed
to 625 ppm for 26 weeks (60%) was twice that observed in animals
exposed to 625 ppm for 13 weeks (30%), but was much greater than
the incidence in animals exposed to 312 ppm for 52 weeks (6%).
Thus, the multiple of the exposure concentration times the
exposure duration did not predict the incidence of lymphocytic
lymphoma in these mice. However, this study revealed that the
early incidence of fatal lymphocytic lymphoma in the high-dose
animals appeared to limit the expression of tumors at other
sites. Substantially higher levels of some tumor types were
observed in the dose group with low levels of lymphatic lymphoma
than in the dose group with high levels of lymphatic lymphoma.
For example, a much higher incidence of hemangiosarcoma of the
heart was observed in mice exposed to 312 ppm for 52 weeks (30%)
than in mice exposed to 625 ppm for 26 weeks (10%). Other tumor
types observed at a higher incidence in mice that survived 45-65
weeks that were not observed in the NTP study because of early
deaths due to lymphatic lymphoma include squamous cell neoplasms
of the forestomach, alveolar-bronchiolar neoplasms, Harderian
gland adenomas, adenocarcinomas of the mammary gland, granulosa
cell neoplasms of the ovary, and hepatocellular neoplasms. This
study is significant in that it demonstrated that: (1) exposure
to lower levels of 1,3-butadiene than those used in the study
that served as the basis for the EPA risk assessment allows the
expression of neoplasms at other sites because of a lower number
of early mortalities; (2) a clearer dose response relationship
for 1,3-butadiene-induced lymphocytic lymphomas was obtained
using the lower exposure levels because of increased survival;
and, (3) the multiple of exposure duration and concentration does
not predict the incidence of lymphocytic lymphomas. These
findings are relevant to the current EPA assessment of 1,3-
butadiene carcinogenicity because they demonstrate that the
induction of neoplasms in mice at multiple sites (i.e., some that
were not considered in the current assessment) occurs at lower
concentration levels than those used to derive the cancer potency
factor.

A study characterizing the lymphomas observed in B6C3F1 mice exposed to 1,250 ppm 1,3-butadiene for 28 to 45 weeks reported that the lymphomas consisted of well-differentiated lymphoblasts of T-cell origin (i.e., cells that develop into lymphocytes that subsequently migrate to the thymus gland) with variable but elevated levels of murine leukemic virus antigens (virus proteins found in mice that cause leukemia) (Irons et al., 1986c). In order to test the role of the endogenous retrovirus (i.e., a type of virus that is known to cause cancer), murine leukemic virus, in the development of the thymic lymphoma/leukemia in B6C3F1 mice, NIH Swiss mice (which do not express the retrovirus) were exposed to 1,3-butadiene under identical conditions as the B6C3F1 mice (Irons et al., 1989). This study revealed that B6C3F1 and NIH Swiss mice exposed to 1,250 ppm 6 hours/day, 5 days/week, for 1 year had similar increases in chromosomal aberrations and micronuclei in bone marrow and micronuclei in the peripheral blood, but NIH Swiss mice had a much lower incidence of lymphoma (14%) than did the B6C3F1 mice (57%). The tumors in both strains were morphologically similar, but the lymphoblasts in NIH Swiss mice did not have surface antigens for the murine leukemic virus. These results demonstrate that expression of the retrovirus is not entirely responsible for the incidence of lymphoma. However, the murine leukemic virus may influence the incidence of the lymphoma in B6C3F1 mice.

The carcinogenicity study in rats that was reviewed in the EPA carcinogenicity assessment but was available only as an unpublished report from Hazleton, has been published (Owen et al., 1987). This report (Owen et al., 1987) contains the same information as the Hazleton report that is summarized in the EPA carcinogenicity assessment. In summary, the data suggested treatment related increases in mammary gland, thyroid, and testicular tumors. The authors proposed that the carcinogenic effect was likely an indirect effect mediated through the endocrine system rather than through the production of reactive intermediates.

7.6.3.4   Carcinogenicity - Epidemiological Studies

The results of another epidemiologic study of 1,3-butadiene-exposed workers have been reported since the original EPA assessment (EPA, 1985). This study examined the mortality of 2,586 workers employed for at least 6 months between 1943 and 1979 at a 1,3-butadiene manufacturing facility (Downs et al., 1987). Data regarding exposure levels were not available, but workers were divided according to 4 qualitative exposure categories based on employment records. The categories of exposure were: low exposure, routine exposure (included process workers), nonroutine exposure (intermittent exposure; maintenance workers), and unknown exposures. The overall mortality of the workers was significantly below the U.S. national average (standardized mortality ratio (SMR) = 80). However, the SMR for lympho- and reticulo-sarcoma of the whole cohort was

significantly greater than the U.S. national average (SMR = 235; 95% CI = 101 to 463). Lympho- and reticulosarcoma are malignant diseases of the lymphatic system and the reticuloendothelial system, respectively. The reticuloendothelial system are cells scattered throughout the body that have the power to ingest particulate matter. When calculated by exposure category, the routine exposure group had significant increases in lymphohematopoietic and kidney cancer and the nonroutine exposure group had a significantly increased rate of leukemia when compared to the U.S. national average. These rates of cancer were elevated when compared to the local cohort, but were not statistically significant. Limitations of this study included an unreliable designation of race, lack of worker histories, and the observations that nearly half of the cohort worked at the facility for less than 5 years and that many workers had spent time working at neighboring styrene-butadiene rubber plants.

An update of the Downs et al. (1987) study and updates of two studies originally contained in the EPA 1985 assessment have also been published. In the update of the study by Downs et al. (1987), the workers' mortality experience through 1985 was examined (Divine, 1990). One additional death from lymphosarcoma had occurred since the previous analysis by Downs et al. (1987). Findings were similar to those reported in the 1987 study. Excess mortality due to lymphatic and hematopoietic cancers were seen primarily in those occupational categories with the greatest known exposure to 1,3-butadiene, in those with less than 10 years of employment, and in those employed during World War II.

In the update of the study by Matanoski and Schwartz (1987), followup of workers was improved and extended through 1982 (Matanoski et al., 1990). The cohort in the update was restricted to 12,100 workers by limiting employees from the one Canadian plant to those who had worked 10 years or more or who had reached age 45 during employment. Overall mortality in these workers was less than the U.S. national average (SMR = 81). The only significant increase in mortality observed in the workers was in arteriosclerotic heart disease among black employees compared to the U.S. national average (SMR = 1.48, 95% CI: 1.23-1.76). The workers were subdivided according to the job held the longest. The categories of employment included production, utilities, maintenance, and a combination of all others. Significant increases in mortality were observed among the production workers. Combined race data showed a significant increase in other lymphatic malignancies (SMR = 2.60, 95% CI: 1.19-4.94) and blacks had significant increases in all lymphopoietic cancers (SMR = 5.07, 95% CI: 1.87-11.07) and in leukemia (SMR = 6.56, 95% CI: 1.35-19.06). Whites had elevated mortality due to lymphatic (SMR = 2.30, CI: 0.92-4.73) and hematopoietic (SMR = 1.10, CI: 0.58-1.87) malignancies, but the increase was not statistically significant. This study is somewhat limited in that the race designation of approximately 15% of the workers was unknown but was assumed to be white.

A nested case-control study of the lymphopoietic neoplasms from the cohort of butadiene workers studied by Matanoski and Schwartz (1987) was conducted by Santos-Burgoa (1988). The 59 cases of lymphopoietic neoplasms were matched to controls based on plant, age, hire date, duration of work, and survival to the death date of the case. Exposures to 1,3-butadiene for the cases and controls were estimated through a ranked job exposure matrix which was multiplied by the duration of exposure to yield estimates of cumulative exposure. In a matched analysis based on a categorization of exposure above and below the geometric mean, a statistically significant odds ratio of approximately 2.0 (OR = 2.0) for all lymphopoietic neoplasms and OR = 9.0 for leukemia was calculated for butadiene exposure. Both types of analysis that were performed in this study showed a significant trend for leukemia with cumulative butadiene exposure, but not for all lymphopoietic neoplasms.

An occupational epidemiological pilot study was conducted (Ward et al., 1992) to evaluate the effects of 1,3-butadiene exposure on the frequencies of lymphocytes containing mutations at the hypoxanthine guanine phophoribosyl transferase (hprt) locus in workers in a 1,3-butadiene production plant. Seven workers from areas of the plant where the highest exposures to 1,3-butadiene occur were compared to four workers from plant areas where 1,3-butadiene exposures were low. In addition, four workers from the investigating laboratory were also studied as outside controls. All the subjects were non-smokers. An air sampling survey indicated that average 1,3-butadiene levels in the high exposures area were about 3.5±7.5 ppm while they were 0.03±0.03 in the low exposure area. The low-exposed controls and the outside controls mean variant frequencies, 1.19 and 1.03 respectively, were not significantly different, but the mean frequency of mutant lymphocytes in the seven exposed subjects (4.09) was significantly higher when compared to the means of the eight controls. The observation of an elevated mean in the exposed subjects indicates that exposures occurring in areas where higher concentrations of 1,3-butadiene have been documented were sufficient to induce higher frequencies of somatic cell mutants. Additional studies are being conducted to confirm the effects that have been observed.

7.7 Carcinogenic Risk for Baseline and Control Scenarios

Table 7-11, summarizes the annual cancer incidences for all the scenarios. 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 emissions are 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 vehicles modifications.

From Table 7-11 it can also be observed that the expanded use scenarios provide little additional reduction in the cancer cases.

Table 7-11. Annual Cancer Incidence Projections for 1,3-Butadiene. [a,b]

Year-Scenario	Emission Factor g/mile	Urban Cancer Cases	Rural Cancer Cases	Total Cancer Cases	Percent Reduction from 1990	
					EF	Cancer
1990 Base Control	0.0156	258	46	304	–	–
1995 Base Control	0.0094	177	32	209	40	31
1995 Expanded Reformulated Fuel Use	0.0093	175	32	207	40	32
2000 Base Control	0.0071	149	27	176	54	42
2000 Expanded Reformulated Fuel Use	0.0069	145	26	171	56	44
2000 Expanded Adoption of California Standards	0.0069	146	26	172	56	43
2010 Base Control	0.0067	173	31	204	57	33
2010 Expanded Reformulated Fuel Use	0.0064	164	30	194	59	36
2010 Expanded Adoption of California Standards	0.0062	158	28	186	60	39

[a]Projections have inherent uncertainties in emission estimates, dose-response, and exposure.
[b]Cancer incidence estimates are based on upper bound estimates of unit risk, determined from animal studies. 1,3-Butadiene is classified by EPA as a Group B2, probable human carcinogen based on sufficient evidence in two rodent studies and inadequate epidemiologic evidence.

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.

7.8 Non-Carcinogenic Effects of Inhalation Exposure to 1,3-Butadiene

Since the focus of this report is on the carcinogenic potential of the various compounds, the noncancer information will be dealt with in a more cursory fashion. No attempt has been made to synthesize and analyze the data encompassed below. Also, no attempt has been made to accord more importance to one type of noncancer effect over another. The objective is to research all existing data, describe the noncancer effects observed, and refrain from any subjective analysis of the data.

1,3-Butadiene is used primarily as a monomer in the production of rubber and plastics (Chemical and Engineering News, 1986). It is also found in automobile exhaust (CARB, 1991). Although no human data on the metabolism of 1,3-butadiene exist, animal studies indicate that this chemical is rapidly absorbed following inhalation (Hattis and Wasson, 1987). Inhalation of 1,3-butadiene
is mildly toxic in humans at low concentrations (not otherwise specified) and may result in a feeling of lethargy and drowsiness. At very high concentrations, 1,3-butadiene causes narcosis leading to respiratory paralysis and death. The first signs of toxicity observed in humans are central nervous system symptoms including blurred vision, nausea, paresthesia (a sense of numbness, prickling, or tingling), and dryness of the mouth, throat, and nose, followed by fatigue, headache, vertigo, decreased blood
pressure and pulse rate, and unconsciousness (Sandmeyer, 1981). Retrospective epidemiological studies indicate the possibility of higher than normal mortality rates from cancer and certain cardiovascular diseases, mainly chronic rheumatic and arteriosclerotic heart diseases, among middle-aged rubber workers (McMichael et al., 1974, 1976). Workers exposed to unknown concentrations of 1,3-butadiene during the manufacture of rubber complained of irritation of the eyes, nasal passages, throat, and lungs (Wilson, 1944). An increased rate of emphysema among rubber workers was reported by McMichael et al. (1976). No human studies on the renal, hepatic, or immunological effects of inhaled 1,3-butadiene were located in the available literature.

An LC_{50} of 129,000 ppm in rats after 4 hours of exposure and an LC_{50} of 122,000 ppm in mice after 2 hours of exposure were reported (Shugaev, 1969), indicating that 1,3-butadiene is only mildly acutely toxic. After chronic exposure to 1,250 ppm 1,3-butadiene, mice exhibited respiratory changes such as chronic inflammation of the nasal cavity, fibrosis, cartilaginous

metaplasia, osseous metaplasia, and atrophy of the sensory epithelium (NTP, 1984). No histopathological cardiovascular lesions were found in mice following subchronic exposure (Crouch et al., 1979) or rats (Owen et al., 1987) following chronic exposure to 1,3-butadiene; however, NTP (1984) observed endothelial hyperplasia in the hearts of mice after 61 weeks of exposure. In a chronic study, high incidences of liver necrosis and epithelial hyperplasia in the forestomach of mice were found at 625 ppm (LOAEL) (NTP, 1984), but no nonneoplastic gastrointestinal lesions were found in rats exposed chronically (Owen et al., 1987) or mice exposed subchronically (NTP, 1984). Macrocytic-megaloblastic anemia was observed in mice exposed to 1,250 ppm butadiene for 6-24 weeks (Irons et al., 1986a, 1986b). Bone marrow damage was expressed as reduced numbers of red blood cells, decreased hemoglobin concentration and hematocrit, and increased mean corpuscular volume of circulating erythrocytes. Decreases in red blood cell counts and hemoglobin concentrations were reported in male mice after an intermediate duration exposure of at least 62.5 ppm (Melnick et al., 1989b). However, other studies found no hematological effects in animals following subchronic and chronic exposure to high exposure concentrations of 1,3-butadiene (Carpenter et al., 1944; Crouch et al., 1979; Owen et al., 1987).

1,3-Butadiene appears to be a developmental toxicant. When exposed to concentrations up to 8,000 ppm of 1,3-butadiene during gestation days 6-15, depressed body weight gain among dams was observed at all concentrations, and fetal growth was significantly decreased in the 8,000 ppm group. Major skeletal abnormalities (wavy ribs, irregular rib ossification) were observed in the 1,000 and 8,000 ppm groups (Irvine, 1981). In studies conducted by NTP (Morrissey et al., 1990), pregnant Sprague Dawley rats exposed to 1,000 ppm 1,3-butadiene by inhalation on gestation days 6-15 exhibited depressed body weight gain, but there was no evidence of developmental toxicity in their offspring. In contrast, male and female fetuses of mice similarly exposed exhibited reduced weight at levels of 40 ppm and higher, and 200 ppm and higher, respectively.

Melnick et al. (1990) reported that testicular atrophy was observed in male B6C3F1 mice exposed to 625 ppm 1,3-butadiene for 65 weeks, and ovarian atrophy was observed in female B6C3F1 mice exposed to ≥20 ppm for 65 weeks. A concentration-related increase in the incidence of sperm-head abnormalities occurred in mice after exposure to 1,000 and 5,000 ppm of 1,3-butadiene for 6 hours/day for 5 days (Hackett et al., 1988a). Dominant lethality (i.e., a gene mutation that must only occur in one copy of the gene to result in death of the offspring) in mice was also observed during the first 2 postexposure weeks after the males were exposed to 200, 1,000 or 5,000 ppm (Hackett et al., 1988b), suggesting that more mature cells (spermatozoa and spermatids) may be altered by 1,3-butadiene exposure.

CARB used the two-year inhalation studies with mice (Huff et al., 1985; Melnick et al., 1988, 1989a, 1989b; Miller, 1989) exposed to 0, 6.25, 20, 62.5, 200, and 625 ppm 1,3-butadiene to establish a LOAEL. These studies were designed as cancer bioassays. Gonadal atrophy was observed at a high incidence in exposed animals of both sexes at levels of 200 ppm and above, but not in any of the control animals. In the later study, using the entire dose range, levels of 6.25 ppm and higher also produced gonadal atrophy in females. Thus, a NOAEL was not established in these studies, but a LOAEL of 6.25 ppm was observed. In contrast, the Hazelton rat bioassay (Owen et al., 1987) did not report any reproductive effects even at 8000 ppm level.

Neither an inhalation reference concentration (RfC) nor an oral reference dose (RfD) is available for 1,3-butadiene at this time.

7.9 References for Chapter 7

Andjelkovich, D., J. Taulbee, and M. Symons. 1976. Mortality experience of a cohort of rubber workers, 1964-1973. J. Occup. Med. 18:387-394.

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.

Barnes, I., V. Bastian, K. H. Becker, and Z. Tong. 1990. Kinetics and products of the reactions of NO_3 with monoalkenes, dialkenes, and monoterpenes. J. Phys. Chem., 94:2413-2419.

Bond, J.A., A.R. Dahl, R.F. Henderson, G.S. Dutcher, J.L. Mauderly, and L.S. Birnbaum. 1986. Species differences in the distribution of inhaled butadiene. Toxicol. Appl. Pharmacol. 84:617-627.

Bond, J.A., A.R. Dahl, R.F. Henderson, and L.S. Birnbaum. 1987. Species differences in the distribution of inhaled butadiene in tissues. Am. Ind. Hyg. Assoc. J. 48:857-862.

Bond, J.A., O.S. Martin, L.S. Birnbaum, A.R. Dahl, R.L. Melnick, and R.F. Henderson. 1988. Metabolism of 1,3-butadiene by lung and liver microsomes of rats and mice repeatedly exposed by inhalation to 1,3-butadiene. Toxicol. Lett. 44:143-151.

Bryant, M.S. and S.M. Osterman-Golkar. 1991. Hemoglobin adducts as dosimeters of exposure to DNA-reactive chemicals. CIIT Activities, 11(10).

CARB. 1991. Butadiene Emission Factors. memo from K. D. Drachand to Terry McGuire and Peter Venturini, July 17, 1991.

CARB. 1992a. Proposed identification of 1,3-butadiene as a toxic air contaminant. Part A Exposure assessment. California Air Resources Board, Stationary Source Division. May 1992.

CARB. 1992b. Proposed identification of 1,3-butadiene as a toxic air contaminant. Part B Health assessment. California Air Resources Board, Stationary Source Division. May 1992.

Carpenter, C.P., C.B. Shaffer, C.S. Weil, and H.F. Smyth, Jr. 1944. Studies on the inhalation of 1,3-butadiene; with a comparison of its narcotic effect with benzol, toluol, and styrene, and a note on the elimination of styrene by the human. J. Ind. Hyg. Toxicol. 26:69-78.

Chan, C.C., H. Ozkaynak, J.D. Spengler, L. Sheldon, W. Nelson, and L. Wallace. 1989. Commuter's exposure to volatile organic compounds, ozone, carbon monoxide, and nitrogen dioxide. Prepared for the Air and Waste Management Association. AWMA Paper 89-34A.4.

Checkoway, H. and T.M. Williams. 1982. A hematology survey of workers at a styrene-butadiene synthetic rubber manufacturing plant. Am. Ind. Hyg. Assoc. J. 43:164-169.

Chemical and Engineering News. 1986. Key Chemicals Profile Butadiene. June 9, 15.

Choy, W.N., D.A. Vlachos, M.J. Cunningham, G.T. Arce, and A.M. Sarrif. 1986. Genotoxicity of 1,3-butadiene. Induction of bone marrow micronuclei in B6C3F1 mice and Sprague-Dawley rats *in vivo*. Environ. Mutagen. 8:18.

Clement International Corporation. 1991. Motor vehicle air toxics health information. For U.S. EPA Office of Mobile Sources, Ann Arbor, MI: September 1991.

Conner, M., J. Lou, and O. Gutierrez de Gotera. 1983. Induction and rapid repair of sister-chromatid exchanges in multiple murine tissues in vitro by diepoxybutane. Mutat. Res. 108: 251-263.

Cote, I.L. and S.P. Bayard. 1990. Cancer risk assessment of 1,3-butadiene. Environ. Health Perspect. 86:149-153.

Crouch, C.N., D.H. Pullinger, and I.F. Gaunt. 1979. Inhalation toxicity studies with 1,3-butadiene: 2. 3 month toxicity study in rats. Am. Ind. Hyg. Assoc. J. 40:796-802.

Csanády, G.A. and J.A. Bond. 1991a. Species differences in the biotransformation of 1,3-butadiene to DNA-reactive epoxides: role in cancer risk assessment. CIIT Activities 11(2): 1-8.

Csanády, G.A. and J.A. Bond. 1991b. Species and organ differences in the metabolic activation of 1,3-butadiene. Toxicologist 11,47.

Cunningham, M.J., W.N. Choy, G.T. Arce, L.B. Rickard, D.A. Vlachos, L.A. Kinney, and A.M. Sarrif. 1986. *In vivo* sister chromatid exchange and micronucleus induction studies with 1,3-butadiene in B6C3F1 mice and Sprague-Dawley rats. Mutagenesis 1:449-452.

Cupitt, L. T. 1987. "Atmospheric persistence of eight air toxics." U.S. Environmental Protection Agency (EPA-600/3-87/004).

Dankovic, D.A., R.J. Smith, J. Seltzer, A.J. Bailer, and L.T. Stayner. 1991. A Quantitative Assessment of the Risk of Cancer Associated with Exposure to 1,3-Butadiene, Based on a Low Dose Inhlation Study in B6C3F Mice. Washington, DC: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health. September 27, 1991, Docket #H-041.

Dean, B.J., and G. Hodson-Walker. 1979. An in vitro chromosome assay using cultured rat-liver cells. Mutat. Res. 64: 329-337.

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.

de Meester, C., F. Poncelet, F. Roberfroid and M. Mercier. 1978. Mutagenicity of butadiene and butadiene monoxide. Biochem. Biophys. Res. Commun. 80: 298-305.

de Meester, C., F. Poncelet, F. Roberfroid and M. Mercier. 1980. The mutagenicity of butadiene towards *Salmonella typhimurium*. Toxicol. Lett. 6: 125-130.

Deutschman, S., R.J. Laib. 1989. Concentration-dependent depletion of non-protein sulfhydryl (NPSH) content in lung, heart and liver tissue of rats and mice after acute inhalation exposure to butadiene. Toxicol. Lett. 45:175-183.

Divine, B.J. 1990. An update on mortality among workers at a butadiene facility - preliminary results. Environ. Health Perspect. 86:119-128.

Downs, T.D., M.M. Crane, K.W. Kim. 1987. Mortality among workers at a butadiene facility. Am. J. Ind. Med. 12:311-330.

Ehrenberg, L., and S. Hussain. 1981. Genotoxicity of some important epoxides. Mutat. Res. 86: 1-113.

Environ Corporation. 1987. Risk assessment issues in EPA's technical report "Air Toxics Emissions from Motor Vehicles". Prepared for the Motor Vehicle Manufacturer Association.

EPA. 1984. Proposed guidelines for carcinogen risk assessment. Federal Register 49(227): 46294-46301.

EPA. 1985. Mutagenicity and carcinogenicity assessment of 1,3-butadiene. U.S. Environmental Protection Agency: Washington, DC: Office of Health and Environmental Assessment. Publication No. EPA/600/8-85/004F.

EPA. 1989. Locating and estimating air emissions from sources of 1,3-butadiene. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Report no. EPA-450/2-89-021.

EPA. 1990. 1989 Urban Air Toxics Monitoring Program. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Report no. EPA-450/4-91-001.

EPA. 1991. Nonroad Engine and Vehicle Emission Study. Office of Air and Radiation, Office of Mobile Sources, Ann Arbor, MI. November 1991. EPA Report No. 21A-2001.

EPA. 1992. Integrated Risk Information System. U.S. Environmental Protection Agency. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

Gervasi, P.G., L. Citti, M. Del Monte, V. Longo, and D. Benetti. 1985. Mutagenicity and chemical reactivity of epoxidic intermediates of isoprene metabolism and other structurally related compounds. Mutat. Res. 156:77-82.

Grossman, E.A. and J. Martonik. 1990. OSHA's approach to risk assessment for setting a revised occupational exposure standard for 1,3-butadiene. Environ. Health Perspect. 86:155-158.

Hackett, P.L., B.J. McClanahan, M.G. Brown, R.L. Buschbom, and M.L. Clark. 1988a. Sperm-head morphology study in B6C3F1 mice following inhalation exposure to 1,3-butadiene: final technical report. Report to National Institute of Environmental Health Sciences, National Toxicology Program by Pacific Northwest Laboratory, Richland, WA. PNL-6459; DE88008620.

Hackett, P.L., T.J. Mast, M.G. Brown, M.L. Clark, and J.J., Evanoff. 1988b. Dominant lethal study in CD-1 mice following inhalation exposure to 1,3-butadiene: final technical report. ISS PNL-6545; DE88010185.

Hattis. D. and J. Wasson. 1987. Pharmacokinetic/mechanism-based analysis of the carcinogenic risk of butadiene. Report to National Institute for Occupational Safety and Health, Rockville, MD, by Massachusetts Institute of Technology, Cambridge, MA. NTIS PB88-202817.

Hazleton Laboratories Europe, Ltd. (HLE). 1981. The toxicity and carcinogenicity of butadiene gas administered to rats by inhalation for approximately 24 months. Prepared for the International Institute of Synthetic Rubber Producers, New York, NY. Unpublished.

Hemminki, K., E. Franssila, and H. Vianio. 1980. Spontaneous

abortions among female chemical workers in Finland. Int. Arch. Occup. Environ. Health 45:123-126.

Huff, J.E., R.L. Melnick, H.A. Solleveld, J.K. Haseman, M. Powers, and R.A. Miller. 1985. Multiple organ carcinogenicity of 1,3-butadiene and B6C3F1 mice after 60 weeks of inhalation exposure. Science 227: 548-549.

IARC. 1986. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Volume 39. 1,3-Butadiene. International Agency for Research on Cancer. World Health Organization, Lyon, France. p. 156-179.

IARC. 1987. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Supplement 7. Overall Evaluations of Carcinogenicity: An updating of IARC monographs volumes 1 to 42. International Agency for Research on Cancer. World Health Organization, Lyon, France. p. 136-137.

IARC. 1992. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Volume 54. Occupational Exposures to Mists and Vapours from Strong Inorganic Acids; and Other Industrial Chemicals. International Agency for Research on Cancer. World Health Organization, Lyon, France. p. 237-286.

ICF/Clement. 1986. Rulemaking support for 1,3-butadiene. Draft Final Report: Characterization of risks associated with occupational exposure to 1,3-butadiene. Prepared for the Office of Health Standards, Occupational Safety and Health Administration, U.S. Department of Labor. March 10, 1986.

Irons, R.D., C.N. Smith, W.S. Stillman, R.S. Shah, W.J. Steinhagen, and L.J. Leiderman. 1986a. Macrocytic-megaloblastic anemia in male B6C3F1 mice following chronic exposure to 1,3-butadiene. Toxicol. Appl. Pharmacol. 83:95-100.

Irons, R.D., C.N. Smith, W.S. Stillman, Shah, W.J. Steinhagen, and L.J. Leiderman. 1986b. Macrocytic-megaloblastic anemia in male NIH Swiss mice following chronic exposure to 1,3-butadiene. Toxicol. Appl. Pharmacol. 85:450-455.

Irons, R.D., W.S. Stillman, R.S. Shah, M.S. Morris, and M. Higuchi. 1986c. Phenotypic characterization of 1,3-butadiene-induced thymic lymphoma in male B6C3F1 mice. The Toxicologist 6:27.

Irons, R.D., M. Oshimura, and J.C. Barrett. 1987a. Chromosome aberrations in mouse bone marrow cells following *in vivo* exposure to 1,3-butadiene. Carcinogenesis 8:1711-1714.

Irons, R.D., W.S. Stillman, and M.W. Cloyd. 1987b. Selective

activation of endogenous esotropic retrovirus in hematopoietic tissues of B6C3F1 mice during the preleukemic phase of 1,3-butadiene exposures. Virology 161:457-462.

Irons, R.D., H.P. Cathro, W.S. Stillman, W.H. Steinhagen, and R.S. Shah. 1989. Susceptibility to 1,3-butadiene-induced leukemogenesis correlates with endogenous esotropic retroviral background in the mouse. Toxicol. Appl. Pharmacol. 101:170-176.

Irvine, L.F.H. 1981. 1,3-Butadiene: Inhalation teratogenicity study in the rat: final report. Harrogate, England: Hazleton Laboratories Europe Ltd. OTS Submission, Microfiche No. 050 545.

Jauhar, P.P., P.R. Henika, J.T. MacGregor, C.M. Wehr, M.D. Shelby, S.A. Murphy, and B.H. Margolin. 1988. 1,3-Butadiene: induction of micronucleated erythrocytes in the peripheral blood of B6C3F1 mice exposed by inhalation for 13 weeks. Mutat. Res. 209:171-176.

Jelitto, B., R.R. Vangala, and R.J. Laib. 1989. Species-differences in DNA damage by butadiene: role of diepoxybutane. Arch. Toxicol. 13: 246-249.

Kreiling, R., R.J. Laib, and H.M. Bolt. 1986a. Alkylation of nuclear proteins and DNA after exposure of rats and mice to [1,4-14C]1,3-butadiene. Toxicol. Lett. 30:131-136.

Kreiling, R., R.J. Laib, J.G. Filser, and H.M. Bolt. 1986b. Species differences in butadiene metabolism between mice and rats evaluated by inhalation pharmacokinetics. Arch. Toxicol. 58:235-238.

Kreiling, R., R.J. Laib, J.G. Filser, and H.M. Bolt. 1987. Inhalation pharmacokinetics of 1,2-epoxybutene-3 reveal species differences between rats and mice sensitive to butadiene-induced carcinogenesis. Arch. Toxicol. 61:7-11.

Kreiling, R., R.J. Laib, and H.M. Bolt. 1988. Depletion of hepatic nonprotein sulfhydryl content during exposure of rats and mice to butadiene. Toxicol. Lett. 41:209-214.

Laib, R.J. and R. Kreiling. 1987. Evaluation of the reactive principles responsible for genotoxicity as a prerequisite for carcinogenic exposure monitoring of the halogenated ethylenes and butadiene. Clin. Toxicol. 23:1172.

Lawley, P.D. and P. Brookes. 1967. Interstrand cross-linking of DNA by difunctional alkylating agents. J. Mol. Biol. 25: 143-160.

Lemen, R.A., T.J. Meinhardt, M.S. Crandall, J.M. Fagan, and D.P. Brown. 1990. Environmental epidemiologic investigations in the styrene-butadiene rubber production industry. Environ. Health Perspect. 86:103-106.

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., R.R. Schulhof, R.E. Jackson, M.M. Jimenez, G. Z. Whitten, G.M. Wilson, T.C. Meyers, and J.L. Fieber. 1992. Modeling the effects of reformulated gasolines on ozone and toxics concenetrations in the Baltimore and Houston areas. Systems Applications International, San Rafael, California (SYSAPP-92/127).

Luker, M.A. and B.J. Kilbey. 1982. A simplified method for the simultaneous detection of intragenic and intergenic mutations (deletions) in *Neurospora crassa*. Mutat. Res. 92: 63-68.

Matanoski, G.M., L. Schwartz, J. Sperrazza, and J. Tonascia. 1982. Mortality of workers in the styrene-butadiene rubber polymer manufacturing industry. Johns Hopkins University School of Hygiene and Public Health, Baltimore, MD. Unpublished.

Matanowski, G.M. and J. Schwartz. 1987. Mortality of worker in styrene-butadiene polymer production. J. Occup. Med. 29:675-680.

Matanoski, G.M., C. Santos-Burgoa, S.L. Zeger, and L. Schwartz. 1990. Mortality of a cohort of workers in the styrene-butadiene polymer manufacturing industry (1943-1982). Environ. Health Perspect. 86:107-117.

McMichael, A.J., R. Spirtas, L.L. Kupper. 1974. An epidemiologic study of mortality within a cohort of rubber workers, 1964-1972. J. Occup. Med. 16:458-464.

McMichael, A.J., R. Spirtas, J.F. Gamble, and P.M. Tousey. 1976. Mortality among rubber workers. Relationship to specific jobs. J. Occup. Med. 18:178-185.

Meinhardt, T.J., R.A. Lemen, M.S. Crandall, and R.J. Young. 1982. Environmental epidemiologic investigation of the styrene-butadiene rubber industry. Scand. J. Work Environ. Health 8:250-259.

Melnick, R.L., J.E. Huff, J.K. Haseman, and E.E. McConnell. 1988. Chronic toxicity results and ongoing studies of 1,3-butadiene by the National Toxicology Program. Ann. New York Acad. Sci. 534:649-662.

Melnick, R.L., J. Huff, and R. Miller. 1989a. Toxicology and carcinogenicity of 1,3-butadiene. In: Mohn U, ed. Assessment of inhalation hazards: Integration and extrapolation using diverse data. New York, NY: Springer-Verlag, 177-188.

Melnick, R.L., J.H. Roycroft, B. Chou, and R. Miller. 1989b. Carcinogenicity of 1,3-butadiene in mice at low inhalation exposure concentrations. Proc. Am. Ass. Cancer Res. 143.

Melnick, R.L., J.E. Juff, J.H. Roycroft, B.J. Chou, and R.A. Miller. 1990. Inhalation toxicology and carcinogenicity of 1,3-butadiene in B6C3F1 mice following 65 weeks of exposure. Environ. Health Perspect. 86:27-36.

Miller, R.A. 1989. Neoplastic lesions induced by 1,3-butadiene in B6C3F1 mice. Assessment of inhalation hazards: Integration and extrapolation of diverse data. Conference at Hannover Medical School, February 16-24, 1989. Hannover, Federal Republic of Germany.

Morrissey, R.E., B.A. Schwetz, P.L. Hacket, M.R. Sikov, B.D. Hardin, B.J. McClanahan, J.R. Decker, and T.J. Mast. 1990. Overview of reproductive and developmental toxicity studies of 1,3-butadiene in rodents. Environ. Health Perspect. 86:79-84.

Murray, M.P. and J.E. Cummins. 1979. Mutagenic activity of epoxy embedding reagents employed in electron microscopy. Environ. Mutagen. 1: 307-313.

NTP. 1984. NTP technical report on the toxicology and carcinogenesis studies of 1,3-butadiene in B6C3F1 mice. National Toxicology Program. Research Triangle Park, NC. NTP TR 288. NTP-83-071. NIH Pub. No. 84-2544.

NTP. 1985. Draft report on the toxicology and carcinogenesis studies of 4-vinylcyclohexane in F433/N rats and B6C3F1 mice. NIH Publication NO. 85-2559.

Olszewska, E. and B.J. Kilbey. 1975. The mutagenic activity of diepoxybutane in yeast. Mutat. Res. 33: 383-390.

Owen, P.E., J.R. Glaister, I.F. Gaunt, and D.H. Pullinger. 1987. Inhalation toxicity studies with 1,3-butadiene. 3. 2-year toxicity/carcinogenicity study in rats. Am. Ind. Hyg. Assoc. J. 48:407-413.

Perry, P. and H.J. Evans. 1975. Cytological detection of mutagen-carcinogen exposure by sister chromatid exchange. Nature. 258: 121-125.

Platt, U., D. Perner, A. M. Winer, G. W. Harris, and J. N. Pitts,

Jr. 1980. Detection of NO$_3$ in the polluted troposphere by differential optical absorption. Geophys. Res. Lett., 7:89-92.

Poncelet, F. C. de meester, M. Duverger-van Bogaert, M. Lambotte-Vandepaer, M. Roberfroid, and M. Mercier. 1980. Influence of experimental factors on the mutagenicity of vinylic monomers. Arch. Toxicol. Suppl. 4: 63-66.

Recio, L., O. Moss, J. Bond, and G.A. Csanády. 1991. Biotransfromation of butadiene by hepatic and pulmonary tissues from mice, rats, and humans: relationship to butadiene carcinogenicity and *in vivo* mutagenicity. Proceedings from the Air and Waste Management Association's specialty conference "Air Toxics Pollutants from Mobile Sources: Emissions and Health Effects." October 16-18, 1991.

Sandmeyer, E.E. 1981. Aliphalic hydrocarbons: Butadiene. In: Clayton GD, Clayton FE, eds. Patty's industrial hygiene and toxicology, 3rd revised ed. New York, NY: John Wiley and Sons.

Sankaranarayanan, K. 1983. The effects of butylated hydroxytoluene on radiation and chemically-induced genetic damage in *Drosophila melanogaster*. Mutat. Res. 108: 203-223.

Sankaranarayanan, K., W. Ferro, and J.A. Ziljlstra. 1983. Studies on mutagen-sensitive strains on *Drosophila melanogaster*. III. A comparison of the mutagenic sensitivities of the *ebony* (UV and X-ray sensitive) and *Canton-S* (wild-type) strains to MMS, ENU, DEB, and 2,3,6-Cl$_3$-PDMT. Mutat. Res. 100: 59-70.

Santos-Burgoa, C. 1988. Dissertation submitted to the Johns Hopkins University, Baltimore, Maryland, August 1988.

Schmidt, U. and E. Loeser. 1985. Species differences in the formation of butadiene monoxide from 1,3-butadiene. Arch. Toxicol. 57:222-225.

Schmidt, U. and E. Loeser. 1986. Epoxidation of 1,3-butadiene in liver and lung tissue of mouse, rat, monkey and man. Adv. Exp. Med. Biol. 197:951-957.

Sharief, Y., A.M. Brown, L.C. Backer, J.A. Campbell, B. Westbrook-Collins, A.G. Stead, and J.W. Allen. 1986. Sister chromatid exchange and chromosome aberration analyses in mice after in vivo exposure to acrylonitrile, styrene, or butadiene monoxide. Environ. Mutagen. 8:439-448.

Shugaev, B.B. 1969. Concentrations of hydrocarbons in tissues as a measure of toxicity. Arch. Environ. Health 18:878-882.

Simmon, V.F. and J.M. Baden. 1980. Mutagenic activity of vinyl compounds and derived epoxides. Mutat. Res. 78: 227-231.

Tice, R.R., R. Boucher, C.A. Luke, and M.D. Shelby. 1987. Comparative cytogenetic analysis of bone marrow damage induced in male B6C3F1 mice by multiple exposure to gaseous 1,3-butadiene. Environ. Mutagen. 9: 235-250.

Truchi, G., S. Bonatti, L. Citti, P.G. Gervasi, and A. Abbondandolo. 1981. Alkylating properties and genetic activity of 4-vinylcyclohexane metabolites and structurally related epoxides. Mutat. Res. 83: 419-430.

Turnbull, D., J.V. Rodricks, S.M. Brett. 1990. Assessment of the potential risk to workers from exposure to 1,3-butadiene. Environ. Health. Perspect. 86: 159-171.

Voogd, C.E., J.J. van de Stel, and J.A. Jacobs. 1981. The mutagenic action of aliphatic epoxides. Mutat. Res. 89: 269-282.

Wade, M.J., J.W. Moyer, and C.H. Hine. 1979. Mutagenic action of the series of epoxides. Mutat. Res. 66: 367-371.

Ward, J.B. Jr., M.M. Ammenheuser, E.B. Whorton, Jr., and M.S. Legator. 1992. *hprt* Mutant Lymphocyte Frequencies in Workers at a Butadiene Production Plant. Abstract presented at the Fourth European International Society for the Study of Xenobiotics (ISSX), Bologna, Italy, July 3-6, 1992.

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.

Wilson, R.H. 1944. Health hazards encountered in the manufacture of synthetic rubber. J. Am. Med. Assoc. 124:701-703.

Zaborowski, D., Z. Swietlinska, and J. Zuk. 1983. Induction of mitotic recombination by UV and diepoxybutane and its enhancement by hydroxyurea in *Saccharomyces cerevisiae*. Mutat. Res. 120: 21-26.

Zhou, X.T., L.R. Li, M.Y. Cui, et al. 1986. Cytogenetic monitoring of petrochemical workers [Abstract]. Environ. Mutagen. 8:96.

Zimmering, S. 1983. The *mei-g*[a] test for chromosome loss in *Drosophila*: a review of assays of 21 chemicals for chromosome breakage. Environ. Mutagen. 5: 907-921.

8.0 ACETALDEHYDE

8.1 Chemical and Physical Properties

The information below is excerpted from the EPA health assessment draft document (EPA, 1987) and Perry and Chilton, 1973.

Acetaldehyde is a saturated aldehyde with a pungent and suffocating odor, but at more dilute concentrations the odor is fruity and pleasant. It has the chemical formula CH_3CHO. It is a colorless liquid, volatile at room temperature, and 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, alcohol, ether, acetone, and benzene. The chemical and physical properties are listed in Table 8-1.

As the vapor pressure of acetaldehyde is very high and it is soluble in water, the most important environmental behavior will be in air and water. This is due to vaporization from the soil (and other sources) into the air and leaching from soil into the water. Acetaldehyde may remain bound in the soil because of its high reactivity, but it is also readily metabolized by soil microorganisms.

Acetaldehyde is a component of photochemical smog, and as such its movement within the atmosphere corresponds to that of the smog front. The high solubility of acetaldehyde in water increases the likelihood of its being leached into the soil.

In the atmosphere, acetaldehyde would be degraded through photooxidation and oxidation by the hydroxyl radical. The main product of photooxidation in the presence of NO_x is peroxyacetyl nitrate.

Table 8-1. Chemical and Physical Properties of Acetaldehyde.

Properties	Value
Molecular weight	44.06 g/mole
Melting point	-123.5°C (-190.3°F)
Boiling point	20.16°C (68.3°F)
Density at 18°C (64.4°F)	0.783 g/ml
Vapor pressure at 20°C (68°F)	0.97 atm.
Flash point (closed cup)	-38.0°C (-36.4°F)
Solubility in water at 25°C	infinite
Conversion at 25°C (77°F) and 760 mm Hg	1 ppm = 1.8 mg/m^3

8.2 Formation and Control Technology

Acetaldehyde is another aldehyde which is found in 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.

8.3 Emissions

8.3.1 Emission Fractions Used in the MOBTOX Emissions Model

Like 1,3-butadiene and formaldehyde, emission fractions for acetaldehyde were developed using vehicle emissions data (Appendix B2). Acetaldehyde emission fractions for different components included in the scenarios are included in Appendix B6. Emission fractions for the various vehicle class/catalyst technology groups were based on the same number of cars and studies as the formaldehyde emission fractions.

To calculate TOG fractions for vehicles running on MTBE blends and 10% ethanol, adjustment factors were applied to the baseline emission fractions for each vehicle class/catalyst combination, in the same manner as was done for 1,3-butadiene and formaldehyde. The average percent change numbers for vehicle class/catalyst combinations by study are contained in Appendix B4. The 15% MTBE and 10% ethanol adjustment factors for LDGVs/LDGTs with various catalyst technologies are summarized in Table 8-2. Note that use of oxygenated fuels increases acetaldehyde emissions for all catalyst technologies, and that acetaldehyde increases more than 200% for all catalyst technologies with 10% ethanol use.

These 15% MTBE and 10% ethanol numbers were estimated using data from the same studies as formaldehyde. Once again, since the average percent change was calculated for 15% MTBE (2.7% weight percent oxygen), and 11.0% MTBE (2.0% oxygen) was assumed for reformulated fuel and California standards components, average percent changes in the formaldehyde TOG fraction from 0 to 15% MTBE were multiplied by 2.0/2.7. For HDGVs with three-way catalysts and with no catalysts, we assumed the same 15% MTBE and 10% ethanol adjustment factors as for LDGVs/LDGTs with the same catalyst technologies.

Table 8-2. 15% MTBE and 10% Ethanol Emission Fraction Adjustment Factors for Acetaldehyde.

Vehicle Class	Catalyst Technology	15% MTBE Adjustment Factor	10% EtOH Adjustment Factor
LDGV/LDGT	3-way	1.0826	2.1369
LDGV/LDGT	3-way + ox	1.0136	2.2453
LDGV/LDGT	oxidation	1.2114	2.9609
LDGV/LDGT	non-cat	1.4377	2.1445

8.3.2 Emission Factors for Baseline and Control Scenarios

The fleet average acetaldehyde emission factors as determined by the MOBTOX emissions model are presented in Table 8-3. When comparing the base control scenarios relative to 1990, the emission factor is reduced by 40% in 1995, by 57% in 2000, and by 62% in 2010. The expansion of reformulated fuel use in 1995 actually has no net impact on the emission factor. In 2000, the expansion of reformulated fuel usage also has no net impact on the emission factor, whereas the expanded California standard scenario increases the emission factor by 1%, relative to 1990. In 2010, there is a decrease from the 2010 base control for the reformulated fuels scenario of 1% and the California standards scenario of 4%.

8.3.3 Nationwide Motor Vehicle Acetaldehyde Emissions

The nationwide acetaldehyde metric tons are presented in Table 8-4. Total metric tons are determined by multiplying the emission factor (g/mile) by the VMT determined for the particular year. The VMT, in billion miles, was determined to be 1793.07 for 1990, 2029.74 for 1995, 2269.25 for 2000, and 2771.30 for 2010. When comparing the base control scenarios relative to 1990, the metric tons are reduced by 32% in 1995, by 46% in 2000, and by 42% in 2010, which is actually an increase when compared to 2000.

8.3.4 Other Sources of Acetaldehyde

The motor vehicle contribution to ambient acetaldehyde levels contains both direct (primary) and secondary acetaldehyde formed from photooxidation of VOC, though the rate of photooxidation is much less than that of formaldehyde. It appears that roughly 39% of acetaldehyde emissions may be attributable to motor vehicles. Section 8.5.2 contains a complete explanation of how this number is determined.

Table 8-3. Annual Emission Factor Projections for Acetaldehyde.

Year-Scenario	Emission Factor g/mile	Percent Reduction from 1990
1990 Base Control	0.0119	–
1995 Base Control	0.0071	40
1995 Expanded Reformulated Fuel Use	0.0071	40
2000 Base Control	0.0051	57
2000 Expanded Reformulated Fuel Use	0.0051	57
2000 Expanded Adoption of California Standards	0.0052	56
2010 Base Control	0.0045	62
2010 Expanded Reformulated Fuel Use	0.0044	63
2010 Expanded Adoption of California Standards	0.0041	66

Table 8-4. Nationwide Metric Tons Projection for Acetaldehyde.

Year-Scenario	Emission Factor g/mile	Metric Tons
1990 Base Control	0.0119	21,338
1995 Base Control	0.0071	14,411
1995 Expanded Reformulated Fuel Use	0.0071	14,411
2000 Base Control	0.0051	11,573
2000 Expanded Reformulated Fuel Use	0.0051	11,573
2000 Expanded Adoption of California Standards	0.0052	11,800
2010 Base Control	0.0045	12,471
2010 Expanded Reformulated Fuel Use	0.0044	12,194
2010 Expanded Adoption of California Standards	0.0041	11,362

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 acetaldehyde emissions (Eimitus et al., 1978). Acetaldehyde is also released through the burning of tobacco (Braven et al., 1967), the combustion of organic fuels, coal refining, and coal waste processing (Versar Inc., 1975), and also as a product of plastics combustion (Boettner et al., 1973).

Manufacturing plants that produce acetaldehyde also emit acetaldehyde, as do manufacturing plants that produce ethanol, phenol, acrylonitrile, and acetone (Eimitus et al., 1978; Mannsville Chemical Products Corp., 1984; Delaney and Hughs, 1979). Chemical processes that involve acetaldehyde as an intermediate also emit acetaldehyde. This includes the production of peracetic acid, pentaerythritol, pyridine, terephthalic acid, 1,3-butylene glycol, and crotonaldehyde.

8.4 Atmospheric Reactivity and Residence Times

The processes involved in transformation and residence times were previously discussed in Section 5.4. For a more detailed explanation of the various parameters involved in these processes please refer to Section 5.4. The information that follows on transformation and residence times has been mainly excerpted from a report produced by Systems Applications International for the EPA (Ligocki and Whitten, 1991).

8.4.1 Gas Phase Chemistry of Acetaldehyde

The atmospheric transformation chemistry of acetaldehyde (CH_3CHO) is similar in many respects to that of formaldehyde. Like formaldehyde, it can be both produced and destroyed by atmospheric chemical transformation. The reaction rate of acetaldehyde with OH is in fact about the same as formaldehyde. However, there are important differences between the two. Acetaldehyde photolyses, but much more slowly than formaldehyde. Whereas formaldehyde produces CO upon reaction or photolysis, acetaldehyde produces organic radicals that ultimately form peroxyacetyl nitrate (PAN) and formaldehyde.

8.4.1.1 Formation

Acetaldehyde is formed from the atmospheric oxidation of many types of organic compounds. Unlike formaldehyde, acetaldehyde is

not produced in the atmospheric oxidations of methane and isoprene, but may be produced in the atmospheric oxidation of other naturally occurring organic compounds such as terpenes. In urban areas, the oxidation of olefins such as propene (C_3H_6), and paraffins such as propane (C_3H_8) and ethanol (C_2H_5OH) produces acetaldehyde.

Paraffins (also termed alkanes and saturated hydrocarbons) are organic compounds containing only single-bonded carbon. Paraffins are generally present in urban atmospheres in high concentrations, but react relatively slowly. The pathways by which paraffins are converted to aldehydes such as formaldehyde and acetaldehyde have been summarized by the National Research Council (NRC, 1981). Briefly, the process is initiated by the reaction of a paraffin (such as propane) with OH. This reaction proceeds forming an organic radical that rapidly reacts with atmospheric O_2 to form an organic peroxy radical (often represented as RO_2). In urban atmospheres, these RO_2 radicals typically react with NO, forming NO_2 and fueling the photochemical ozone production cycle. The organic intermediate formed in these reactions rapidly produces aldehydes. The specific aldehydes formed in a given reaction depend upon the initial chain length of the paraffin and the position along the chain at which the initial OH attack occurred. It can easily be seen that a whole family of aldehydes could be produced in varying yields in the oxidation of a single compound.

Olefins (also termed alkenes and unsaturated hydrocarbons) are species containing one or more double bonds. Both OH and O_3 react rapidly with olefins by addition to these reactive double bonds, again forming radical intermediates that decay through a variety of pathways to form aldehydes. In these cases, the particular aldehyde produced will depend upon the location of the double bond.

8.4.1.2 Gas Phase Reactions

Acetaldehyde reacts more rapidly than formaldehyde with the OH and NO_3 radicals. Acetaldehyde reactions with the HO_2, oxygen atoms, O_3, and Cl radicals are not important to the atmospheric chemistry of acetaldehyde due to low concentrations in the atmosphere and/or low to negligible reaction rates.

Acetaldehyde absorbs ultraviolet (UV) radiation from wavelengths below 290 nanometers (nm) to about 345 nm. Although there are three possible pathways for acetaldehyde photolysis, only one is important at wavelengths >290 nm.

The resulting photolysis rate is less than 10 percent of the formaldehyde photolysis rate. Therefore, photolysis is a relatively minor atmospheric transformation pathway for acetaldehyde.

8.4.1.3 Reaction Products

The oxidation of acetaldehyde by OH, oxygen atoms, and NO_3 radicals form a CH_3CO radical that rapidly reacts with atmospheric O_2 to form the peroxyacetyl radical, $CH_3C(O)OO$. This radical can then react with atmospheric NO and NO_2. The reaction with NO_2

produces peroxyacetyl nitrate (PAN), whereas the reaction with NO ultimately produces formaldehyde. Minor products of the peroxyacetyl radical reactions are peroxyacetic acid and acetic acid. Although acetaldehyde is a PAN precursor, methylglyoxal and other species derived from the oxidation of aromatic compounds are more important PAN precursors in urban atmospheres than acetaldehyde. The photolysis of acetaldehyde produces the CH_3O_2 radical, which reacts with NO to form formaldehyde. Thus, the major acetaldehyde decomposition products are formaldehyde and PAN, both of which are of concern as toxic and/or irritant species. However, in neither case is acetaldehyde a dominant source of these species.

8.4.2 Aqueous Phase Chemistry of Acetaldehyde

Acetaldehyde is slightly soluble, and will be incorporated into clouds and rain, but to a much lesser degree than formaldehyde. The rate of the acetaldehyde-OH reaction is roughly two-thirds of the aqueous formaldehyde reaction rate. The product of the aqueous phase oxidation of acetaldehyde is expected to be acetic acid (Jacob et al., 1989).

Acetaldehyde, like formaldehyde, can participate in sulfur chemistry within clouds. Aqueous acetaldehyde combines with aqueous SO_2 to form the stable adduct 1-hydroxy-1-ethanesulfonate (HES) (Olson and Hoffmann, 1989). However, this species does not appear to be of major importance in cloud chemistry.

8.4.3 Acetaldehyde Residence Times

Residence times for acetaldehyde were calculated by considering gas-phase chemical reactions with OH and NO_3 photolysis, in-cloud chemical reaction with OH, and wet and dry deposition. The results of the residence time calculation for acetaldehyde are presented in Table 8-5. During the daytime, under clear-sky conditions, the residence time of acetaldehyde is determined primarily by its reaction with OH, with photolysis accounting for only 2 to 5 percent of the removal. Calculated residence times under these conditions were on the order of a few hours. The National Research Council (NRC, 1981) did not estimate an atmospheric residence time for acetaldehyde, but stated that it would be comparable to the half-life of formaldehyde (2.6 hours, corresponding to a residence time of 3.8 hours). The residence

TABLE 8-5.   Atmospheric residence time calculation for acetaldehyde.   All times are in hours unless otherwise noted.

	Los Angeles		St. Louis		Atlanta		New York	
	July	Jan	July	Jan	July	Jan	July	Jan
Clear sky – day	4	20	3	30	3	30	5	60
Clear sky – night	18	700	170	3000	21	300	40	3000
Clear sky – avg	6	50	4	80	4	70	7	160
Cloudy – day	8	50	6	80	6	80	11	140
Cloudy – night	150	1800	300	3000	150	2000	300	3000
Cloudy – avg	14	130	9	190	10	180	17	400
Rainy – day	--[*]	50	6	60	6	70	11	100
Rainy – night	--[*]	400	300	150	130	200	200	200
Rainy – avg	--[*]	110	9	90	10	120	17	140
Monthly Climatological Average	7	70	6	130	6	110	11	200

[*]Not calculated since July rainfall is zero for Los Angeles.

times presented in Table 8-5 are somewhat longer that those calculated for formaldehyde (Ligocki et al., 1991) because of the slower photolysis rate for acetaldehyde.

In contrast to the situation for formaldehyde, neither in-cloud oxidation nor wet deposition is important for acetaldehyde. In-cloud oxidation accounted for only 1 percent or less of the atmospheric removal of acetaldehyde, compared to 10 to 25 percent of the daytime chemical destruction of formaldehyde and 20 to 90 percent of the nighttime chemical destruction of formaldehyde. The presence of clouds would also be expected to decrease the formation rate of acetaldehyde; thus, cloud cover may actually decrease acetaldehyde concentrations despite the predicted increase in residence time.

At night, for Los Angeles, Atlanta, and New York, the reaction of acetaldehyde with NO_3 leads to residence times on the order of tens of hours during the summertime. However, because of the low NO_3 concentration predicted for St. Louis, the loss of acetaldehyde by reaction with NO_3 is only comparable to the loss by reaction with OH, and neither is rapid.

Dry deposition is not an important removal mechanism for acetaldehyde. Residence times due to dry deposition were estimated to range from 20 days under summer, daytime conditions to over a year for the other conditions. For the cases considered here, dry deposition was a minor removal mechanism except under winter, nighttime conditions. Under these conditions, dry deposition is slow, but all other processes are slower.

The differences in acetaldehyde residence time among cities within a season were not as large as the difference between seasons. The calculated summer residence times are short in most cases, whereas the winter residence times are on the order of days. Thus, acetaldehyde must be considered to be persistent in wintertime. Like formaldehyde, however, the effect of this longer winter residence time is difficult to assess for acetaldehyde because of the importance of secondary formation. Rates of formation of acetaldehyde will be roughly an order of magnitude slower in the wintertime. Thus, it is difficult to predict whether ambient concentrations of acetaldehyde will increase or decrease in winter.

The major uncertainty in the residence time calculation for acetaldehyde is the OH radical concentration, which varies from day to day by roughly a factor of two. The uncertainty in the OH rate constant is much smaller than this (about 13 percent). The uncertainties associated with the photolysis rate, NO_3 concentrations, the rate constant, and dry deposition velocity are of minor importance for acetaldehyde because these processes are relatively slow.

8.4.4 Limited Urban Airshed Modeling Results for Acetaldehyde

The Urban Airshed Model (UAM) has been previously discussed in Section 5.4. Please refer to this section for details about the model, its inputs, and modifications. Much of the information below has been excerpted from reports conducted for EPA by Systems Applications International (SAI) (Ligocki et al., 1992, Ligocki and Whitten, 1991).

St. Louis Study

The Carbon Bond Mechanism-IV chemical mechanism in the UAM uses the "lumped structure approach". In this approach individual chemical species are broken up into reactive units based on the type of bonds and functional groups present in the molecule. In this model, the species ALD2 represents acetaldehyde, the aldehyde functional group of higher aldehydes, and olefins containing internal double bonds which react rapidly in the atmosphere to produce aldehydes. These are the primary ALD2 aldehydes.

Secondary ALD2 is produced through the reactions of paraffins (hydrocarbons with single carbon bonds), olefins (hydrocarbons with double carbon bonds), and other species. A large number of aldehydes of varying size can be produced by the oxidation of a single hydrocarbon.

The magnitude of the changes required to model acetaldehyde explicitly, specifically secondary acetaldehyde, placed this beyond the scope of the St. Louis study. Instead, the results of the St. Louis air toxics simulations presented previously (Ligocki et al., 1991) were re-examined in terms of the ALD2 concentrations.

Results are presented as time-series plots of predicted hourly average ALD2 concentrations and include curves from both the reactive and inert simulations. The results from the base-case simulations are shown in Figure D-4 and Figure D-5 in Appendix D for two representative urban grid cells. The grid cell represented in Figure D-4 is located near the area of maximum mobile-source emissions, and thus represents the area with maximum primary ALD2 impact. The grid cell represented in Figure D-5 is located 8 km downwind, and represents an area where secondary ALD2 production is maximized.

In near-source areas of the modeling domain, ALD2 behaved as a primary species, with concentration peaks in the early morning and early evening (Figure D-4). In downwind areas, however, ALD2 behaved as a secondary species, with concentration peaks in the midafternoon (Figure D-5). The simulation also suggested that motor vehicles may be a more important contributor to ambient acetaldehyde levels than they are to formaldehyde levels.

The dominance of primary ALD2 shown in Figure D-4 for the morning commute hours, combined with the 68 percent contribution of motor vehicle of ALD2, suggest that a large fraction of the simulated ALD2 is attributable to motor vehicles. However, a smaller fraction of the secondary ALD2 is attributable to motor vehicles, because the mobile contribution to the major ALD2 precursor emissions is smaller, particularly in the afternoon when the main ALD2 secondary production occurs.

Simulated ALD2 concentrations were three times as high as measured acetaldehyde concentrations. Because simulated formaldehyde concentrations agreed well with measured concentrations, it is likely that this discrepancy for acetaldehyde is due to the inclusion of higher aldehydes in the ALD2 composite species. If this is the case, urban ambient concentrations of higher aldehydes may be comparable to those of formaldehyde and acetaldehyde.

The results from the day-to-day carryover sensitivity simulations, with the exception of the first few hours of the simulation, are comparable to the base-case results. The peak concentrations were not affected by the change in initial concentrations.

Baltimore-Washington and Houston Area Simulations

For the Baltimore-Washington and Houston area simulations, primary and secondary acetaldehyde were modeled explicitly. The modifications made to UAM to model this species explicitly are described in Ligocki et al. (1992).

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. Maximum daily average acetaldehyde for the 1988 base scenario was 6.1 ppb. Motor vehicle-related acetaldehyde accounted for about 36% of total acetaldehyde emissions, based on the 1995 no motor vehicle scenario. Motor vehicle-related acetaldehyde also accounted for about 15% of total simulated ambient acetaldehyde. 90 to 95% of this acetaldehyde was secondary.

Summer Baltimore-Washington area simulations appear to somewhat overpredict the measured data. Since most of the simulated acetaldehyde is secondary, the concentrations are very

sensitive to the product distribution between acetaldehyde and other higher aldehydes in the chemical mechanism.

In the winter 1988 base scenario, the maximum daily average acetaldehyde concentration was 5.2 ppb, slightly lower than in summer. Simulations for the winter Baltimore-Washington area episode resulted in very small decreases in primary and secondary acetaldehyde. Motor vehicle-related acetaldehyde emissions were about the same with reformulated gasoline use. Motor vehicle-related acetaldehyde accounted for about 13% of the maximum simulated concentration, based on the 1995 no motor vehicle scenario.

For the summer 1987 base scenario in Houston, the maximum daily average acetaldehyde concentration was 18.2 ppb. Motor vehicle-related acetaldehyde accounted for about 13% of total acetaldehyde emissions and 18% of the maximum simulated concentration for the 1987 base scenario. Simulations for the summer Houston episode predicted slight decreases in simulated daily average concentration throughout most of the domain with use of reformulated gasoline. Simulated concentrations of acetaldehyde were in good agreement with measured concentrations.

8.5 Exposure Estimation

8.5.1 Annual Average Exposures Using HAPEM-MS

The data presented in Table 8-6 represent the results determined by the HAPEM-MS modeling that was described previously in Section 4.1.1. These numbers have been adjusted to represent the increase in VMT expected in future years.

The HAPEM-MS exposure estimates in Table 8-6 represent the 50th percentiles of the population distributions of exposure, i.e., half the population will be above and half below these values. High end exposures can also be estimated by using the 95th percentile of the distributions. According to the HAPEM-MS sample output for benzene, the 95th percentile is 1.8 times higher than the 50th percentile for urban areas, and 1.2 times high for rural areas. Applying these factors to the exposure estimates in Table 8-6, the 95th percentiles for urban areas range from 0.32 $\mu g/m^3$ for the 2000 expanded reformulated fuel use and the 2010 expansion of the California standards scenarios, to 0.65 $\mu g/m^3$ for the 1990 base control scenario. The 95th percentiles for rural areas range from 0.11 to 0.24 $\mu g/m^3$, respectively.

8.5.2 Comparison of HAPEM-MS Exposures to Ambient Monitoring Data

As stated in section 4.1.2, four national air monitoring programs/databases contain data on air toxics and the data for

Table 8-6. **Annual Average HAPEM-MS Exposure Projections for Acetaldehyde.**

Year-Scenario	Urban Exposure µg/m^3	Rural Exposure µg/m^3
1990 Base Control	0.36	0.20
1995 Base Control	0.24	0.13
1995 Expanded Reformulated Fuel Use	0.24	0.13
2000 Base Control	0.19	0.10
2000 Expanded Reformulated Fuel Use	0.18	0.10
2000 Expanded Adoption of California Standards	0.19	0.10
2010 Base Control	0.19	0.10
2010 Expanded Reformulated Fuel Use	0.19	0.10
2010 Expanded Adoption of California Standards	0.18	0.09

acetaldehyde is found in only two. The Aerometric Information
Retrieval System (AIRS), and the Urban Air Toxic Monitoring Program
(UATMP) have data for acetaldehyde. The urban exposure data for
acetaldehyde from the two databases are summarized in Table 8-7.

In the 1990 Urban Air Toxics Monitoring Program (UATMP), 332
measurements of acetaldehyde were taken at 12 sites. These sites
were in the cities listed below.

Baton Rouge, LA	Chicago, IL
Camden, NJ	Houston, TX
Orlando, FL	Pensacola, FL
Port Neches, TX	Sauget, IL
Toledo, OH	Washington, D.C.
Wichita, KS	

The highest average was 4.48 $\mu g/m^3$ (2.49 ppb) at an urban
commercial site in Baton Rouge, Louisiana. Twenty-two samples were
collected at this site. The lowest average was 1.34 $\mu g/m^3$ (0.75
ppb) at a suburban residential site in Houston, Texas. Twenty-
three samples were collected at this site. The overall average of
the averages for each site was 3.10 $\mu g/m^3$ (1.72 ppb). Ozone was
removed from the ambient air collected in this program through the
use of an ozone denuder. The use of an ozone denuder in the
sampling system resulted in higher, but more accurate, reported
acetaldehyde concentrations. Only the 1990 UATMP data will be used
for the comparisons in this study.

HAPEM-MS assumes that the dispersion and atmospheric chemistry
of acetaldehyde is similar to CO. This assumption would appear to
be somewhat valid for acetaldehyde since it is less reactive than
formaldehyde, but acetaldehyde is transformed in the atmosphere to
some extent. To test the reasonableness of the HAPEM-MS modeling
results, the HAPEM-MS results for 1990 are compared to ambient
monitoring results for recent years. Before comparing the HAPEM-MS
results to the ambient data, the ambient monitoring data must be
adjusted to represent the amount that is attributed to motor
vehicles. The data derived from emission inventories and
atmospheric modeling conducted by SAI for St. Louis (Ligocki and
Whitten, 1991) estimate that 39% of the ambient acetaldehyde can be
apportioned to motor vehicles. This number actually represents
acetaldehyde and higher aldehydes.

This estimate is higher than the estimates in the Houston and
Baltimore-Washington Area UAM-Tox simulations (Ligocki et al.,
1992). In these studies, acetaldehyde was modeled explicitly;
thus, the estimates do not represent both acetaldehyde and higher
aldehydes. In Baltimore-Washington, motor vehicle-related
acetaldehyde accounted for about 15% of total simulated ambient
acetaldehyde in summer, while in Houston, motor vehicle-related
acetaldehyde accounted for about 18% of the maximum simulated
concentration.

The estimate of 39% will be used in this study to represent
the nationwide average percentage of ambient acetaldehyde
attributable to motor vehicles, while acknowledging the apparent

area-to-area variations and the possibility that this may overestimate the motor vehicle contribution to ambient acetaldehyde, possibly in part because the estimate actually represents both acetaldehyde and higher aldehydes. The numbers in the second column of Table 8-7 below are 39% of the ambient levels and thus represent estimated motor vehicle levels.

The motor vehicle apportionment of the ambient monitoring data ranges from 0.94 to 1.21 $\mu g/m^3$. When the adjustment factor of 0.622 for the ambient mobile source levels, that was determined in Section 5.5.2 is applied, this range becomes 0.58 to 0.75 $\mu g/m^3$. Due to a potential ozone interfernece problem with the ambient data other than the 1990 UATMP, only the 1990 UATMP adjustment estimate, 0.75 $\mu g/m^3$, will be used for the comparison to HAPEM. When compared to the HAPEM-MS 1990 base control level of 0.36 $\mu g/m^3$, the 1990 UATMP adjusted ambient monitoring data is observed to be approximately two times greater then the HAPEM-MS base control level. The fact that modeled levels are 62% lower than monitored data is consistent with secondary-formed acetaldehyde. 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. All analysis based on the HAPEM-MS ambient mobile source levels will have this factor applied. Adjusted urban, rural, and nationwide exposures are found in Table 8-8.

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. 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.

8.5.3 Short-Term Microenvironment Exposures

The primary emphasis for acetaldehyde exposure will be exposure in microenvironments that are enclosed, increasing the exposure to tailpipe emissions. These microenvironments include in-vehicle and parking garage exposure, though, actual exposure information is only available for in-vehicle exposure. This information is taken from the In-Vehicle Air Toxics Characterization Study in the South Coast Air Basin (Shikiya et al., 1989), which focused on the driver's exposure to VOC's in the southern California area. See the information in Section 4.2 for more details about the methodology, and Section 5.5.3 for a description of the study.

Table 8-7. Air Monitoring Results for Acetaldehyde.

Program	Years	Ambient Data[a] µg/m^3	Estimated Mobile Source Contribution[b] µg/m^3
AIRS	1988	2.93	1.14
	1987	2.41	0.94
UATMP	1989	2.45	0.96
	1990	3.10	1.21

[a]Caution should be taken in comparing these numbers. The methods of averaging the data are not consistent between air monitoring databases. The sampling methodology is also inconsistent.

[b]The ambient data are adjusted to represent the motor vehicle contribution to the ambient concentration, which for acetaldehyde is estimated to be 39%, based on emissions inventory apportionment and modeling.

**Table 8-8.   Adjusted Annual Average HAPEM-MS Exposure
Projections for Acetaldehyde.**

Year-Scenario	Exposure ($\mu g/m^3$)		
	Urban	Rural	Nationwide
1990 Base Control	0.75	0.41	0.67
1995 Base Control	0.49	0.35	0.44
1995 Expanded Reformulated Fuel Use	0.49	0.35	0.44
2000 Base Control	0.38	0.21	0.33
2000 Expanded Reformulated Fuel Use	0.38	0.20	0.33
2000 Expanded Adoption of California Standards	0.39	0.21	0.33
2010 Base Control	0.39	0.21	0.34
2010 Expanded Reformulated Fuel Use	0.38	0.21	0.34
2010 Expanded Adoption of California Standards	0.36	0.19	0.31

The in-vehicle exposure level of acetaldehyde was determined in this study to have a mean of 13.7 µg/m³ and a maximum measured level of 66.7 µg/m³. This compares to ambient levels of 2.41 to 3.10 µg/m³ determined through air monitoring studies and presented in Table 8-7. Since for the majority of the population these are short-term acute exposures to acetaldehyde, the concern would be with non-cancer effects. The primary acute effect of exposure to acetaldehyde vapors is irritation of the eyes, skin, and respiratory tract. At high concentrations, irritation and ciliastatic effects can occur. Clinical effects include erythema, coughing, pulmonary edema, and necrosis. 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 (3.6×10^5 µg/m³). Please see Section 8.8 for more information on non-cancer effects.

A RfC of 9.0 µg/m³ per day over a lifetime has been developed by EPA. An RfC is an estimate of the continuous exposure to the human population that is likely to be without deleterious effects during a lifetime. The mean and maximum levels, 13.7 µg/m³ and 66.7 µg/m³ respectively, observed in Shikiya et al., (1989) are higher than RfC developed by EPA.

Due to more stringent fuel and vehicle regulations, short-term exposure to acetaldehyde in microenvironments is expected to decrease in future years.

8.6 Carcinogenicity of Acetaldehyde and Unit Risk Estimates

8.6.1 Most Recent EPA Assessment

An external review draft document entitled <u>Health Assessment Document for Acetaldehyde</u> (EPA, 1987) has been prepared. Much of the information contained in this section has been taken from this document and the most recent IRIS summary (EPA, 1992).

8.6.1.1 Description of Available Carcinogenicity Data

The majority of information that exists to evaluate the carcinogenicity of acetaldehyde emissions relies on mutagenicity studies and a few animal studies.

<u>Genotoxicity</u>

Acetaldehyde has been shown in studies by several different laboratories to induce sister chromatid exchange (SCE) in cultured mammalian cells, e.g., Chinese hamster cells (Obe and Ristow, 1977; Obe and Beer, 1979; de Raat et al., 1983) and human peripheral lymphocytes (Ristow and Obe, 1979; Jansson, 1982; Böhlke et al., 1983; Norrpa et al., 1985; Obe et al., 1986) in a dose-related manner. A study by He and Lambert (1985) provided evidence that SCE-inducing lesions may be persistent for several cell generations. The induction of SCEs by acetaldehyde has also been detected in the bone marrow cells of whole mammals, namely mice and Chinese hamsters (Obe et al., 1979; Korte and Obe, 1981). In addition to acetaldehyde's ability to induce SCEs, it has been

shown to be a clastogen in mammalian cell cultures (Bird et al., 1982) and plants (Rieger and Michaelis, 1960). Acetaldehyde has produced chromosomal aberrations (micronuclei, breaks, gaps, and exchange-type aberrations) also in a dose-related manner (Bird et al., 1982; Böhlke et al., 1983). In a study by Eker and Sanner (1986), acetaldehyde and formaldehyde were both able to initiate cell transformation, though formaldehyde was 100 times more potent. In *Drosophila*, chromosomal effects (i.e., reciprocal translocations) were not found after acetaldehyde treatment (Woodruff et al., 1985). The clastogenicity of acetaldehyde in whole mammals has not been sufficiently evaluated. In the one study that was available, female rats were intra-amniotically injected on the 13th day of gestation, and the treated embryos had high frequencies of chromosomal gaps and breaks (Barilyak and Kozachuk, 1983).

Although acetaldehyde did not produce chromosomal translocations in *Drosophila*, it was found to induce gene mutations (sex-linked recessive lethals) at the same concentration when administered by injection (Woodruff et al., 1985). Positive results for gene mutations were reported in the nematode, *Caenorhabitis* (Greenwald and Horvitz, 1980), and an equivocal result was obtained for mitochondrial mutations in yeast (Bandas, 1982). *Salmonella* testing in numerous strains has been reported as negative (Commoner, 1976; Laumbach et al., 1976; Pool and Wiesler, 1981; Marnett et al., 1985; Mortelmans et al., 1986). In two studies utilizing *Escherichia coli* to detect a mutagenic effect, one yielded positive results (Veghelyi et al., 1978) and the other study negative results (Hemminki et al., 1980). There were no available data on the ability of acetaldehyde to produce gene mutations in cultured mammalian cells.

Acetaldehyde has not been shown to cause DNA strand breaks in mammalian cells *in vitro* (Sina et al., 1983; Saladino et al., 1985; Lambert et al., 1985). However, if acetaldehyde produces SCEs and chromosomal aberrations by DNA-DNA or DNA-protein cross-linking, it may not necessarily produce DNA strand breaks (Bradley et al., 1979). Acetaldehyde has been shown to produce crosslinks between protein and DNA in the nasal respiratory mucosa of rats (Lam et al., 1986).

In conclusion, 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*. Acetaldehyde produced gene mutations in *Drosophila* but not in *Salmonella*; no studies were found for cultured mammalian cells. Thus, the available evidence indicates that acetaldehyde is mutagenic and may pose a risk for somatic cells. On the other hand, it has been suggested that acetaldehyde may be capable of deactivating free cysteine in bronchial epithelial cells, thereby suppressing the "thiol defense" of the epithelium against the attack of mutagens and carcinogens (Braven et al. 1967; Fenner and Braven, 1968). Current knowledge, however, is inadequate with regard to germ cells (reproductive cells) 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.

Animal Data

Acetaldehyde has been tested for carcinogenicity in hamsters by intratracheal instillation and inhalation and in rats by subcutaneous injection and inhalation. In the inhalation/instillation study of hamsters (Feron, 1979), two testing protocols were used. In part one, male hamsters were exposed to 0 or 1500 ppm acetaldehyde by inhalation 7 hours/day, 5 days/week, for 52 weeks. These animals were also exposed intratracheally to benzo[a]pyrene (BaP) for a total concentration at the end of 52 weeks ranging from 0 to 52 mg. Exposure to acetaldehyde by inhalation and intratracheal BaP induced inflammatory changes, hyperplasia and metaplasia of the nasal, laryngeal, and tracheal epithelium, and tumors of the nose and larynx. Acetaldehyde enhanced the development of BaP-initiated tracheobronchial carcinoma yielding twice the incidence of squamous cell carcinoma compared with the same dose of BaP alone. No neoplastic effects due to acetaldehyde alone were found.

In the second part of Feron (1979), male and female hamsters were intratracheally instilled with 4 or 8 µL acetaldehyde, BaP, BaP and 4 µL acetaldehyde, diethylnitrosamine (DENA, a tumor promotor), or DENA and 4 µL acetaldehyde. Acetaldehyde alone produced no tumors in the larynx, trachea, or bronchi. However, large numbers of tracheal papillomas and lung adenomas were found in groups treated with acetaldehyde plus BaP or DENA. There was no evidence of acetaldehyde enhancing the development of DENA-initiated respiratory tract tumors.

In an extension of the above study (Feron et al., 1982), male and female hamsters were exposed to a high concentration of acetaldehyde vapor alone or simultaneously with either BaP or DENA. The animals were exposed 7 hours/day, 5 days/week, for 52 weeks to a time weighted average concentration of 2028 ppm. Tumors were slightly increased in the nose and significantly increased in the larynx of animals exposed to acetaldehyde vapor alone, but no tracheal tumors were observed. The incidence of carcinomas in the trachea and bronchi were significantly higher in hamsters exposed to acetaldehyde and treated with high doses of BaP than in hamsters treated with the same dose of BaP but exposed to air. There was no evidence that acetaldehyde exposure increased the incidence or affected the type of DENA-induced tumors in any part of the respiratory tract.

Watanabe and Sugimoto (1956) reported spindle-cell sarcoma in 20% to 25% of the rats tested at the site of repeated acetaldehyde injection. No conclusion can be drawn from this study because neither the total doses of acetaldehyde nor the tumor incidence in controls could be determined from available data.

The carcinogenicity of acetaldehyde was studied in albino SPF Wistar rats (Woutersen and Appelman, 1984; Woutersen et al., 1985,

1986). The studies are summarized in Table 8-9. The animals were
exposed by inhalation to atmospheres containing 0, 750, 1500, or
3000 ppm acetaldehyde for 6 hours/day, 5 days/week for 27 months.
The concentration in the highest dose group was gradually reduced
from 3000 to 1000 ppm because of severe growth retardation,
occasional loss of body weight, and early mortality in this group.
Interim sacrifices were carried out at 13, 26, and 52 weeks. One
tumor was observed in the 52 week sacrifice group and none at
earlier times. Exposure to acetaldehyde increases the incidence of
tumors in an exposure-related manner in both male and female rats.
In addition, there were exposure-related increased in the incidence
of multiple respiratory tract tumors. Adenocarcinomas were
increased significantly in both male and female rats at all
exposure levels, whereas squamous cell carcinoma were increased
significantly in male rats at middle and high doses and in the
female rats only at the high dose. The squamous cell carcinomas
incidence showed a clear dose-response relationship. The incidence
of adenocarcinomas was highest in the mid-exposure group (1500 ppm)
in both male and female rats, but this was probably due to the high
mortality and competing squamous cell carcinomas at the highest
exposure level. In the low-exposure group, the adenocarcinoma
incidence was higher in males than in females.

In a concurrent study, referred to as the "recovery study", 30
animals of each sex were exposed to the same concentrations of
acetaldehyde for 52 weeks followed by a recovery period of 26 weeks
or 52 weeks. Significant increases in nasal tumors were observed
in male and female rats, including adenocarcinomas and squamous
cell carcinomas, in both recovery groups. These findings indicate
that after 52 weeks of exposure to acetaldehyde, proliferative
epithelial lesions of the nose may develop into tumors even without
continued exposure.

Table 8-9. Animal Data Used for EPA's Unit Risk Estimates.

REPORT	ANIMAL	EXPOSURE CONCENTRATION	LENGTH OF EXPOSURE	MAJOR RESULTS
Woutersen and Appelman (1984) also as Wouterson et al., (1986) Woutersen et al. (1985) "lifetime study"	albino SPF Wistar rats male and female	0, 750, 1500, and 3000 ppm (lowered to 1000 ppm) of acetaldehyde	6h/d, 5d/wk, for 27 months	1. Acetaldehyde vapor exposure caused two types of tumors in the nasal tract of rats in an exposure related manner. The four exposure levels gave tumor incidences of 1, 21, 52, and 51% respectively. 2. Degeneration of nasal tissue was observed at all dose levels. 3. Exposure appears not to affect any other organ directly except for lesions of the larynx and, to a minor degree, the trachea. 4. Animals in the high exposure group (3000 ppm) suffered severe growth retardation, respiratory distress, and high early mortality.
Woutersen and Appelman (1984) recovery subgroup of original study	albino SPF Wistar rats male and female	same exposures as used above subjected to a 26 or a 52 week recovery period	same as used above but for 52 weeks	With respect to recovery, during the first 26 weeks the nasal tumor rates and death rates were essentially the same as the lifetime exposure group. From 26-52 weeks both low- and mid-exposure recovery groups had significantly decreased nasal tumor rates. This indicates that nasal lesions may still develop into tumors even after exposure stops, and that the nasal tissue may also be able to repair some damage.

Human Data

The only epidemiological study involving acetaldehyde exposure showed an increased crude incidence rate of total cancer in acetaldehyde production workers as compared to the general population (Bittersohl, 1974). The study was performed on workers from an aldol and aliphatic acetaldehyde factory. The study showed a five times higher cancer rate than that of the general population. An incidence rate of 6000/100,000 population for total cancer was calculated for this study, which contrasts with an incidence rate of 1200/100,000 for the general population of Germany during the same period. Because the incidence rate was not age adjusted, and because this study has several other major methodological limitations (concurrent exposure to cigarette smoke and other chemicals, short duration, small number of subjects, and lack of information on subject selection, age, and sex distribution), the evidence is considered inadequate for the carcinogenicity of acetaldehyde in humans.

8.6.1.2 Weight-of-Evidence Judgement of Data and EPA Classification

The data used for the quantitative estimates for acetaldehyde are limited to the Woutersen and Appelman (1984) and the Woutersen et al., (1985) rat inhalation studies (summarized in Table 8-9) showing an exposure related increase in nasal tumors in Wistar rats and supported by positive results for mutagenicity. This evidence for carcinogenicity of acetaldehyde in animals is considered to be sufficient based on the U.S. EPA cancer assessment guidelines. Neither of the hamster studies (Feron, 1979; Feron et al., 1982) are considered satisfactory based on the fact that one was an intratracheal instillation study and the other was an inhalation study which had very high exposure levels of acetaldehyde and only one exposure group.

The only epidemiological study, Bittersohl (1974), showed an increase in crude incidence rate of total cancer in acetaldehyde production workers as compared with the general population. Because the incidence rate was not age adjusted, and because this study has several other major methodological limitations, the evidence is considered inadequate for the carcinogenicity of acetaldehyde in humans.

On the basis of inadequate evidence for carcinogenicity of acetaldehyde emissions in humans, and relying totally on the sufficient evidence from animals and mutagenicity, acetaldehyde emissions are considered to best fit the weight-of-evidence category B2. This classifies acetaldehyde as a probable human carcinogen.

8.6.1.3 Data Sets Used for Unit Risk Estimates

To actually determine the unit risk of acetaldehyde, only two of the animal studies are selected for risk calculations because they are inhalation studies that involve more than one exposure group. The two rat studies used are Woutersen and Appelman (1984) (also known as Wouterson et al., 1986), and Woutersen et al. (1985). These studies are summarized in Table 8-9.

8.6.1.4 Dose-Response Model Used

The linearized multistage model is used to calculate unit risk estimates using various exposure inputs. All unit risk estimates that currently exist for acetaldehyde are based exclusively on animal data.

8.6.1.5 Unit Risk Estimates

The upper-limit unit risk estimate for acetaldehyde is $2.2 \times 10^{-6} (\mu g/m^3)^{-1}$, derived from the male rat tumor data. Corresponding maximum likelihood estimates (MLE's) were not given.

8.6.2 Other Views and Risk Estimates

This section presents alternate views and/or risk assessments for acetaldehyde.

International Agency for Research on Cancer (IARC)

IARC has classified acetaldehyde as a Group 2B carcinogen. A Group 2B carcinogen is defined as an agent that is *possibly* carcinogenic to humans. This classification is based on inadequate evidence for carcinogenicity in humans and sufficient evidence for carcinogenicity in animals (IARC, 1987).

California Air Resources Board (CARB)

CARB (1992b), like EPA and IARC, has concluded that acetaldehyde is a probable human carcinogen. CARB (1992b) has performed an assessment of the carcinogenic risk of acetaldehyde using the Wouterson et al., (1986) rat nasal carcinoma data (discussed previously in Section 8.6.1) in the linearized time-dependent multistage model. However, their assessment differs from EPA (1987) in the following ways:

(1)   The EPA (1987) risk assessment considered all 55 animals in the experimental groups to be at risk, whereas CARB used only the 49-53 animals of each group that were examined for nasal changes.

(2)   CARB used only the male rat data from Wouterson et al., (1986) whereas EPA used both the male and the female data. CARB stated that the male rat is more sensitive to

tumor induction by acetaldehyde than the female rat and this is the proper sex to select based on CARB procedures for cancer risk assessment.

(3) The EPA (1987) risk assessment combined two experiments by Wouterson et al., the lifetime exposure experiment and an experiment in which one year of exposure was followed by one year of recovery. CARB , however, used only the lifetime exposure experiment.

(4) EPA (1987) used two versions of the linearized multistage model: the standard version and the time-to-tumor version. CARB used only the standard version citing that the information to adequately use the time-to-tumor version was not available in the experimental data and thus should not be used.

(5) The CARB approach uses three different scaling factors to extrapolate the equivalent dose rate from rats to humans. EPA (1987) did not specifically discuss the issue of scaling to extrapolate from rodents to humans for formaldehyde.

The UCL for unit risk for lifetime exposure calculated by CARB (1992b) using the methods and assumptions described above is 4.8×10^{-6} ppb^{-1} (2.7×10^{-6} [µg/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} [µg/m^3]$^{-1}$).

8.6.3 Recent and Ongoing Research

8.6.3.1 Genotoxicity

Dulout and Furnus (1988) determined that the most notable cytogenetic effect of acetaldehyde in cultured Chinese hamster ovary (CHO) cells was aneuploidy (the chromosome number is not an exact multiple of the haploid number) and not chromosomal breakage. Acetaldehyde added for 24 hours to cultures at concentrations of 0.002%, 0.004%, and 0.006% produced an increased frequency of aneuploidy as compared to controls. The aneuploidy was observed at all doses tested, whereas chromosomal aberrations and sister chromosomal exchanges only occurred at the two highest levels.

The effect of acetaldehyde on the frequency of meiotic micronuclei in groups of four hybrid male mice was assessed 13 days after a single intraperitoneal injection of 0, 125, 250, 375, of 500 mg/kg acetaldehyde in saline solution. No significant increases in the frequency of micronuclei were observed (Lahdetie, 1988). The alkaline dilution technique was used by Garberg, et al. (1988) to determine whether the DNA of mouse lymphoma cells exposed to acetaldehyde contain single-strand breaks. Single -strand breaks were not detected in this cell type or in rat hepatocytes, human lymphocytes, and bronchial epithelial cells studied

previously (Sina et al., 1983, Lambert et al., 1985; Saladino et al., 1985).

8.6.3.2 Metabolism and Pharmacokinetics

The following has been excerpted from EPA (1987). The extensive references have been omitted to facilitate the comprehension of this section. For the complete list of references, please consult Chapter 4 of EPA (1987). Other studies that have been published since the issuance of the 1987 draft document support the 1987 position summarized below.

The principal routes of entry of acetaldehyde into the body are by gastrointestinal and inhalation absorption. Acetaldehyde, whether from exogenous (from outside the body) sources or generated from ethanol metabolism, is known to be very rapidly and extensively metabolized oxidatively in mammalian systems to a normal endogenous (inside the organism) metabolite, acetate, primarily by aldehyde dehydrogenases (specific enzymes) widely distributed in body tissue. Acetate enters the metabolic pool of intermediary metabolism and is used in cellular energy production (end products CO_2 and water) or in synthesis of cell constituents. In contrast to the situation for acetaldehyde generated from ethanol metabolism, there are few studies of the kinetics of acetaldehyde of exogenous origin, i.e., from environmental exposure or experimental dosing. It is known, however, that all mammalian species have a high capacity to rapidly and virtually completely metabolize acetaldehyde by most tissues in the body, including the gastrointestinal mucosa and respiratory mucosa and lungs. Although hepatic (liver) capacity is the highest after oral or inhalation administration, experimental evidence indicates that a substantial first-pass metabolism in the liver or respiratory organs effectively limits acetaldehyde access to the systemic circulation. However, adequate studies have not been conducted to establish dose-metabolism relationships, or dose-blood concentration relationships.

Acetaldehyde readily crosses body compartmental membranes into virtually all body tissues, including the fetus, after administration or endogenous generation. Animal experiments have demonstrated a rapid exponential disappearance from circulating blood, consistent with first-order kinetics, with a short half-time of elimination of less than 15 minutes. Since less than 5 percent escapes unchanged in exhaled breath, and acetaldehyde is not known to be excreted into the urine, the elimination from the body is essentially by metabolism.

Acetaldehyde is a highly reactive compound and at high concentrations episode, for example, at the respiratory mucosa with inhalation exposure, it readily forms adducts nonenzymatically with membranal and intracellular macromolecules. Stable and reversible adduct formation including cross-linking have been demonstrated with proteins, nucleic acids (including DNA), and phospholipids. Moreover, even at physiological levels (10 to 150 µmol/L blood), acetaldehyde has been found to form adducts with cellular macromolecules. From these observations, it has been considered

that acetaldehyde-adduct formation may play a role in the organ and cellular injury associated with acetaldehyde toxicities, and in the potential promoter or carcinogenic effect assigned to this compound. Acetaldehyde also readily reacts nonenzymatically with cysteine and glutathione (proteins with sulfur groups [thiols] attached) to form stable and reversible adducts, respectively. Hence, acetaldehyde may be an effective depleter of these important cellular nonprotein thiols, which represent a thiol defense against the attack of toxic aldehydes and other mutagens and carcinogens.

8.7 Carcinogenic Risk for Baseline and Control Scenarios

Table 8-10 summarizes the annual cancer incidences for all the scenarios. These numbers are presented as decimals due to the fact that the cancer cases are low enough that rounding the decimal up or down would significantly affect the total number. The cancer cases do decrease slightly from a comparison drawn between base control scenarios. 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 24% in 1995, 45% in 2000 and 65% 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.

From Table 8-10 it can also be observed that the expanded use of reformulated fuel and the expansion of the California standards provide no significant decrease in the cancer cases and, in several scenarios, the cancer cases increase. As mentioned in previous sections, the exposure estimates are based on changes in direct emissions of acetaldehyde. Changes in reactivity of the emissions, which would result in changes to secondary acetaldehyde, are not accounted for. Since it is probable that secondary acetaldehyde could be reduced with the use of oxygenates, the cancer risk estimates given in Table 8-10 should be considered conservative estimates.

Table 8-10. Annual Cancer Incidence Projections for Acetaldehyde.[a,b]

Year-Scenario	Emission Factor g/mile	Urban Cancer Cases	Rural Cancer Cases	Total Cancer Cases	Percent Reduction from 1990	
					EF	Cancer
1990 Base Control	0.0119	4.5	0.8	5.3	–	–
1995 Base Control	0.0071	3.0	0.6	3.6	40	32
1995 Expanded Reformulated Fuel Use	0.0071	3.0	0.6	3.6	40	32
2000 Base Control	0.0051	2.4	0.4	2.8	57	47
2000 Expanded Reformulated Fuel Use	0.0051	2.4	0.4	2.8	57	47
2000 Expanded Adoption of California Standards	0.0052	2.4	0.4	2.8	56	47
2010 Base Control	0.0045	2.6	0.4	3.0	62	43
2010 Expanded Reformulated Fuel Use	0.0044	2.6	0.4	3.0	63	43
2010 Expanded Adoption of California Standards	0.0041	2.4	0.4	2.8	66	47

[a]Projections have inherent uncertainties in emission estimates, dose response, and exposure.
[b]Cancer incidence estimates are based on upper bound estimates of unit risk, determined from animal studies. Acetaldehyde is classified by EPA as a category B2, probable human carcinogen, based on insufficient evidence in humans and sufficient evidence in animals and in mutagenicity bioassays.

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.

8.8 Non-Carcinogenic Effects of Inhalation Exposure to Acetaldehyde

Since the focus of this report is on the carcinogenic potential of the various compounds, the noncancer information will be dealt with in a more cursory fashion. No attempt has been made to synthesize and analyze the data encompassed below. Also, no attempt has been made to accord more importance to one type of noncancer effect over another. The objective is to research all existing data, describe the noncancer effects observed, and refrain from any subjective analysis of the data.

8.8.1 Toxicity

The results of eight acute toxicity studies in mammals, by inhalation in rats (Skog, 1950; Lewis and Tatkin, 1983) and mice (Kane et al., 1980; Barrow, 1982), by the oral route in rats (Windholz et al., 1983; Lewis and Tatkin, 1983; Omel'yanets et al., 1978) and mice (National Research Council, 1977) along with intravenous instillation in guinea pigs (Mohan et al., 1981) and subcutaneous injection in rats and mice (Skog, 1950) all show LD_{50} effects. The acute oral LD_{50} of acetaldehyde ranged from 1232 mg/kg to 5300 mg/kg. The LD_{50} for subcutaneous injection ranged from 560 mg/kg to 640 mg/kg. The acute inhalation LC_{50} was 20,000 ppm in rats exposed to acetaldehyde for 30 minutes. In one study (Lewis and Tatkin, 1983), 4000 ppm for 4 hours killed some exposed rats. The following section will discuss some of these acute toxicity studies in more detail.

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 (Kane et al., 1980; Barrow, 1982). This yielded RD_{50}'s (the concentration that produces a 50% decrease in respiratory rate) of 4946 ppm and 2845 ppm, respectively. The current TLV for acetaldehyde is 100 ppm (American Conference of Governmental Industrial Hygienists, 1980), and is between 0.1 and 0.01 times the cited RD_{50} values. In rats, acetaldehyde increased blood pressure and heart rate after exposure by inhalation (Egle, 1972) and intravenous injection (Mohen et al., 1981; Egle et al., 1973).

Three subchronic inhalation studies in rats and one in hamsters have been conducted. Appelman et al. (1982) used the exposure information discussed below in the RfC section. Rats exposed to the highest concentration exhibited severe dyspnea and marked excitation during the first 30 minutes of exposure. Rats at the highest exposure also exhibited decreased body weight, lymphocytes, and liver weights when compared to controls. The neutrophil counts and lung weights were increased. Histopathological alterations of the respiratory system were seen

at all dose levels, with the nose being the most severe. The study by Appelman et al. (1986) (exposure information is detailed in RfC section) was also observed for non-cancer effects. Uninterrupted exposure to 500 ppm did not produce any changes in condition, behavior, or body weight of the rats; however, rats exposed to 500 ppm with a peak exposure of 3000 ppm exhibited irritation, as indicated by eye blinking, excessive running, and nose twitching. Mean body weights in the latter groups were significantly lower than in controls. In addition, a reduced phagocytotic index was significantly decreased at the highest dose.

The effect of acetaldehyde on pulmonary mechanics was studied following exposure of groups of Wistar rats to acetaldehyde vapors at concentrations of 0 or 243 ppm (0 or 105.3 mg/m^3), 8 hours/day, 5 days/week for 5 weeks. (Hilaro et al., 1985). A significant increase in respiratory frequency, functional residual capacity, residual volume, and total lung capacity was noted. The subchronic inhalation study in hamsters (Kruysse et al., 1975) is discussed below in the RfC section.

One subchronic investigation of the effects of acetaldehyde, on the phospholipid composition of pulmonary surfactant, was found in the literature (Prasanna et al., 1981). Pulmonary surfactant is a lipoprotein complex with a high phospholipid content which prevents alveolar collapse during expiration by maintaining the stability and physical elasticity of the alveolar walls, and by reducing the surface tension of the fluid lining the alveoli. Acetaldehyde injected intraperitoneally to rats at 200 mg/kg significantly reduced the phospholipid concentration of pulmonary surfactant. In two subchronic intravenous studies, one in guinea pigs (Mohen et al., 1981), and the other in rats (Egle et al., 1973), a dosage of 20 mg/kg acetaldehyde or lower caused an immediate and significant increase in blood pressure.

In a chronic inhalation study (Feron, 1979), acetaldehyde vapor at 1500 ppm for 52 weeks produced systemic effects in the hamster: growth retardation, slight anemia, increased UGOT (urinary glutamic-oxaloacetic transaminase) activity, increased urine protein content, increased kidney weights, and histopathological changes in the nasal mucosa and trachea. In a separate study (Feron, 1979), intratracheal instillation of acetaldehyde (2 to 4 percent) to hamsters weekly or biweekly for up to 52 weeks caused severe hyperplastic and inflammatory changes in the bronchioalveolar region of the respiratory tract. In Feron et al. (1982), male and female hamsters exposed to levels of acetaldehyde vapor decreasing from 2500 ppm to 1650 ppm over 52 weeks had lower body weights than controls and distinct histopathological changes in the nose, trachea, and larynx.

Humans are frequently exposed to acetaldehyde from cigarette smoke, vehicle exhaust fumes, or other sources. Metabolism of ethanol would be the major source of acetaldehyde among consumers of alcoholic beverages.

The primary acute effect of exposure to acetaldehyde vapors is irritation of the eyes, skin, and respiratory tract (Sim and Pattle, 1957). At high concentrations, irritation and ciliastatic effects can occur, which could facilitate the uptake of other contaminants (NRC, 1981). Clinical effects include erythema, coughing, pulmonary edema, and necrosis (Dreisbach, 1980). 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 (Sittig, 1979). It was concluded by the Committee of Aldehydes of the National Research Council (1981) that direct pulmonary sensitization to aldehyde vapors appears to be relatively rare and asthma-like symptoms are rarely caused by the inhalation of aldehydes.

The main route of occupational exposure is by inhalation of acetaldehyde vapor. The allowable federal time weighted average (TWA) is 200 ppm (360 mg/m^3) for eight hours per day, five days per week. The American Conference of Governmental and Industrial Hygienists (1985) recommends a threshold limit value (TLV) of 100 ppm (180 mg/m^3) for eight hours per day, five days per week.

8.8.2 Reference Concentration for Chronic Inhalation Exposure (RfC)

At the present time, the reference dose for chronic oral exposure (RfD) assessment is not available but the reference concentration for chronic inhalation exposure (RfC) has recently been completed (EPA, 1992). An RfC is an estimate of the continuous exposure to the human population that is likely to be without deleterious effects during a lifetime.

Two short term studies conducted by the same research group are the principal studies used. While these studies are short term in duration, together they establish a concentration-response for lesions after only 4 weeks of exposure.

Appelman et al. (1986) conducted two inhalation studies on male Wistar rats exposing them 6 hrs/day, 5 days/week for 4 weeks to 0, 150, and 500 ppm (0, 273, and 910 mg/m^3, respectively. One group was exposed without interruption, a second group was interrupted for 1.5 hours halfway through the exposure, and a third group was interrupted as described with a peak exposure imposed four times in a three hour period (concentration at peak was six times the basic concentration). Degeneration of the olfactory epithelium was observed in rats exposed to 500 ppm. Interruption of the exposure or interruption combined with peak exposure did not visibly influence this adverse effect. No compound-related effects were observed in rats interruptedly or uninterruptedly exposed to 150 ppm during the 4 week exposure period; therefore, the NOAEL is 150 ppm (no-observed-adverse-effect level).

Appelman et al, (1982) exposed Wistar rats for 6 hour.day, 5 days/week for 4 weeks to 0, 400, 1000, 2200, or 5000 ppm

acetaldehyde (0, 728, 1820, 4004, and 9100 mg/m^3). The nasal cavity was most severely affected and exhibited a concentration-response relationship. At all levels of acetaldehyde exposure in this experiment, there was found nasal olfactory degeneration that increased in severity as the concentration increased. Also as the concentration increased, the laryngeal and tracheal epithelium became involved (1000 to 5000 ppm). Based on the degenerative changes observed in the olfactory epithelium, the 400 ppm level is designated as a LOAEL (lowest-observed-adverse-effect level).

There are also three additional studies used to support the inhalation RfC. Woutersen et al. (1986), which was discussed previously, exposed rats to 0, 750, 1500, and 3000/1000 ppm acetaldehyde vapor. The only exposure related histopathology occurred in the respiratory system and showed a concentration-response relationship. The most severe abnormalities were found in the nasal cavity. Basal cell hyperplasia of the olfactory epithelium was seen in the low- and mid-concentration rats. The decrease in these changes in the olfactory epithelium was attributed to the incidence of adenocarcinomas at the higher levels. The lowest exposure concentration, 750 ppm, is clearly a LOEAL based on the above changes in the olfactory epithelium.

Woutersen and Feron (1987) conducted an inhalation study in which Wistar rats were exposed to 0, 750, 1500, or 3000/1500 ppm acetaldehyde (0, 1365, 2730, 5460/2730 mg/m^3, respectively) for 6 hours/day, 5 days/week for 52 weeks with a 26- or 52-week recovery period. Degeneration of the olfactory epithelium was similar in rats terminated after 26 weeks of recovery and rats killed immediately after exposure termination. Histopathological changes found in the respiratory epithelium were comparable with, but less severe than, those observed immediately after exposure termination. After 52 weeks of recovery, the degeneration of the olfactory epithelium was still visible to a slight degree in animals from all exposure groups. The data suggest that there is incomplete recovery of olfactory and respiratory epithelium changes induced at all exposure concentrations for periods as long as 52 weeks after exposure termination.

Kruysse et al. (1975) conducted a 90-day inhalation study in hamsters. The hamsters were exposed to acetaldehyde vapor at concentrations of 0, 390, 1340, or 4560 ppm (0, 127, 435.3, or 1482 mg/m^3), for 6 hours/day, 5 days/week for 90 days. In this study, as in the previous studies, the histopathological changes attributable to exposure were observed only in the respiratory tract. At the 390 ppm concentration, with one exception, no adverse effects were observed. The 390 ppm concentration was identified by the authors as a NOAEL.

The final RfC was calculated using the NOAEL from Appelman et al. (1986) of 273 mg/m^3, an uncertainty factor (UF) of 1000, and a modifying factor (MF) of 1. The UF of 1000 was obtained by assigning an uncertainty factor of 10 to account for sensitive human populations, another factor of 10 for both uncertainty in

the interspecies extrapolation using dosimetric adjustments and to account for the incompleteness of the data base, and a third factor of 10 to account for subchronic to chronic extrapolation. The MF of 1 is the default and is based upon an assessment of the scientific uncertainties of the toxicological data base not treated with the UF. The final number arrived at for the RfC is 9×10^{-3} mg/m^3 per day over a lifetime.

8.8.3 Reproductive and Developmental Effects

No inhalation studies for reproductive or developmental effects have been performed. In all the *in vivo* studies cited below, acetaldehyde is administered by the oral, intravenous, or intraperitoneal route.

Ali and Persaud (1988) studied the role of acetaldehyde in the pathogenesis of ethanol-induced developmental effects. Sprague-Dawley rats received intraperitoneal injections of a 1% solution of acetaldehyde at a dose of 100 mg/kg/day from days 9 through 12 of gestation. On day 12, the embryos were recovered and examined for
morphological abnormalities and crown-rump and head length. Acetaldehyde produced a significant reduction in head length, but had no significant effect on morphological abnormalities or crown-rump length. The reduction in head length was considered to be important, since it may be a causative factor in the microencephaly and CNS dysfunction found in fetal alcohol syndrome.

Kalmus and Buckenmaier (1989) investigated the effects of acetaldehyde on cultured preimplantation 2-cell stage mouse embryos *in vitro*. Embryos were exposed to 0, 5, 10, 200, or 500 mg acetaldehyde/100 ml culture medium for 60 minutes. Embryo growth was evaluated at a time period corresponding to an embryo age of 105 hours. No effects were observed at 5 and 10 mg/100ml; exposures to 50mg/100ml and higher were lethal. The results indicate that the 2-cell stage embryos are highly resistant to high *in vitro* dosages of acetaldehyde; however, the reason for the apparent resistance is not known.

Zorzano and Herrera (1989) studied the pattern of acetaldehyde appearance in maternal and fetal blood, maternal and fetal liver and placenta after oral ethanol administration or intravenous acetaldehyde administration (10mg/kg) to pregnant Wistar rats. The study demonstrated that acetaldehyde was able to cross the placental barrier at high concentrations; maternal blood concentration had to be greater that 80 µM. The fetal oxidation capacity in liver and placenta was shown to be lower than that of the maternal liver. A threshold above which the removal capacity of acetaldehyde metabolism by the fetoplacental unit would be surpassed was estimated to be 80 µM (maternal blood concentration) in the 21-day pregnant rat and possibly lower at early pregnancy when aldehyde dehydrogenase is absent from fetal liver.

Lahdetie (1988) is the only study available on the *in vivo* effects of acetaldehyde on the male reproductive system. Groups of hybrid male mice were given intraperitoneal injections of saline solution 0, 62.5, 125, or 250 mg acetaldehyde/kg daily for 5 days. No significant effects on sperm were seen for sperm count, sperm morphology, testes weight, or seminal vesicle weight when compared with controls. The authors speculated that, since no significant effects on sperm were seen, although acetaldehyde had been shown to produce mutagenic effects in somatic cells, germ cells were either less sensitive to the genotoxic effects or the acetaldehyde concentrations was too low because of its binding to erythrocytes and limited passage through the blood-testes barrier.

In female, pregnant rats, across several studies (Sreenathan et al., 1982, 1984a,b; Sreenathan and Padmanabhan, 1984; Padmanabhan et al., 1983; Checiu et al, 1984; Barilyak and Kozachuk, 1983; Dreosti et al., 1981), many of the same effects were observed. These include increased fetal resorption, increases in litter malformations, retardation in fetal growth, decreased placental weight, increased placental lesions, decreases in skeletal formation, delayed segmentation and differentiation of the embryo, increased cell fragmentation of the embryo, increased chromosomal abnormalities, and interference in thymidine incorporation into the DNA of the brain and liver.

In female, pregnant mice (Blakley and Scott, 1984a,b; Bannigan and Burke, 1982; Webster et al., 1983; O'Shea and Kaufman, 1979, 1981), there are several studies used to demonstrate reproductive and developmental effects, with most results leading to much uncertainty or doubt as to their advantage to understanding these effects. Several studies found no effects, whereas, some found an increase in fetal resorptions, fetal growth retardation, and increased number of fetuses with malformations. Most of these malformations were neural tube defects.

There are additional data that support the hypothesis that acetaldehyde interferes with placental function. In a series of studies (Henderson et al., 1981, 1982; Asai et al., 1985; Fisher et al., 1981a,b; 1984), the ability of acetaldehyde to interfere with amino acid uptake across the placenta was demonstrated. This demonstrates that this disruption in placental function may create a state of fetal malnutrition that is independent of maternal nutritional status. Such a state may be a factor in intrauterine growth retardation. These studies must be interpreted with some caution. These studies examined the status of term placentas and it remains to be determined what relationship this has to preplacental structures.

There are also several studies (Thompson and Folb, 1982; Higuchi and Matsumoto, 1984; Campbell and Fantel, 1983; Popov et al., 1982; Prescott, 1985) that have examined the direct embryotoxic properties of acetaldehyde utilizing whole embryo cultures (rat and mouse). The majority of these data demonstrate

that acetaldehyde can produce growth retardation and malformations *in vitro*.

The primary support for acetaldehyde-induced reproductive dysfunction is derived from *in vitro* studies examining the influence of acetaldehyde on androgen (male hormone) production. The majority of these studies (Cobb et al., 1978, 1980; Boyden et al., 1981; Badr et al., 1977; Cicero et al., 1980a,b; Santucci et al., 1983; Johnson et al., 1981; Cicero and Bell, 1980) have demonstrated that acetaldehyde significantly depresses HCG-(human chorionic gonadotrophin) stimulated testosterone production; however, the exact mechanism is unknown. This effect has been reported in a number of species, including mice, rats, and dogs.

Only one study has examined the reproductive effects of acetaldehyde aside from endocrine influences. Anderson et al. (1982) assessed the effects of acetaldehyde on sperm capacitation. These authors demonstrated that acetaldehyde did not alter the *in vitro* fertilizing capacity of mouse spermatozoa, though the relevance of this culture system to *in vivo* fertilization is unclear.

In vitro data strongly suggest the possibility of male reproductive toxicity and support the need for such data to be generated in *in vivo* systems.

8.10 References for Chapter 8

Ali, F. and T. Persaud. 1988. Mechanisms of fetal alcohol effects: role of acetaldehyde. Exp. Pathol. 33: 17-21.

American Conference of Governmental Industrial Hygienists. 1980. Documentation of the threshold limit values. Fourth edition: 1980. Cincinnati, OH: American Conference of Governmental Industrial Hygienists; pp. 3-4.

American Conference of Governmental Industrial Hygienists. 1985. TLVs: threshold limit values and biological exposure indices for 1985-86. Cincinnati, OH: American Conference of Governmental Industrial Hygienists; pp. 3-4.

Anderson, R.A. J.M. Reddy, C. Joyce, B.R. Willis, H. Van der Vend, and L.J. Zaneveld. 1982. Inhibition of mouse sperm capacitation by ethanol. Biol. Reprod. 27: 833-840.

Appelman, L.M., R.A. Woutersen, and V.J. Feron. 1982. Inhalation toxicity of acetaldehyde in rats. I. Acute and subacute studies. Toxicology. 23: 293-307.

Appelman, L.M., R.A. Woutersen, V.J. Feron R.N. Hooftman, and W.R.F. Notten. 1986. Effect of variable versus fixed exposure levels on the toxicity of acetaldehyde in rats. J. Appl. Toxicol. 6(5): 331-336.

Asai, M., O. Narita, and S. Kashiwamatu. 1985. Effect of acetaldehyde and/or ethanol on neutral amino acid transport systems in microvillous brush border membrane vesicles prepared from human placenta. Experientia 41: 1566-1568.

Badr, F.M., A. Bartke, S. Dalterio, and W. Bulger. 1977. Suppression of testosterone production by ethyl alcohol. Possible mode of action. Steroids 30: 647-655.

Bandas, E.L. 1982. Study of the role of metabolites and impurities in the mutagenic action of ethanol on yeast mitochondria. Genetika 18: 1056-1061.

Bannigan, J., and P. Burke. 1982. Ethanol teratogenicity in mice: a light microscope study. Teratol. 26: 247-254.

Barilyak, I.R. and S.Y. Kozachuk. 1983. Embryotoxic and mutagenic activity of ethanol after intra-amniotic injection. Tsitologiya i Genetika 17: 57-60.

Barrow, C.S. 1982. Sensory irritation of inhaled aldehydes: structure activity studies. CIIT 2, no. 9.

Bird, R.P., H.H. Draper, and P.K. Basrur. 1982. Effect of malonaldehyde and acetaldehyde on cultured mammalian cells; production of micronuclei and chromosomal aberrations. Mutat. Res. 101: 237-246.

Bittersohl, G. 1974. Epidemiologic investigations on cancer incidence in workers contacted by acetaldol and other aliphatic aldehydes. Arch. Geschwulstforsch. 43: 172-176.

Blakley, P.M. and W.J. Scott. 1984a. Determination of the proximate teratogen of the mouse fetal alcohol syndrome. 1. Teratogenicity of ethanol and acetaldehyde. Tox. Appl. Pharmacol. 72: 355-363.

Blakley, P.M. and W.J. Scott. 1984b. Determination of the proximate teratogen of the mouse fetal alcohol syndrome. 2. Pharmacokinetics of the placental transfer of ethanol and acetaldehyde. Tox. Appl. Pharmacol. 72: 364-371.

Boettner, E.A., G.L. Ball, and B. Weise. 1973. Combustion products from the incineration of plastics. Ann Arbor, MI: University of Michigan, School of Public Health. Available from: NTIS, Springfield, VA; PB-222001/0.

Böhlke, J.U., S. Singh, and H.W. Goedde. 1983. Cytogenetic effects of acetaldehyde in lymphocytes of Germans and Japanese: SCE, clastogenic activity, and cell cycle delay. Hum. Genet. 63: 285-289.

Boyden, T.W., M.A. Silvery, and R.W. Pamenter. 1981. Acetaldehyde acutely impairs canine testicular testosterone secretion. Eur. J. Pharmacol. 70: 571-576.

Bradley, M., I.C. Hsu, and C.C. Harris. 1979. Relationships between sister chromatid exchange and mutagenicity, toxicity, and DNA damage. Nature 282: 318-320.

Braven, J., G.J. Bonker, M.L. Fenner, and B.L. Tonge. 1967. The mechanism of carcinogenesis by tobacco smoke. Some experimental observations and a hypothesis. Br. J. Cancer 21: 623-633.

Campbell, M.A., and A.G. Fantel. 1983. Teratogenicity of acetaldehyde *in vitro*: relevance to fetal alcohol syndrome. Life Sci. 32: 2641-2647.

CARB. 1992a. Preliminary Draft: Proposed identification of acetaldehyde as a toxic air contaminant. Part A Exposure assessment. California Air Resources Board, Stationary Source Division. August, 1992.

CARB. 1992b. Preliminary Draft: Proposed identification of acetaldehyde as a toxic air contaminant. Part B Health assessment. California Air Resources Board, Stationary Source Division. August, 1992.

Checiu, M. S. Sandor, and Z. Garban. 1984. The effect of ethanol on early development in mice and rats. VI. In vitro effect of acetaldehyde upon preimplantation stages in the rat. Morphol. Embryol. (Bucur) (Romania) 30: 175-184.

Cicero, T.J., and R.D. Bell. 1980. Effects of ethanol and acetaldehyde on the biosynthesis of testosterone in the rodent testes. Biochem. Biophys. Res. Commun. 94: 814-819.

Cicero, T.J., R.D. Bell, and T.M. Badger. 1980a. Acetaldehyde directly inhibits the conversion of androstenedione to testosterone in the testes. Adv. Exp. Med. Biol. 132: 211-217.

Cicero, T.J., R.D. Bell, E.R. Meyer, and T.M. Badger. 1980b. Ethanol and acetaldehyde directly inhibit testicular steroidogenesis. J. Pharmacol. Exp. Therap. 213: 228-233.

Cobb, C.F., M.F. Ennis, F. Michael, D.H. Van Thiel, J.S. Gavaler, and R. Lester. 1978. Acetaldehyde and ethanol are direct testicular toxins. Surg. Forum 29: 641-644.

Cobb, C.F., M.F. Ennis, D.H. Van Thiel, J.S. Gavaler, and R. Lester. 1980. Isolated testes perfusion: a method using a cell- and protein-free perfusate useful for the evaluation of potential drug injury. Metabolism 29:71-79.

Commoner, B. 1976. Reliability of bacterial mutagenesis techniques to distinguish carcinogenic chemicals. EPA-600/1-76-022. U.S. Environmental Protection Agency.

Delaney J.L. and T.W. Hughs. 1979. Source assessment: manufacture of acetone and phenol from cumene. Research Triangle Park, NC: U.S. Environmental Protection Agency, Industrial Environmental Research Laboratory: EPA Report No. EPA-600/2-79-019d.

de Raat, W.K., P.B. Davis, and G.L. Bakker. 1983. Induction of sister-chromatid exchanges by alcohol and alcoholic beverages after metabolic activation by rat-liver homogenate. Mutat. Res. 167: 89-105.

Dreisbach, R.H. 1980. Acetaldehyde, metaldehyde, and paraldehyde. In: Handbook of poisoning: prevention, diagnosis, and treatment. Los Angeles, CA: Lange Medical Publications; pp. 177-180.

Dreosti, I.E., F.J. Ballard, G.B. Belling, I.R. Record, S.J. Manuel, and B.S. Hetzel. 1981. The effect of ethanol and acetaldehyde an DNA synthesis in growing cells and on fetal development in the rat. Alcohol: Clin. Exp. Res. 5: 357-362.

Dulout F. and T. Furnus. 1988. Acetaldehyde induced aneuploidy in cultured Chinese hamster cells. Mutagenesis 3: 207-211.

Egle, J.L., Jr. 1972. Effects of inhaled acetaldehyde and propionaldehyde on blood pressure and heart rate. Toxicol, Appl. Pharmacol. 23: 131-135.

Egle, J.L., P.M. Hudgins, and F.M. Lai. 1973. Cardiovascular effects of intravenous acetaldehyde and propionaldehyde in the anesthetized rat. Toxicol. Appl. Pharmacol. 24: 636-644.

Eimutis, E.C., R.P. Quill, and G.M. Rinaldi. 1978. Source assessment: noncriteria pollutant emissions. Research Triangle Park, NC: U.S. Environmental Protection Agency, Industrial Environmental Research Laboratory; EPA Report No. EPA-600/2-78-004t.

Eker, P. and T. Sanner. 1986. Initiation of in vitro cell transformation by formaldehyde and acetaldehyde as measured by attachment-independent survival of cells in aggregates. Eur. J. Cancer Clin. Oncol. 22(6): 671-676.

EPA. 1987. Health assessment document for acetaldehyde. Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA-600/8-86/015A. (External Review Draft).

EPA. 1992. Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

Fenner, M.L. and J. Braven. 1968. The mechanism of carcinogenesis by tobacco smoke. Further experimental evidence and a prediction from the thiol-defense hypothesis. Br. J. Cancer 22: 474-479.

Feron, V.J. 1979. Effects of exposure to acetaldehyde in Syrian hamsters simultaneously treated with benzo(a)pyrene or diethylnitrosamine. Prog. Exp. Tumor Res. 24: 162-176.

Feron, V.J., A. Kruysse, and R.A. Woutersen. 1982. Respiratory tract tumors in hamsters exposed to acetaldehyde vapour alone or simultaneously to benzo(a)pyrene or diethylnitrosamine. Eur. J. Cancer Clin. Oncol. 18: 13-31.

Fisher, S.E., M. Atkinson, I. Holzman, R. David, and D.H. Van Thiel. 1981a. Effects of ethanol upon placental uptake of amino acids. Prog. Biochem. Pharmacol. 18: 216-223.

Fisher, S.E., M. Atkinson, D.H. Van Thiel, E. Rosenblum, R. David, and I. Holzman. 1981b. Selective fetal malnutrition: effects of ethanol and acetaldehyde upon *in vitro* uptake of alpha amino isobutyric acid by human placenta. Life Sci. 29: 1283-1288.

Fisher, S.E., M. Atkinson, and D.H. Van Thiel. 1984. Selective fetal malnutrition: the effect of nicotine, ethanol, and acetaldehyde upon *in vitro* uptake of a alpha-aminoisobutyric acid by human placenta. Dev. Pharmacol. Ther. 7: 229-238.

Garberg, P., E-L Akerblom, and G. Bolcsfoldi. 1988. Evaluation of a genotoxicity test measuring DNA strand breaks in mouse

lymphoma cells by alkaline unwinding and hydroxyapatite elution. Mutat. Res 203: 155-176.

Greenwald, I.S., and H.R. Horvitz. 1980. unc-93 (e1500): a behavioral mutant of *Caenorhabditis elegans* that defines a gene with a wild-type null phenotype. Genetics 96: 147-164.

He, S.M. and B. Lambert. 1985. Induction and persistence of SCE-inducing damage in human lymphocytes exposed to vinyl acetate and acetaldehyde *in vitro*. Mutat. Res. 158: 201-208.

Hemminki, K., K. Falck, H. Vainio. 1980. Comparison of alkylation rates and mutagenicity of directly acting industrial and laboratory chemicals. Arch. Toxicol. 46: 277-285.

Henderson G.I., D. Turner, R.V. Patwardhan, L. Lumeng, A.M. Hoyumpa, and S. Schenker. 1981. Inhibition of placental valine uptake after acute and chronic maternal ethanol consumption. J. Pharmacol. Exper. Ther. 216: 465-472.

Henderson G.I., R.V. Patwardhan, S. McLeroy, and S. Schenker. 1982. Inhibition of placental amino acid uptake in rats following acute and chronic ethanol exposure. Alcoholism 6: 495-500.

Higuchi, Y., and N. Matsumoto. 1984. Embryotoxicity of ethanol and acetaldehyde: direct effects on mouse embryo *in vitro*. Congen. Anom. (Senten IJO) 24: 9-28.

Hilaro et al. 1985.

IARC. 1987. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Supplement 7. Overall Evaluations of Carcinogenicity: An updating of IARC monographs volumes 1 to 42. International Agency for Research on Cancer. World Health Organization, Lyon, France.

Jacob, D. J., E. W. Gottlieb, and M. J. Prather. 1989. Chemistry of a polluted boundary layer. J. Geophys. Res., 94:12975-13002.

Jansson, T. 1982. The frequency of sister chromatid exchanges in human lymphocytes treated with ethanol and acetaldehyde. Hereditas 97: 301-303.

Johnson, D.E., Y.-B. Chiao, J.S. Gavaler, and D.H. Van Thiel. 1981. Inhibition of testosterone synthesis by ethanol and acetaldehyde. Biochem. Pharmacol. 36: 1827-1831.

Kalmus G. and C. Buckenmaier. 1989. Effects of ethanol and acetaldehyde on cultured pre-implantation mouse embryos. Experientia 45: 484-487.

Kane, L.E., R. Dombroske, and Y. Alarie. 1980. Evaluation of sensory irritation from some common industrial solvents. Am. Ind. Hyg. Assoc. J. 41: 451-455.

Korte, A. and G. Obe. 1981. Influence of chronic ethanol uptake and acute acetaldehyde treatment on the chromosomes of bone-marrow cells and peripheral lymphocytes of Chinese hamsters. Mutat. Res. 88: 389-395.

Kruysse, A., V.J. Feron, and H.P. Til. 1975. Repeated exposure to acetaldehyde Vapor. Arch. Environ. Health. 30: 449-452.

Lahdetie, J. 1988. Effects of vinyl acetate and acetaldehyde on sperm morphology and meiotic micronuclei in mice. Mutat. Res. 202: 171-178.

Lam, C.W., M. Casanova, and H. D'A. Heck. 1986. Decreased extractability of DNA from proteins in the rat nasal mucosa after acetaldehyde exposure. Fund. Appl. Toxicol. 6: 541-550.

Lambert, B., Y. Chen, S.M. He, and M. Sten. 1985. DNA cross-links in human leucocytes treated with vinyl acetate and acetaldehyde *in vitro*. Mutat. Res. 146: 301-303.

Laumbach, A.D., S. Lee, J. Wong, U.N. Streips. 1976. Studies on the mutagenicity of vinyl chloride metabolites and related chemicals. Proceedings of the 3rd International Symposium on the Prevention and Detection of Cancer, Vol. 1, pp. 155-169.

Lewis. R.J., and R.L. Tatkin. 1983. Registry of toxic effects of chemical substances. 1981-1982 ed. Cincinnati, OH: U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health; p. 156; DHHS (NIOSH) publ. no. 83-107.

Ligocki, M.P., R.R. Schulhof, R.E. Jackson, M.M. Jimenez, G. Z. Whitten, G.M. Wilson, T.C. Meyers, and J.L. Fieber. 1992. Modeling the effects of reformulated gasolines on ozone and toxics concentrations in the Baltimore and Houston areas. Systems Applications International, San Rafael, California (SYSAPP-92/127).

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).

Mannsville Chemical Products Corporation. 1984. Chemical products synopsis. Acetic acid. Cortland, NY: Mannsville Chemical Products Corporation.

Marnett, L.J., H.K. Hurd, M.C. Hollstein, D.E. Levin, H. Esterbauer, and B.N. Ames. 1985. Naturally occurring carbonyl compounds are mutagenic in *Salmonella* tester strain TA104. Mutat. Res. 148: 381-382.

Mohan, M., U. Rai, L.P. Reddy, C.V. Prasanna, and S. Ramakrishnan. 1981. Cardiovascular effects of acetaldehyde in guinea pigs. Indian J. Physiol. Pharmacol. 25: 246.

Mortelmans, K. S. Haworth, T. Lawlor, W. Speck, B. Tainer, and E. Zeiger. 1986. *Salmonella* mutagenicity tests: II. Results from testing of 270 chemicals. Environ. Mutagen. 8: 1-39.

Norppa, H., F. Tursi, P. Pfäffli, J. Mäki-Paakkanen, and H. Järventaus. 1985. Chromosome damage induced by vinyl acetate through *in vitro* formation of acetaldehyde in human lymphocytes and Chinese hamster ovary cells. Cancer Res. 45: 4816-4821.

National Research Council. 1977. Drinking water and health: part II, chapters 6 and 7. Washington, D.C.: National Academy of Sciences; pp. VI-219-VI-220. Available from: NTIS, Springfield, VA; PB-270423.

National Research Council. 1981. Formaldehyde and other aldehydes. Washington, D.C.: National Academy Press.

Obe, G. and B. Beer. 1979. Mutagenicity of aldehydes. Drug and Alcohol Dependence 4: 91-94.

Obe, G. and H. Ristow. 1977. Acetaldehyde, but no ethanol, induces sister chromatid exchanges in Chinese hamster cells *in vitro*. Mutat. Res. 56: 211-213.

Obe, G., R. Jonas, and S. Schmidt. 1986. Metabolism of ethanol *in vitro* produces a compound which induces sister-chromatid exchanges in human peripheral lymphocytes *in vitro*: acetaldehyde, not ethanol, is mutagenic. Mutat. Res. 174: 47-51.

Obe, G., A.T. Natarajan, M. Meyers, and A. Den Hertog. 1979. Induction of chromosomal aberrations in peripheral lymphocytes of human blood *in vitro*, and of SCEs in bone-marrow cells of mice *in vitro* by ethanol and its metabolite acetaldehyde. Mutat. Res. 68: 291-294.

Olson, T.M. and M.R. Hoffmann, 1989. Hydroxyalkylsulfonate formation: its role as a S(IV) reservoir in atmospheric water droplets. Atmos. Environ., 23: 985-997.

Omel'yantes, N.I., N.V. Mironets, N.V. Martyshchenko, I.A. Gubareva, L.F. Piven, and S.N. Starchenko. 1978. Experimental substantiation of the maximum permissible concentrations of acetone and acetaldehyde in reclaimed potable water. Biol. Aviakosm Med. 1978: 67.

O'Shea, K.S. and M.H. Kaufman. 1979. Teratogenic effects of acetaldehyde: implications for the study of fetal alcohol syndrome. J. Anat. 128: 65-76.

O'Shea, K.S. and M.H. Kaufman. 1981. Effects of acetaldehyde on neuroepithelium of early mouse embryos. J. Anat. 132: 107-118.

Padmanabhan, R., R.N. Sreenathan, and S. Singh. 1983. Studies of the lethal and teratogenic effects of acetaldehyde in the rat. Conge. Anom. (Senten IJO) 23: 13-23.

Perry, R.H. and Chilton, C.H. 1973. Chemical Engineer's Handbook, Fifth Edition. McGraw-Hill Book Company.

Pool, B.L., and M. Wiessler. 1981. Investigations on the mutagenicity of primary and secondary α-acetoxynitrosamines with *Salmonella typhimurium*: activation and deactivation of structurally related compounds by S-9. Carcinogenesis 2: 991-997.

Popov, V.B., B.L. Vaisman, V.P. Puchkov, and T.V. Ignat'eva. 1982. Toxic effects of ethanol and its biotransformation products on post-implantation embryos in culture. Bull. Exp. Biol. Med. (Rus.) 92: 1707-1710.

Prasanna, C.V., S. Ramakrishnan, B. Krishnan, and A. Srinivasa Rao. 1981. Effect of acetaldehyde on lung surfactant. Indian J. Exp. Biol. 19: 580-581.

Prescott, P.K. 1985. The effect of acetaldehyde and 2,3-butanediol on rat embryos developing *in vitro*. Biochem. Pharmacol. 34: 529-532.

Rieger, R., and A. Michaelis. 1960. Chromosomenaberrationen nach Einwirkung von Acetaldehyd auf Primawurzeln von *Vicia faba*. Biol. Zbl. 79: 1-5.

Ristow, H. and G. Obe. 1979. Acetaldehyde induces cross-links in DNA and causes sister-chromatid exchanges in human cells. Mutat. Res. 58: 115-119.

Saladino, A.J., J.C. Willey, J.F. Lechner, R.C. Grafstrom, M. LaVeck, and C.C. Harris 1985. Effects of formaldehyde, acetaldehyde, benzoyl peroxide, and hydrogen peroxide on cultured normal human bronchial epithelial cells. Cancer Res. 45: 2522-2526.

Santucci, L., T.J. Graham, and D.H. Van Thiel. 1983. Inhibition of testosterone production by rat Leydig cells with ethanol and acetaldehyde: prevention of ethanol toxicity with 4-methylpyrazole. Alcoholism 7: 135-139.

Shikiya, D.C., C.S. Liu, M.I. Kahn, J. Juarros, and W. Barcikowski. 1989. In-vehicle air toxics characterization study in the south coast air basin. South Coast Air Quality Management District, El Monte, CA. May, 1989.

Sim, V.M., and R.E. Prattle. 1957. Effect of possible smog irritants on human subjects. JAMA J. Am. Med. Assoc. 165: 1908-1913.

Sina, J.F., C.L. Bean, G.R. Dysart, V.I. Taylor, and M.O. Bradley. 1983. Evaluation of the alkaline elution/rat hepatocyte assay as a predictor of carcinogenic/mutagenic potential. Mutat. Res. 113: 357-391.

Sittig, M. 1979. Haxardous and toxic effects of industrial chemicals. Park Ridge, NJ: Noyes Data Corporation; pp. 1-2.

Skog, E. 1950. A toxicological investigation of lower aliphatic aldehydes. I. toxicity of formaldehyde, acetaldehyde, propionaldehyde butyraldehyde; as well as acrolein and crotonaldehyde. Acta Pharmacol. Toxicol. 6: 299-318.

Sreenathan, R.N. and R. Padmanabhan. 1984. Structural changes of placenta following maternal administration of acetaldehyde in the rat. Z. Mikrosk. Anat. Forsch. (E.Germ.) 98: 597-600.

Sreenathan, R.N., R. Padmanabhan, and S. Singh. 1982. Teratogenic effects of acetaldehyde in the rat. Drug Alcohol Depend. 9: 339-350.

Sreenathan, R.N., S. Singh, and R. Padmanabhan. 1984a. Implications of the placenta in the acetaldehyde-induced intra-uterine growth retardation. Drug Alcohol Depend. 13: 199-204.

Sreenathan, R.N., S. Singh, and R. Padmanabhan. 1984b. Effects of acetaldehyde on skeletogenesis in the rat. Drug Alcohol Depend. 14: 165-174.

Thompson, P.A.C., and P.I. Folb. 1982. An *in vitro* model of alcohol and acetaldehyde teratogenicity. J. Appl. Toxicol. 2: 190-195.

Veghelyi, P.V., M. Osztovics, G. Kardos, L. Leisztner, E. Szaszovszky, S. Igali, and J. Imrei. 1978. The fetal alcohol syndrome: symptoms and pathogenesis. Acta Paediatria Academiae Scientiarum Hungaricae 19: 171-189.

Versar Inc. 1975. Identification of Organic Compounds in Effluents From Industrial Sources.

Watanabe, F. and S. Sugimoto. 1956. Study on the carcinogenicity of aldehyde, 3rd report: four cases of sarcomas of rats appearing in the areas of repeated subcutaneous injections of acetaldehyde. Gann 47: 599-601.

Webster, W.S., D.A. Walsh, S.E. McEwen, and A.H. Lipson. 1983. Some teratogenic properties of ethanol and acetaldehyde in C57BL/6J mice; implications for the study of fetal alcohol syndrome. Teratol. 27: 231-243.

Wilson, A. et al. 1991. Air toxics microenvironment exposure and monitoring study. South Coast Air Quality Management District, El Monte, CA.

Windholz, M., S. Budavari, R.F. Blumetti, and E.S. Otterbein, eds. 1983. Acetaldehyde. In: The Merck Index: an encyclopedia of chemicals, drugs, and biologicals. 10th ed. Rahway, NJ: Merck and Co., Inc.; p. 30.

Woodruff, R.C., J.M. Mason, R. Valencia, and S Zimering. 1985. Chemical mutagenesis testing in *Drosophila*. V. Results of 53 coded compounds tested for the National Toxicology Program. Environ. Mutagen. 7: 677-702.

Woutersen, R.A., and Appelman L.M. 1984. Lifespan inhalation carcinogenicity study of acetaldehyde in rats. III. Recovery after 52 weeks of exposure. Report No. V84.288/190172. CIVO-Institutes TNO, The Netherlands.

Woutersen, R.A. Van Garderen-Hoetmer, A., and Appelman, L.M. 1985. Lifespan (27 months) inflation carcinogenicity study of acetaldehyde in rats. Report No. V85.145/190172. CIVO-Institutes TNO, The Netherlands.

Woutersen, R.A., L.M. Appelman, A. Van Garderen-Hoetmer, and V.J. Feron. 1986. Inhalation toxicity of Acetaldehyde in rats. III. Carcinogenicity study. Toxicology. 41: 213-231.

Woutersen, R.A., and V.J. Feron. 1987. Inhalation toxicity of acetaldehyde in rats. IV. Progression and regression of nasal lesions after discontinuation of exposure. Toxicology. 47: 295-305.

Zorzano, A. and E, Herrera. 1989. Disposition of ethanol and acetaldehyde in late pregnant rats and their fetuses. Pediat. Res. 25: 102-106.

9.0 DIESEL PARTICULATE MATTER

9.1 Chemical and Physical Properties

Diesel exhaust particulate matter consists of a solid core composed mainly of carbon, a soluble organic fraction, sulfates, and trace elements. When comparing the size distribution of diesel particles to gasoline particles the majority of the diesel particles range from 0.1 to 1.0 μm with a peak at around 0.15 μm, while the gasoline particles range from 0.01 to 0.1 μm with the peak at around 0.02 (NRC, 1982). When a particle is less than 1 micron (μm) in diameter it is small enough to be inhaled deeply into the lungs. Although the gasoline particles are smaller, the light-duty diesel engines emit from 30 to 100 times more particles than comparable catalyst-equipped gasoline vehicles (NRC, 1982). At temperatures above 500°C the particles themselves are actually solid chain aggregates of carbon-hydrogen spheres with diameters ranging from 100 to 800 angstroms (Å). These are mainly attributed to the incomplete combustion of fuel hydrocarbons, though some may be due to engine oil or other fuel components. Photomicrographs show that diesel particles have a very light, fluffy structure, with a density of about 0.07 g/m^3 (NRC, 1982).

At temperatures below 500°C, the particles become coated with adsorbed and condensed high molecular weight organic compounds. Typically, about 25 percent of the particle consists of extractable organics, although different vehicles may have extractable fractions of 5-90 percent, depending on operating conditions. These compounds include open-chain hydrocarbons of 14-35 carbon atoms, alkyl-substituted benzenes, and derivatives of the polycyclic aromatic hydrocarbons (PAH), such as ketones, carboxyaldehydes, acid anhydrides, hydroxy compounds, quinones, nitrates, and carboxylic acids (Johnson, 1988). There are also heterocyclic compounds containing sulfur, nitrogen, and oxygen atoms within the aromatic ring. Inorganic compounds also are present and include sulfur dioxide, nitrogen dioxide, and sulfuric acid (NRC, 1982).

To best describe the diesel particle content adequately, the temperature at the time of the sample collection and the means by which that temperature was reached must be specified. Diesel particulate matter is generally defined as any material that is collected, at a temperature of 52°C or less, on a filtering medium after dilution of the raw exhaust gases (NRC, 1982). Water that condenses on the filter is not considered to be diesel particulate matter.

9.2 Formation and Control Technology

The chemical mechanism which accounts for carbon formation by diesel engines is not completely established (EPA, 1990b); the major weight of scientific opinion seems to support some role for intermediate formation of polycyclic aromatic matter (POM) in the process. Carbon is a stable combustion product normally of rich

flames; carbon formation normally takes place over a rather narrow temperature range. A significant fraction of the diesel particulate matter consists of oil-derived hydrocarbons and related solid matter. The formation of the carbon particle is thought to involve polymerization of gaseous intermediates at the surface of the smaller particles. Growth and agglomeration of the carbon particles are probably gas-to-particle processes. The POM's that are produced in the combustion process are adsorbed onto the surface of the carbon particle. Several of these, such as benzo[a]pyrene (B[a]P) and 1-nitropyrene, are known or potential human carcinogens. Recent data have indicated that the particles themselves may have intrinsic toxic and carcinogenic properties.

Studies of diesel particle composition have produced some information about the fate of fuel sulfur (EPA, 1990b). Sulfate has been found to be a significant component of diesel particles. Generally, the sulfate found in particles accounts for only about 2% of the fuel sulfur, the balance being emitted as sulfur dioxide. At present, no means of reducing sulfate formation is available other than reducing the sulfur concentration of diesel fuel.

EPA's Five City Study (EPA, 1989) determined that POM contributed to 27% of the average excess aggregate cancer incidence in the five cities. Of this 27%, diesel particulate matter was the major contributor, accounting for 45% of the total POM.

The control of diesel emissions can take two forms. The first is controlling emissions before they are formed with either engine modifications or aftertreatment systems to the exhaust system. Each of these takes many forms and are in various stages of development (EPA, 1990b).

One way to modify the engine is by refining the combustion process and many different modifications are in use. Many new diesel engines being made today are going from the indirect to the direct injection engine. These direct injection engines are low-emitting and fuel efficient. Also being considered are changes to the combustion chamber design to decrease emissions.

At this time, most diesel engines still rely on mechanical engine control systems. On newer engines, there is now expanded use of computerized electronic control systems that increase the potential flexibility in controlling emissions.

Another technology used to control emissions is the combined technology of turbocharging and intercooling. Most heavy duty diesel engines have them and were required for virtually all engines in 1991. The turbocharger increases the air mass in the cylinder and the intercooler reduces the temperature. This system is successful in reducing both NO_x and diesel particle emissions as well as increasing fuel economy and power output. Also being considered for use on some heavy duty engines is

intake manifold tuning. At this time, it is being used on high-performance cars to enhance the airflow.

Control of the lubricating oil is important to diesel particulate matter reduction since 10 to 50% of the particulates being formed are from engine oil. Oil consumption can be reduced primarily by improved engine manufacturing specifications and engine seals.

A second way to control emissions is to add aftertreatment technologies to the exhaust system. A trap oxidizer is being considered. This is located in the exhaust system to trap the particulate matter and provide some means of cleaning the filter by burning the collected particulate matter. Passive system traps, traps that attain the proper conditions for regeneration during normal operation, require the use of a catalyst in most cases. Some catalysts being considered are platinum, palladium, rhodium, silver, vanadium, and copper. Cerium has also been considered as a fuel additive to be used with the catalyst. There is still development to be done in this area.

Catalytic convertors are another technology being evaluated along with fuel modifications. By reducing the sulfur (now at a maximum of 0.05% by weight) and aromatic hydrocarbon content, emissions of diesel particles and POM can be reduced. Fuel additives are also being tested.

Alternative fuels are being researched for use in diesel engines. The fuels being tested at this time are natural gas, methanol, and liquified petroleum gas.

9.3 Emissions

9.3.1 Diesel Particulate Matter Emission Standards

Diesel particulate matter emission standards for light duty vehicles (LDVs), light duty trucks (LDTs), and HDDEs are summarized in Table 9-1. The LDV and LDT categories include both gasoline and diesel powered vehicles.

9.3.2 Methodology

As mentioned in section 3.2, the urban diesel particulate matter national fleet average emission factors derived by Sienicki (1992a, 1992b) are used for this analysis. The general methodology used by Sienicki to calculate urban diesel particulate matter is summarized in the appendix of MVMA and EMA (1986). All input data

Table 9-1. Diesel Particulate Matter Emission Standards.

Year	LDV[a] (gpm)	LDT1[bc] (gpm)	LDT2[bc] (gpm)	LDT3[bc] (gpm)	LDT4[bc] (gpm)	HDDE Urban bus (g/bhp-hr)	HDDE Other diesels (g/bhp-hr)
1982–1986	0.60	0.60	0.60	0.60	0.60	None	None
1987	0.20	0.26	0.26	0.26	0.26	None	None
1988–1990	0.20	0.26	0.26	0.26	0.26	0.60	0.60
1991	0.20	0.26	0.26	0.26	0.26	0.25	0.25
1992	0.20	0.26	0.26	0.26	0.26	0.25	0.25
1993	0.20	0.26	0.26	0.26	0.26	0.10	0.25
1994	0.08/0.10[d]	0.26	0.26	0.10	0.12	0.05	0.10
1995+	0.08/0.10[d]	0.08/0.10[d]	0.08/0.10[d]	0.10	0.12	0.05	0.10

[a]1994 standards are phased in over three years: 40% MY 1994, 80% MY 1995, 100% MY 1996 and after.

[b]1995 standards are phased in over three years: 40% MY 1995, 80% MY 1996, 100% MY 1997 and after.

[c]Light light-duty trucks consist of weight categories LDT1 and LDT2, and are less than or equal to 6000 lbs. gross vehicle weight rating (GVWR). Heavy light-duty trucks consist of weight categories LDT3 and LDT4, and are greater than 6000 lbs GVWR. LDT1 = light light-duty trucks up to 3750 lbs loaded vehicle weight (LVW). LDT2 = light light-duty trucks greater than 3750 lb LVW. LDT3 = heavy light-duty trucks up through 5750 lbs adjusted loaded vehicle weight (ALVW). LDT4 = heavy light-duty trucks greater than 5750 lbs ALVW. LVW = curb weight (nominal vehicle weight) plus 300 lbs. ALVW = numerical average of curb weight and GVWR.

[d]The first number is the 5 year/50,000 mile standard; the second number is the 10 year/100,000 mile standard.

and values calculated in each step of Sienicki's analysis are contained in Appendix G.

Urban diesel particulate matter emissions can simply be considered as the product of urban diesel vehicle miles travelled and the diesel particle emission rate:

$$(1) \quad DP = UVMT_d \times ER$$

where:
DP = Urban Diesel Particulate Matter Emissions (g)
$UVMT_d$ = Urban Diesel Vehicle Miles Travelled (mi)
ER = Diesel Particulate Matter Emission Rate (g/mi).

DP is calculated separately for each vehicle class by model year for the 20 most recent model years; these values are then added to obtain overall urban diesel particulate matter for the year of interest. However, to calculate $UVMT_d$ and ER for individual classes in a model year, a series of steps, which will be detailed in the following sections, must first be employed.

An overall national fleet emission factor (EF) in grams per mile can be calculated by dividing the total DP, after applying a freeway road use adjustment (described later), by total UVMT for both gas and diesel vehicles:

$$2) \quad EF = Total\ DP/Total\ UVMT$$

9.3.2.1 Calculation of Urban Diesel Vehicle Miles Travelled

$UVMT_d$ is determined as the product of fleet VMT, diesel mile fraction (DMF), and the diesel urban fraction (DUF):

$$(3)\ UVMT_d = VMT \times DMF \times DUF.$$

The DMF is the ratio of diesel miles travelled divided by total miles travelled. It can be calculated using the following equation:

$$(4)\ DMF = DSF/\{(DSF + (1 - DSF)(VMT_g/VMT_d)\}$$

where:
DSF = Diesel Sales Fraction
VMT_g = gasoline annual vehicle miles travelled
VMT_d = diesel annual vehicle miles travelled.

DSF is obtained by dividing diesel market shares by 100 (listed for each vehicle class by model year in Appendix G). Sienicki based the diesel market shares he used on industry opinion. The industry opinion he used predicts lower LDDV and LDDT sales than MOBILE4 (EPA, 1988). Sienicki used several sources to obtain his gasoline and diesel annual VMT rates. For all vehicle classes except buses, two sources were used -- the MVMA and EMA analysis (MVMA and EMA, 1986) and MOBILE4 (EPA, 1991a). The annual VMT rates for buses were based on data from the American Public Transport Association (APTA, 1990).

Sienicki calculated fleet VMT by model year in each class (VMT_y) as the product of the VMT fraction for each model year in a given class ($VMTf_y$) and the total VMT for each class (TVMT):

$$(5) \quad VMT_y = VMTf_y \times TVMT$$

$VMTf_y$ is calculated by multiplying vehicle sales per vehicle class for each model year (VEH_y) by annual VMT for gas and diesel vehicles (VMT_y) and the survival rate (SR_y), then dividing the product by the sum of products for all model years:

$$6) \quad VMTf_y = \frac{VEH_y \times VMT_y \times SR_y}{\sum_{y=1}^{20} (VEH_y \times VMT_y \times SR_y)} .$$

Vehicle sales per vehicle class were estimated by first establishing a base 20 year sales fleet from historical data, up to the year 1990. The sales fleet from the MVMA and EMA analysis (MVMA and EMA, 1986) was updated using data mostly from MVMA (1991). For estimation of diesel particulate matter in future years (1995, 2000, 2010), Sienicki had to estimate growth in the size of the sales fleet for each class (and hence, growth in VMT). This was done using fuel usage growth in the transportation sector as a surrogate. Fuel usage predictions were obtained from the Department of Energy (DOE, 1991). Survival rates for all vehicles except buses were obtained from MVMA and EMA (1986). Estimation of survival rates for buses are described in Sienicki and Mago (1991). TVMT is the product of vehicle sales per vehicle class for a 20 year period ending in the target year, annual VMT, and annual SR. Mathematically, it can essentially be expressed as the denominator in Equation 6.

Sienicki's final step in determining UVMT for each class in a given model year was to multiply VMT and DMF by the diesel urban fraction (DUF), the fraction of miles travelled in urban areas by diesel vehicles in a vehicle class. Sienicki used the same DUFs found in UMTRI (1988).

9.3.2.2 Calculation of Diesel Particulate Matter Emission Rate

ERs for all classes prior to 1987 were obtained from MVMA and EMA (1986). HDDE particulate matter emission rates for 1988 through 1991 were based on mean values for each class from federal certification test results. Model years 1992 and 1993 were assumed to be the same as 1991, and for 1994 and later years, emission rates were set at the design target for the 0.10 g/bhp-hr standard at 0.084 g/bhp-hr. LDDV and LDDT rates were set to EPA emission standards (the 10 year/100,000 mile standard for 1994 and later years). Bus emission rates were assumed to be the same as those for vehicles in class VIIIB until 1993, when

they were set to a design target of 0.084 g/bhp-hr. For 1994 and
later, they were assumed to be 0.06 g/bhp-hr.

Conversion factors (CFs) were used to convert heavy duty
emission rates from g/bhp-hr to grams per mile. CFs describe the
average work per mile required for each vehicle class, and can be
calculated as the ratio of fuel density to the product of brake
specific fuel consumption (BSFC) and fuel economy (MPG) (EPA,
1988; MVMA, 1983):

$$6) \quad CF = Fuel\ Density/(BSFC \times MPG)$$

Conversion factors predicted for future years must take into
account any improvements in vehicle efficiency. Although EPA's
MOBILE4 emissions model assumes no improvement in heavy duty
vehicle efficiency after 1986 (EPA, 1988), Sienicki claims that a
number of factors, such as increased market penetration of radial
tires and aerodynamic bodies, higher efficiency radial tires and
electronic emission control will continue to improve vehicle
efficiency. He developed efficiency improvement factors based on
fuel economy improvement and market penetration analysis prepared
by industry market analysts for each vehicle class. CFs for
future years were then calculated using previous years' CFs
divided by one plus the efficiency increase expected:

$$7) \quad CF_{y+1} = CF_y/(1+ \%\ efficiency\ increase/100)$$

EPA recently developed new conversion factors for heavy duty bus
engines (Kitchen and Damico, 1992) to more accurately reflect the
effect that different types of bus operations have on relative
levels of emissions of specific pollutants. These new heavy duty
bus conversion factors are not used in this analysis.

Sienicki also adjusted gram per mile emission rates for the
use of low sulfur fuel in 1991 and later years to obtain final
gram per mile emission rates for the 20 most recent model years
starting with the model year of interest. Sienicki assumed a
0.025 g/bhp-hr reduction in particulate matter resulting from a
0.10 weight percent change in fuel sulfur, based on results from
a recent study which addressed the effects of fuel composition on
diesel exhaust (Ullman, 1989). An earlier study (Ingham and
Warden, 1987) predicted particle reductions from fuel sulfur that
were in the same range, although slightly lower. Sienicki
assumed an average fuel sulfur level of 0.25 weight percent for
1990 and earlier years, reduced to a standard of 0.10 weight
percent in 1991-1993, and a standard of 0.05 weight percent in
1994 and later years (EPA, 1990a).

9.3.2.3 Calculation of Urban Diesel Particulate Matter Emissions

Once UMVT and ER have been calculated, DP for each model
year in a class can then be calculated using Equation 1. The sum
of DPs for the 20 most recent model years in all classes can be
combined to yield a total DP estimate for the year of interest.
After calculating a total DP for each year of interest, Sienicki
then applied a final adjustment to his total DP estimates to

account for freeway road use. While the heavy duty transient
emission test cycle assumes 25% freeway operation, a recent
University of Michigan study (1988) estimated that class VIIIB
vehicles accumulated 73% of their VMT on freeways when in large
urban areas. Sienicki adjusted for this discrepancy using the
following equation:

$$8) \quad DPAdj = DP \times [(1 - FMF) \times (4/3 - 1/3 \times FFR) + FMF \times FFR]$$

where: DPAdj = adjusted diesel particulate mass
                FMF = freeway mileage factor
                FFR = freeway factor ratio

FMF values were reported in the recent University of Michigan
study (1988), and FFR is the ratio of freeway and non-freeway
emission rates in grams per mile. A detailed explanation of this
equation, and the derivation of terms in this equation can be
found in Sienicki and Mago (1991).

9.3.2.4 Calculation of the Urban Diesel Particulate Matter
National Fleet Average Emission Factor

     The urban diesel particulate matter national fleet average
EF can be calculated using Equation 2. Total UVMT in Equation 2
is the sum of $UVMT_d$ and gasoline urban vehicle miles travelled
($UVMT_g$):

$$9) \quad Total\ UVMT = UVMT_g + UVMT_d$$

$UVMT_g$ is calculated using the following equation:

$$10) \quad UVMT_g = VMT \times (1 - DMF) \times GUF$$

where: GUF = gasoline urban fraction

Sienicki used the same GUFs found in MVMA and EMA (1986). Urban
diesel particulate matter national fleet average EFs for 1990,
1995, 2000, and 2010 are 0.0573, 0.0305, 0.0160, and 0.009
g/mile, respectively.

     Sienicki's fleet average EFs can be compared to EFs derived
from information in a previous EPA air toxics report (Carey,
1987). Projected 1995 national fleet average EFs assuming low
and high diesel sales can be estimated using vehicle class EFs
and urban VMT fractions from this EPA report. The low diesel
sales scenario in the 1987 EPA report assumed that diesel sales
remained constant at mid-1980's levels, while the high sales
scenario assumed an increase consistent with EPA projections at
that time. The low diesel urban sales EF was 0.0359 g/mi, and
the high diesel urban sales EF was 0.0507 g/mi. The lower EF for
1995 predicted by Sienicki (0.0305 g/mi) is partly due to
development of stricter standards than predicted in 1987, and
also to such factors as even lower light duty diesel vehicle
market shares than in either of the 1987 EPA report scenarios,
Sienicki's low sulfur fuel and freeway road use adjustments, and
the smaller g/bhp-hr to g/mi conversion factors he used.

For this report, Sienicki's fleet average EFs without the freeway road use adjustments will be used for the risk estimates. This is consistent with past EPA practice. However, further investigation of the use of such freeway road use adjustments is warranted. Resulting urban diesel particulate matter national fleet average EFs for 1990, 1995, 2000, and 2010 are summarized in Table 9-2.

Table 9-2. Urban Diesel Particulate Matter National Fleet Average EFs.

Year	EF (g/mi)
1990	0.0669
1995	0.0356
2000	0.0188
2010	0.0105

9.3.3 Nationwide Diesel Particulate Matter Emissions

Sienicki's urban diesel particulate matter fleet average EFs are based on the mix of vehicle classes expected in an urban area. Urban and rural VMT fractions differ, particularly for some of the heavy duty vehicle classes where more rural use occurs. Since these heavy duty subclasses are responsible for the majority of diesel particulate matter emissions, it would not be appropriate to use urban fleet average EFs to calculate nationwide diesel particulate matter emissions. Instead, the EFs by vehicle class calculated by Sienicki (without the freeway road use adjustment) were combined using the nationwide VMT splits from the MOBILE4.1 fuel consumption model (EPA, 1992) to estimate nationwide fleet average EFs. Using this approach, the nationwide fleet average EFs for 1990, 1995, 2000, and 2010 are 0.0910, 0.0523, 0.0291, and 0.0178 g/mi respectively.

These nationwide fleet average EFs were then multiplied by total nationwide fleet VMT obtained from the MOBILE4.1 fuel consumption model to estimate nationwide diesel particulate matter emissions. No recent rural diesel particulate matter fleet average EFs were available; thus, separate rural and urban diesel particulate matter emission levels could not be estimated. Nationwide diesel particulate matter emission estimates are listed in Table 9-3. The 1990 nationwide diesel particulate matter emission estimate of 163,118 metric tons compares with a higher 1990 estimate of 384,000 metric tons for diesel vehicles in a recent EPA report on air pollutant emission estimates (EPA, 1991b).

Table 9-3. Nationwide Diesel Particulate Matter Emissions.

Year	Total Nationwide Fleet VMT (mi)	Nationwide Diesel Particulate Matter (metric tons)
1990	1793.07×10^9	163,118
1995	2029.74×10^9	106,080
2000	2269.25×10^9	66,076
2010	2771.30×10^9	49,441

9.4 Atmospheric Reactivity and Residence Times of Particulate Phase Polycyclic Organic Matter (POM)

POM species can exist in both the gas and particulate phases in the atmosphere. The distribution between the two phases is determined by the vapor pressure of the species, the ambient temperature, and the amount of airborne particulate matter present. Cold temperatures and higher aerosol concentrations lead to greater association of POM with particles. The focus of this section is on particulate phase POM, since most of the POM emitted by motor vehicles is in this form. The information that follows on transformation and residence times has been mainly excerpted from a report produced by Systems Application International for the EPA (Ligocki and Whitten, 1991).

9.4.1 Particulate-Phase Chemistry

The determination of rate constants for POM that are adsorbed to particles is difficult, because these rates are strongly influenced by the characteristics of the surface to which the POM are adsorbed. Thus, the observations reported in the literature regarding the reactivity of adsorbed POM tend to appear contradictory. Early studies and extrapolation from reactivity studies of POM in organic solution suggested that POM compounds react rapidly on surfaces (NAS, 1972). Later work demonstrated that, although POM present on substrates such as silica and alumina photolyze rapidly, POM present on coal fly ash and carbon black were resistant to photochemical degradation (Korfmacher et al., 1980, 1981; Behymer and Hites, 1985). Significant differences in photochemical degradation rates have been reported between two different fly ashes (Dlugi and Güsten, 1983).

Nonetheless, some POM may be capable of being oxidized in the particulate phase. Fox and Olive (1979) reported that 90 percent of anthracene present on atmospheric particulate matter disappeared in four days when exposed to sunlight, whereas only a small fraction of the anthracene which was exposed to ambient air but shielded from light disappeared. The conversion of POM present on diesel particulate matter and exposed to ozone has been reported (Van Vaeck and Van Cauwenberghe, 1984) and appears

9–10

to be an important removal pathway for some POM. However,
Grosjean and co-workers (1983) found no degradation of
benzo[a]pyrene and perylene adsorbed onto a variety of
substrates, including diesel soot, over a three hour exposure to
100 ppb ozone. The conversion of POM present in soot and exposed
to NO_x has also been reported (Butler and Crossley, 1981).
Unfortunately, the species responsible for this observed
oxidation was not determined. Even though POM react readily with
N_2O_5 in the gaseous phase, the reaction of N_2O_5 with adsorbed POM
is significantly lower.

9.4.2 Aqueous Phase Chemistry

POM are slightly soluble in water, and will be incorporated
to some degree into clouds and rain. For species which are
associated with particles, the water-affinity of the particle
surface will determine the degree to which they will be
incorporated into clouds and/or rain. Polycyclic ketones and
quinones are much more soluble in water than the parent POM and
will be incorporated into clouds and rain to a much greater
degree.

9.4.3 Reaction Products

Most POM reactions proceed by addition, forming polycyclic
aromatic ketones, quinones, epoxides, and nitro compounds.

Much of the focus on POM oxidation products has centered on
the nitro-POM, since several of these compounds, such as the
dinitropyrenes, are known to be extremely potent mutagens.
Although the yields of these species are generally not large,
they may still account for a significant fraction of the observed

mutagenicity of ambient POM. In diesel exhaust particulate matter, 3-nitrofluoranthene was the major constituent.

9.4.4 Polycyclic Organic Matter Residence Times

For particulate species, the rate of removal by wet and dry deposition will depend upon the particle size distribution. Large particles are removed rapidly from the atmosphere by sedimentation and impaction. Smaller particles do not contain sufficient mass to sediment or impact, but diffuse much more rapidly than do large particles. As a result, removal rates of atmospheric particles are governed by the competition between these two types of processes, and generally reach a minimum somewhere in the range 0.1 to 1.0 micrometer (μm). This size range is often referred to as the accumulation mode, because particles in this size range tend to persist, and hence accumulate. The National Ambient Air Quality Standard for PM_{10} is based on the particulate matter less than 10 μm in diameter.

Particle size distributions for a few POM have been reported. Evidence suggests that the larger, less volatile POM tend to be present on smaller particles than the smaller, more volatile POM (Pistikopoulos et al., 1990), but that all POM are primarily associated with submicron particles. Van Vaeck and Van Cauwenberghe (1978) measured particle size distributions for a set of POM finding that 90% of the 4-ring POM and 91% of the 5-ring POM are associated with particles ≤1.5 μm in diameter. These data are comprehensive, and were used in calculating the wet and dry deposition of POM.

Residence times are presented for two individual POM species: an intermediate POM (pyrene) and a particulate-phase POM (benzo[a]pyrene). These examples provide comparisons of the importance of chemical transformation to other species versus physical removal, and differences between POM species of varying size. It should be noted, however, that in some cases it is not appropriate to view atmospheric reactions as destruction pathways for toxic species, because the products formed from its destruction may be equally toxic, or even more toxic. Most POM reactions, for instance, proceed by addition, forming polycyclic aromatic ketones, quinones, epoxides, and nitro compounds.

Residence times for POM as a class are also presented. For POM as a class, however, atmospheric residence times are determined by physical processes only. Chemical reactions may transform individual compounds, but available evidence suggests that the products of this transformation are also POM species. Therefore, the residence time of POM as a class may be determined by wet and dry deposition only.

9.4.4.1 Pyrene

Pyrene has a vapor pressure that falls within the range where either gas phase or particulate phase processes might dominate depending upon ambient conditions. Under wintertime conditions, and/or high particle loading conditions, a majority of pyrene concentration may be associated with particles. Therefore, its atmospheric residence time is determined by both gas-phase and particulate-phase processes.

Residence times for pyrene were calculated by considering gas-phase chemical reactions with OH and N_2O_5, particulate phase chemical reaction with O_3, aqueous phase chemical reactions with OH and O_3, and wet and dry deposition. The calculated residence times for pyrene are presented in Table 9-4. Because of the similarities between the chemical reactivity and physical properties of pyrene and fluoranthene, the residence times presented in Table 9.1 also can be considered to apply to fluoranthene.

The calculated residence times for fluoranthene and pyrene range from 0.8 to 1.6 hours under summer, daytime, clear-sky conditions. These residence times are roughly half as long as those calculated for naphthalene, a POM present virtually exclusively in the gas phase. As with naphthalene, gas phase reaction with OH is the most important atmospheric removal pathway. However, for fluoranthene and pyrene, particulate-phase processes including reaction with O_3 and wet and dry deposition are also significant.

Under cloudy conditions, in-cloud chemical destruction accounts for 10 to 30 percent of pyrene removal at night in the summer. In the daytime and in the winter, in-cloud processes are less important. Both the OH and O_3 oxidations contribute to the aqueous reactivity, with the OH pathway more important in the summertime, and both pathways important in the winter.

Wet deposition is very rapid for particulate-phase fluoranthene/pyrene. Particle scavenging leads to residence times on the order of 2 to 20 hours in the wintertime. Dry deposition is a major removal mechanism for fluoranthene/pyrene at night,especially in winter and under cloudy-sky conditions. Dry deposition of particulate-phase fluoranthene/pyrene is more efficient than that of gas-phase fluoranthene/pyrene.

Major uncertainties in the estimate of residence times for fluoranthene/pyrene include the order-of-magnitude uncertainty in the particle scavenging rate and the rate of reaction of the particulate-phase species with ozone. Also significant is the factor-of-two uncertainty in the OH radical concentration.

TABLE 9-4. Atmospheric residence time calculation for fluoranthene/pyrene. All times are in hours unless otherwise noted.

	Los Angeles		St. Louis		Atlanta		New York	
	July	Jan	July	Jan	July	Jan	July	Jan
Clear sky - day	1.2	9	0.8	18	0.8	14	1.6	30
Clear sky - night	60	80	80	90	70	60	70	130
Clear sky - avg	2	18	1.3	40	1.3	30	2	60
Cloudy - day	3	18	1.8	30	1.8	30	3	50
Cloudy - night	50	110	70	80	50	80	80	130
Cloudy - avg	5	40	3	50	3	50	5	80
Rainy - day	--[*]	2-9[**]	1.5-1.8[**]	1.0-6[**]	1.7	2-9[**]	3	0.9-6[**]
Rainy - night	--[*]	1.2-8[**]	4-18[**]	0.7-5[**]	6-17[**]	1-7[**]	4-20[**]	0.7-5[**]
Rainy - avg	--[*]	1.5-8[**]	2-3[**]	0.8-5[**]	2-3[**]	1-8[**]	3-5[**]	0.7-5[**]
Monthly Climatological Average	2	20	2	30-40[**]	2	18-30[**]	3	20-60[**]

[*]Not calculated since July rainfall is zero for Los Angeles.
[**]Range of values calculated using high and low estimates for particle scavenging.

9.4.4.2 Benzo[a]pyrene (B[a]P)

Benzo[a]pyrene is present in the particulate phase under most conditions. Therefore, its atmospheric residence time is primarily determined by particulate-phase processes. Residence times for benzo[a]pyrene were calculated by considering gas-phase chemical reaction with OH, particulate phase chemical reaction with O_3, aqueous phase chemical reactions with OH and O_3, and wet and dry deposition. The calculated residence times for benzo[a]pyrene are presented in Table 9-5. By comparison to fluoranthene/pyrene, the calculated summertime residence times are longer for benzo[a]pyrene. This is due to the greater association with particles of benzo[a]pyrene. Interestingly, however, the calculated wintertime residence times for benzo[a]pyrene are shorter than fluoranthene/pyrene. In fact the difference in residence time between summer and winter is only a factor of two to three for benzo[a]pyrene.

The reaction of O_3 with particulate-phase benzo[a]pyrene is predicted to be the dominant removal mechanism for benzo[a]pyrene under most conditions. The reaction of gas-phase benzo[a]pyrene with OH is also expected to be significant in the summertime, despite the relatively small fraction of benzo[a]pyrene present in the gas phase.

Unlike the gaseous POM, wet and dry deposition are significant
atmospheric removal mechanisms for benzo[a]pyrene . Wet deposition leads to atmospheric residence times of 0.5 to 3 hours on rainy days and contributes significantly to monthly climatological average residence time. Dry deposition is less important, but still contributes up to 30 percent of the removal.

Major uncertainties in the estimate of residence times for benzo[a]pyrene include the order of magnitude uncertainties in the rate of reaction of the particulate-phase species with ozone, and the particle scavenging rate. The calculated residence times for benzo[a]pyrene are, therefore, significantly more uncertain than those calculated for fluoranthene/pyrene.

9.4.4.3 Other POM Species

Among the particulate-phase POM, the residence times calculated for benzo[a]pyrene are probably valid for other reactive species such as perylene. However, more stable POM, such as the benzofluoranthenes, benzo[e]pyrene, and coronene, may be removed primarily by physical processes, and would have residence times up to ten times longer than that calculated for benzol[a]pyrene.

9.4.4.4 POM as a Class

The summertime residence times for pyrene and B[a]P suggest that POM are transformed relatively rapidly in the summertime. For

TABLE 9-5. Atmospheric residence time calculation for benzo[a]pyrene. All times are in hours unless otherwise noted.

	Los Angeles		St. Louis		Atlanta		New York	
	July	Jan	July	Jan	July	Jan	July	Jan
Clear sky – day	4	13	3	20	4	15	5	30
Clear sky – night	11	30	11	40	11	20	11	90
Clear sky – avg	5	19	5	30	5	17	6	50
Cloudy – day	6	19	6	30	6	18	7	50
Cloudy – night	11	30	11	40	11	20	11	90
Cloudy – avg	7	20	7	40	7	20	8	70
Rainy – day	--[*]	0.5–3[**]	0.5–3[**]	0.5–4[**]	0.5–3[**]	0.5–3[**]	0.5–3[**]	0.5–4[**]
Rainy – night	--[*]	0.5–3[**]	0.5–3[**]	0.5–4[**]	0.5–3[**]	0.5–3[**]	0.5–3[**]	0.5–4[**]
Rainy – avg	--[*]	0.5–3[**]	0.5–3[**]	0.5–4[**]	0.5–3[**]	0.5–3[**]	0.5–3[**]	0.5–4[**]
Monthly Climatological Average	6	10–18[**]	4–6[**]	20–30[**]	5–6[**]	8–17[**]	5–7[**]	16–40[**]

[*]Not calculated since July rainfall is zero for Los Angeles.
[**]Range of values calculated using high and low estimates for particle scavenging.

the case of species such as fluoranthene and pyrene, their oxidation products will condense onto atmospheric particles. At that point, they may be relatively stable against further oxidation, and may persist until removed by wet and dry deposition.

The atmospheric residence time of a generic non-reactive particulate-phase POM that is removed only by physical processes (i.e., wet and dry deposition) is presented in Table 9-6. Because the algorithms used to calculate the dry deposition velocities and wet deposition rates did not contain any city-specific information, the calculated clear-sky and rainy residence times are the same for all cities. The differences in the monthly climatological average residence times reflect only the differences in monthly rainfall among the cities. Atmospheric residence times range from less than a day to three days in both summer and winter.

9.4.5 Urban Airshed Modeling of POM

The explicit modeling of POM is difficult to achieve due to the inherent complexity of POM 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.

Since POM basically consists of three distinct species categories, all three would have to be taken into consideration. These are the naphthalenes, which are an order of magnitude higher than the concentrations of any of the other POM (thought not among the more toxic constituents of POM); the other gas-phase POM concentrations that are much greater than the particulate-phase concentrations; and the particulate phase itself. Each of these species has its own transformation and reactivity parameters that need to be taken into consideration.

Due to these many considerations and parameters, and the absence of software to implement these factors, the Urban Airshed Modeling of POM was not accomplished in the St. Louis study (Ligocki and Whitten, 1991).

However, POM was treated explicitly in the Baltimore-Washington and Houston area studies (Ligocki et al., 1992). POM was assigned to three species categories in the UAM-Tox (as described above), based upon molecular weight (MW):

    NAPH   MW < 160
    POM1   160 < MW < 220
    POM2   220 < MW

The species NAPH consists largely of naphthalene and substituted naphthalenes, which account for the bulk of the POM mass. NAPH reacts rapidly with OH and slowly with N_2O_5. POM1 and POM2 are represented as nonreactive. Additional information on the modifications made to UAM to model POM explicitly are described in the reference cited above.

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. The maximum daily average POM for the 1988 base scenario was 6.8 $\mu g/m^3$. Simulated daily average POM concentrations were much lower in the Washington area (0.5-1.0 $\mu g/m^3$) than in the Baltimore area (1-6.8 $\mu g/m^3$). Motor vehicle-related NAPH accounted for about 15% of total NAPH emissions, motor vehicle-related POM1 accounted for about 43% of total POM1 emissions, and motor vehicle-related POM2 accounted for about 35% of total POM2 emissions. Furthermore, motor vehicle-related POM accounted for about 15% of the total simulated POM concentration, based on the 1995 no motor vehicle scenario.

Since no data were available on measured POM concentrations in the Baltimore-Washington area, simulated concentrations were compared to measured concentrations from other cities. Concentrations of POM in Washington were in line with concentrations in other cities, but concentrations in Baltimore appear to be overpredicted.

In the winter 1988 base scenario, the maximum daily average POM concentration was 4.4 $\mu g/m^3$, lower than in summer. NAPH emissions decreased because they were primarily influenced by evaporative emissions from asphalt paving. Emissions of POM1 and POM2, the larger POM components, increased significantly in winter because of residential wood combustion. Motor vehicle-related POM concentrations with federal reformulated gasoline use decreased more in winter than in summer, ranging from 4 to 8 percent. Motor vehicle-related POM accounted for about 10% of the maximum simulated concentration, based on the 1995 no motor vehicle scenario.

For the summer 1987 base scenario in Houston, the maximum daily average POM concentration was 3.4 $\mu g/m^3$. Motor vehicle-related NAPH accounted for about 17% of total NAPH emissions, motor vehicle-related POM1 accounted for about 24% of total POM1 emissions, and motor vehicle-related POM2 accounted for about 19% of total POM2 emissions. Furthermore, motor vehicle-related POM accounted for about 18% of the maximum simulated concentration, based on the 1995 no motor vehicle scenario. Simulations for the summer Houston episode predicted larger decreases than in the Baltimore-Washington area with the use of reformulated gasoline. Simulated concentrations of POM were in good agreement with concentrations measured in other cities.

TABLE 9-6. Atmospheric residence time calculation for a generic particulate-phase POM which is removed by physical processes only. All times are in hours unless otherwise noted.

	Los Angeles		St. Louis		Atlanta		New York	
	July	Jan	July	Jan	July	Jan	July	Jan
Clear sky - day	60	120	60	120	60	120	60	120
Clear sky - night	90	90	90	90	90	90	90	90
Clear sky - avg	70	100	70	100	70	100	70	100
Rainy - day	--[*]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]
Rainy - night	--[*]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]
Rainy - avg	--[*]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]	0.5-4[**]
Monthly Climatological Average	70	16-60[**]	15-50[**]	30-80[**]	12-40[**]	13-50[**]	15-50[**]	18-60[**]

[*]Not calculated since July rainfall is zero for Los Angeles.
[**]Range of values calculated using high and low estimates for particle scavenging.

9.5 Exposure Estimation

9.5.1 Annual Average Exposures Using HAPEM-MS

To obtain urban and rural annual average exposures, urban diesel particulate matter national fleet average emission factors in Table 9-2 were first multiplied by the urban and rural g/mile to µg/m^3 conversion factors obtained from HAPEM-MS for 1988 (Johnson et al., 1992):

$$CONV_{urban} = 29.4 \ (µg/m^3)/(g/mile)$$
$$CONV_{rural} = 15.9 \ (µg/m^3)/(g/mile)$$

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. The adjustment factors used for 1990, 1995, 2000, and 2010 are 1.031, 1.123, 1.218, and 1.412, respectively. Resulting urban and rural annual average exposures for 1990, 1995, 2000, and 2010 are given in Table 9-7.

9.5.2 Comparison of HAPEM-MS to Ambient Monitoring Data

Using ambient monitoring data (EPA, 1991b, 1991c), the concentration of diesel particulate matter in ambient air samples can be estimated. For 1990, the national average total suspended particle concentration is estimated to be about 48 µg/m^3 (EPA, 1991c). This can be multiplied by percent contribution of diesel particulate matter to TSP, which is calculated to be 5.12%. This percentage was obtained by dividing an estimate for diesel emissions of 384,000 metric tons (EPA, 1991b) by a TSP estimate of 7.5×10^6 metric tons (EPA, 1991c). The resultant concentration of diesel particulate matter obtained by multiplying 48 µg/m^3 by 5.12% is 2.46 µg/m^3. This number was then adjusted for integrated exposure, resulting in integrated exposure estimate of 1.52 µg/m^3. The HAPEM-MS 1990 urban diesel particulate matter annual average exposure of 2.03 µg/m^3 is about 134% of this value. The HAPEM-MS 1990 rural diesel particulate matter annual average exposure of 1.10 µg/m^3 is about 72% of this value.

9.6 Carcinogenicity of Diesel Particulate Matter and Unit Risk Estimates

9.6.1 Most Recent EPA Assessment

A draft health assessment document for diesel emissions has been prepared (EPA, 1990b). Much of the information contained in this section has been taken from this document. An update of this document is expected shortly.

Table 9-7. Diesel Particulate Matter Annual Average Exposures.

Year	Exposure ($\mu g/m^3$)		
	Urban	Rural	Nationwide
1990	2.03	1.10	1.80
1995	1.18	0.64	1.05
2000	0.67	0.36	0.60
2010	0.44	0.24	0.39

9.6.1.1 Description of Available Carcinogenicity Data

To evaluate the carcinogenicity of diesel engine particulate emissions, controlled animal and mutagenicity studies were conducted as well as studies of populations occupationally exposed to diesel exhaust. The following paragraphs contain a brief summary of the EPA evaluation of these studies; the EPA draft document discusses these studies in more detail (EPA, 1990b).

Genotoxicity

Extensive Ames test studies with *Salmonella* have unequivocally demonstrated direct-acting mutagenic activity in both the particle and gaseous fractions of diesel exhaust (Huisingh et al., 1978; Siak et al., 1981; Claxton, 1981, 1983; Claxton and Kohan, 1981; Dukovich et al., 1981; Lewtas, 1983; Brooks et al., 1984; Matsushita et al., 1986). The induction of gene mutations has been reported in several *in vitro* mammalian cell lines after exposure to extracts of diesel particulate matter (Casto et al., 1981; Chescheir et al., 1981; Curren et al., 1981; Liber et al., 1981; Mitchell et al., 1981; Barfnecht et al., 1982; Li and Royer, 1982; Brooks et al., 1984; Morimoto et al., 1986). Dilutions of whole diesel exhaust did not induce sex-linked recessive lethals in *Drosophila* (Schuler and Niemeier, 1981) or specific-locus mutations in male mouse germ (sperm) cells (Russell et al., 1980).

Structural chromosome aberrations and sister chromatid exchanges (SCE) in mammalian cells have been induced by particles and direct diesel exhaust (Guerrero et al., 1981; Mitchell et al., 1981; Lewtas, 1983: Morimoto et al., 1986; Pereira et al., 1982; Tucker et al., 1986). Sister chromatid exchanges, but not chromosomal aberrations, were observed in Chinese hamster cells upon exposure to particle extracts (Brooks et al., 1984). Whole exhaust induced micronuclei, but not SCE or structural aberrations were found in bone marrow of male Chinese hamsters exposed to whole diesel emissions for 6 months. In shorter exposure (7 weeks), neither micronuclei nor structural aberrations were increased in bone marrow of female Swiss mice

(Pereira et al., 1981a). Likewise whole diesel exhaust did not induce dominant lethal or heritable translocations in male mice exposed for 7.5 and 4.5 weeks, respectively (Russell et al., 1980).

Analysis of caudal sperm for sperm head abnormalities was conducted (Pereira et al., 1981b) after exposure to diesel exhaust particles and it was found that the exposed incidence of abnormalities was not above the control levels. Conversely, male Chinese hamsters exposed to diesel particulate matter (Pereira et al., 1981c) exhibited almost a threefold increase in sperm head abnormalities.

Animal Studies

As early as 1955, there was evidence (Kotin et al., 1955) for tumorigenicity and carcinogenicity of acetone extracts of diesel exhaust in skin tumorigenesis tests. Also data suggested a difference in response depending on the engine operating mode. Until the mid 1980's, no chronic studies assessing inhalation of diesel exhaust, the relevant mode for human exposure, had been reported. This is, however, the route of exposure which was used in the most extensive, recent studies. Studies employing rats and an adequate experimental design were nearly all positive in demonstrating diesel exhaust-induced increases in tumorigenicity. The 9.5 percent increase in tumor incidence for female Wistar rats reported by Heinrich et al. (1986a) is supported by the report by Mauderly et al. (1987), which showed a 3.6 percent and 12.8 percent increase in tumor incidence for F344 rats following chronic exposure to diesel exhaust at particle concentrations of 3.5 and 7.0 mg/m^3, respectively. However, only one of the squamous cell tumors reported by Heinrich et al. (1986a) was classified as a carcinoma. In the Mauderly et al. (1987) study, the carcinoma incidence was 0.9, 1.3, 0.5, and 7.5 percent for the control, low, medium, and high exposure groups, respectively.

The inhalation studies by Wong et al. (1986) and Bond et al. (1990) affirm observations of the potential carcinogenicity of diesel exhaust by providing evidence for DNA damage in rats. Similarly, Iwai et al. (1986) demonstrated diesel exhaust-induced tumorigenicity in rats exposed to an exhaust particle concentration of 4.9 mg/m^3, although the sample size was small. This study also reported development of a splenic lymphoma, which represents the only nonpulmonary tumor resulting from inhalation exposure to diesel exhaust. The long-term inhalation study by Ishinishi et al. (1986) showed a greater incidence of carcinomas (6.5 percent) in rats following 30-month exposure to diesel exhaust at 4 mg/m^3, but not at lower (0.4, 1.0, or 2.0 mg/m^3) exposure levels. However, Brightwell et al. (1986) demonstrated a dose-dependent increase in tumor incidence for male and female F344 rats exposed to filtered, but no unfiltered diesel exhaust (five 16 hour periods per week), at concentrations as low as 2.2 mg/m^3 and also at 6.6 mg/m^3. Filtered and unfiltered exhaust are used to discriminate between the gaseous and particle effects. This study indicated that, for unfiltered exhaust, the tumor incidence was higher for female rats (0 percent, 15 percent, or

54 percent at 0.0, 2.2, or 6.6 mg/m^3) than for male rats (1 percent, 4 percent, or 23 percent for 0.0, 2.2, or 6.6 mg/m^3). The filtered exhaust showed no increase in tumors when compared to controls. Thus, these studies demonstrated carcinogenic effects in rats at exposure levels ranging from 2.2 to 7.0 mg/m^3.

The inhalation of whole diesel exhaust by NMRI mice (Heinrich et al., 1986a,b; Stober, 1986), Sencar mice (Pepelko and Peirano, 1983), and in rats (Takaki et al., 1989) also provided evidence of carcinogenicity. In Orthoefer et al. (1981), the exposure of Strain A mice to irradiated diesel exhaust (to simulate sunlight exposure and resulting reactions) did not produce any significant signs of gross toxicity or affect the growth rates of the mice. Exposures ranged from 20 hr/day, 7 days\week, for 7 weeks, diluted 1:13 in one experiment to an 8 week inhalation at 6 mg/m^3 in another. The mice were then held for an additional 26 weeks in clean air after cessation of exposure. Exposures to either irradiated or nonirradiated exhaust did not result in significantly increased lung tumor incidences compared with controls. Due to short exposures selected for these studies they are considered to be screening tests. The short exposure and holding periods prior to sacrifice are based upon the rapid increase in tumor rates in positive tests. The observed increase in mutagenicity of irrradiated exhaust observed in chronic bioassys is discussed in Chapter 12, Section 12.4.3.

Both the Heinrich et al. (1986a) and Brightwell et al. (1986) studies provide negative results for tumorigenicity of diesel exhaust in hamsters, a species known for its resistance to tumor induction. Negative results were also presented by several other investigators (Takemoto et al., 1986; Schreck et al., 1982; Barnhart et al., 1981; Karagianes et al., 1981), but these studies tended to employ inadequate exposure durations, low exposure concentrations, or inadequate animal numbers per group. A negative study reported by Kaplan et al. (1982) contained a high incidence of tumors in the control group. Similarly, the studies using monkeys (Lewis et al., 1986) and cats (Pepelko and Peirano, 1986) were of inadequate duration (2 years) for these longer-lived species.

Alternate exposure routes including dermal exposure, skin painting, and subcutaneous injection provided additional evidence for tumorigenic effects of diesel exhaust. Evidence for tumorigenicity was demonstrated by Kotin et al. (1955) for mice to which an acetone extract of diesel exhaust particles was applied dermally. Nesnow et al. (1982) also showed that extracts from some diesel engines were potentially tumorigenic following dermal application to rodents. A significant increase in the incidence of subcutaneous tumors in female C57B1 mice was reported by Kunitake et al. (1988) for subcutaneous administration of light-duty diesel exhaust tar extract at doses of 500 mg/kg. Doses at or below 200 mg/kg, however, were negative. Takemoto et al. (1988) provided additional data for this study and reported an increased tumor incidence in the mice following injection of light-duty engine exhaust extract at doses

of 100 and 500 mg/kg. Negative results were reported by Depass et al. (1982) for skin-painting studies using mice and acetone extracts of diesel exhaust particle suspensions. However, in this study the exhaust particles were collected at temperatures of 100°C, a temperature that would minimize the condensation of vapor-phase organics and, therefore, reduce the availability of potentially carcinogenic compounds that might normally be present on diesel exhaust particles. Intraperitoneal injection studies using Strain A mice were generally negative.

Diesel exhaust is composed of gaseous and particle phases and is known to be a complex mixture containing verified and potential carcinogens. Nevertheless, because of the negative results via inhalation, the fact that most POM is adsorbed onto particles, and because the potentially carcinogenic agents present in the gaseous phase, i.e., benzene, formaldehyde, are not known to induce lung tumors, it is unlikely that this component contributes to the tumorigenic responses. A study by Grimmer et al. (1987) demonstrated that a whole exhaust condensate fraction containing polycyclic aromatic hydrocarbons (PAH) with 4 to 7 rings produced a high tumor incidence when implanted into rat lungs. It was also noted that this fraction represented only 0.8 percent of the total weight of the exhaust condensates, and that some tumorigenicity was also associated with nitroaromatic fractions. The PAH fraction produced a tumor incidence similar to that of a low concentration of benzo[a]pyrene (BaP).

Several of the previously discussed studies indicated that only the whole (unfiltered) diesel exhaust is tumorigenic or carcinogenic and that these properties are eliminated or greatly minimized in filtered diesel exhaust exposure. Inhalation experiments using tumor initiators (Brightwell et al., 1986; Heinrich et al., 1986a; Takemoto et al., 1986) did not provide conclusive results regarding the carcinogenic potential of filtered vs. whole diesel exhaust. Although the tumorigenicity of the gaseous fraction is presently unresolved and most experiments using filtered exhaust were negative, most of these experiments did not provide definitive evidence that a maximum tolerated dose was achieved. The carbon core of the exhaust particle has been determined to have carcinogenic potential. The fact that allegedly inert, insoluble biochemically, "noncarcinogenic" particles such as titanium dioxide (Lee et al., 1986) or instillation of activated carbon (Kawabata et al., 1986) have been shown to induce lung cancer at very high concentrations is of concern in this respect. Studies currently in progress, indicating that carbon black, containing essentially no organics, was as effective as diesel exhaust in lung cancer induction, (see Section 9.6.3.1) supports the approach that the carbon core plays a major role in the pulmonary carcinogenicity of diesel exhaust in rats.

Although uncertainties exist regarding the tumorigenic potential of the gaseous component and the carbon core component of diesel exhaust, it is clear that diesel exhaust is carcinogenic in animals inducing pulmonary tumors. This

contention is supported by positive results in numerous, independent studies in male and females of at least two species and by several routes of administration, including inhalation, intratracheal administration, skin painting, and subcutaneous injection.

Human Data

Certain extracts of diesel exhaust also have been demonstrated to be mutagenic and carcinogenic in humans. Since large working populations are currently exposed to diesel exhaust, and since nonoccupational exposures currently are of concern as well, the possibility that exposure to this complex mixture may be carcinogenic to humans has become an important public health issue.

A major difficulty with the occupational studies considered here was the measurement of the actual diesel exhaust exposure. Most studies compared men in job categories with presumably some exposure to diesel exhaust with either standard populations (presumably no exposure to diesel exhaust) or with men in other job categories from industries with little or no potential for diesel exhaust exposure. A few studies have included measurements of diesel fumes, but there is no standard method for the measurement. No attempt is made to correlate these exposures with the cancers observed in any of these studies, nor is it clear exactly which diesel particulate matter should be measured to assess the occupational exposure to diesel exhaust. The occupations involving potential exposure to diesel exhaust are miners, truck drivers, transportation workers, railroad workers, and heavy equipment operators.

The seven cohort studies reviewed by EPA (1990b) have mainly demonstrated an increase of lung cancer. The three cohort studies of bus company workers by Waller (1981), Rushton et al. (1983), and Edling et al. (1987) failed to demonstrate any statistically significant excess risk of lung cancer, but these studies have certain methodological problems such as small sample sizes, short follow-up periods, lack of information on confounding variables, and lack of analysis by duration of exposure or latency that preclude their use in determining the carcinogenicity of diesel exhaust. Although the Waller (1981) study had a 25-year follow-up period, the cohort was restricted to only employees (ages 45 to 64) currently in service. Employees who left the job earlier, as well as those who were still employed after age 64 and who may have died from cancer, were excluded.

Wong et al. (1985) conducted a mortality study of heavy equipment operators that demonstrated a significant increased risk of liver cancer in the total cohort and in various subcohorts. The same analysis also showed statistically significant deficits in cancers of the large intestine and rectum. Metastasis from the cancers of the large intestine and rectum to the liver probably were misclassified as primary liver cancer which lead to an observed excess risk. This study did

demonstrate a nonsignificant positive trend for cancer of the lung with length of membership and latency. Individuals without work histories who started work prior to 1967 when records were not kept may have been the ones who were in the same job for the longest period of time. The workers without job histories included those who had the same job before and after 1967 and thus may have worked about 12 to 14 years longer; these workers exhibited significant excess risks of lung cancer and stomach cancer. If this assumption about their jobs is correct, then these site-specific causes may be linked to diesel exhaust exposure. However, this study has quite a few methodological limitations such as the absence of detailed work histories for 30 percent of the cohort and the availability of only partial work histories for the remaining 70 percent; thus, jobs were classified and ranked according to presumed diesel exposure. Information is lacking regarding duration of employment in the job categories (used for surrogate of exposure), and other confounding factors (alcohol consumption, cigarette smoking, etc.).

A two-year mortality analysis of the American Cancer Society's prospective study by Boffetta et al. (1988), after controlling for age and smoking, demonstrated an excess risk of lung cancer in certain occupations with potential exposure to diesel exhaust (railroad workers, heavy equipment operators, truck drivers, and miners). These excesses were statistically significant among miners (RR = 2.67, 95 percent CI = 1.63 to 4.37) and heavy equipment operators (RR = 2.6, 95 percent CI = 1.12 to 6.06). The elevated risks were nonsignificant in railroad workers (RR = 1.59) and truck drivers (RR = 1.24). RR (OR) and CI are defined in Section 6.6.3.4. A dose response was also observed for the truck drivers. With the exception of miners, exposure to diesel exhaust occurred in the three other occupations showing an increase in the risk of lung cancer. This study exhibited two methodological limitations. These include, the lack of representiveness of the study population composed of volunteers only and the questionable reliability of exposure data based on self-administered questionnaires which were not validated. Despite these limitations this study is suggestive of a causal association between exposure to diesel exhaust and excess risk of lung cancer.

There were two mortality studies conducted on railroad workers by Howe et al. (1983) in Canada and Garshick et al. (1988) in the United States. The Canadian study found relative risks of 1.2 and 1.35 among "possibly" and "probably" exposed groups, respectively. The trend test showed a highly significant dose response relationship with exposure to diesel exhaust and the risk of lung cancer. The main limitation of the study was the inability to separate overlapping exposures of coal dust and diesel fumes. Information on jobs was available at retirement only. There was also insufficient detail on the classification of jobs by diesel exhaust exposure. The exposures could have been noncurrent, but since the data are lacking, it is possible that observed excess could be due to the effect of both coal dust and diesel fumes and not due to just one or the other. However,

it should be noted that, so far, coal dust has not been demonstrated to be a pulmonary carcinogen in studies on coal miners. But lack of data on confounders such as asbestos and smoking makes interpretation of this study difficult. The findings of this study are, at best, suggestive of diesel exhaust being a lung carcinogen.

The most definitive evidence for linking diesel exhaust exposure to lung cancer comes from a railroad worker study conducted in the United States (Garshick et al., 1988) which was funded by EPA. Relative risks of 1.57 (95 percent CI = 1.19 to 2.06) and 1.34 (95 percent CI = 1.02 to 1.76) were found for ages 40 to 44 and 45 to 49, respectively, after the exclusion of workers exposed to asbestos. This study also found that risk of lung cancer increased with increasing duration of employment. This large cohort study with lengthy follow up and adequate analysis, including dose response (based on duration of employment as a surrogate) as well as adjustment for other confounding factors such as asbestos and smoking, makes the observed association between increased lung cancer and exposure to diesel exhaust more meaningful.

Among the seven lung cancer case-control studies reviewed in EPA (1990b), the study by Lerchen et al. (1987) was the only one that did not find increased risk of lung cancer, after adjusting for age and smoking, for diesel fume exposure. The major limitation of this study was lack of adequate exposure data derived from the job titles obtained from occupational histories. Next of kin provided the occupational histories for 50 percent of the cases which were not validated. The power of the study was small (analysis done on males only, 333 cases). On the other hand, statistically nonsignificant excess risks were observed for diesel exhaust exposure by Williams et al. (1977) in railroad workers (OR = 1.4) and truck drivers (OR = 1.34), by Hall and Wynder (1984) for workers who were exposed to diesel exhaust versus workers who were not (OR = 1.4 and 1.7 with two different criteria), and by Damber and Larsson (1987) in professional drivers (OR = 1.2). These rates adjusted for age and smoking. Both Williams et al. (1977) and Hall and Wynder (1984) had high nonparticipation rates of 47 percent and 36 percent, respectively. In addition, the self-reported exposures used in the study by Hall and Wynder (1984) were not validated. This study also had low power to detect excess risk of lung cancer for specific occupations.

The study by Benhamou et al. (1988), after adjusting for smoking, found significantly increased risks of lung cancer among French motor vehicle drivers (RR = 1.42) and transport equipment operators (RR = 1.35). The main limitation of the study was the inability to separate the exposures to diesel exhaust from those of gasoline exhausts since both motor vehicle drivers and transport equipment operators probably were exposed to the exhausts of both types of vehicles. Hayes et al. (1989) combined data from three studies (conducted in three different states) to increase the power to detect an association of lung cancer with different occupations that had high potential for exposure to

diesel exhaust. They found that truck drivers employed for more than 10 years had a significantly increased risk of lung cancer (OR = 1.5, 95 percent CI = 1.1 to 1.9). This study also found a significant trend of increasing risk of lung cancer with increasing duration of employment among truck drivers. These relative odds were computed by adjusting for birth cohort, smoking, and state of residence. The main limitation of this study is again the mixed exposures to diesel and gasoline exhausts, since information on type of engine was lacking. Potential bias may have been introduced since the way in which the cause of death was ascertained for the selection of cases varied in the three studies. The methods used in these studies to classify the occupational categories are different, hence probably leading to incompatibility of occupational categories.

In a case-control study by Steenland et al. (1990) involving truck drivers with at least 35 years of experience, the relative odds ratio was 1.89. This study also showed a dose-response trend with the risk of lung cancer increasing with increasing years of exposure when employment after 1959 was considered. The limitations of this study include possible misclassifications of exposure and smoking, lack of levels of diesel exposure, smaller exposed population, and insufficient latency period.

The most convincing comes from the Garshick et al. (1987) case-control study among railroad workers. After adjustment for asbestos and smoking, the relative odds for continuous exposure were 1.39 (95 percent CI = 1.05 to 1.83). Among the younger workers with longer diesel exhaust exposure, the risk of lung cancer increased with the duration of exposure after adjusting for asbestos and smoking. Even after the exclusion of recent diesel exposure (5 years before death), relative odds increased to 1.43 (95 percent CI = 1.06 to 1.94). This study appears to be a well conducted and well analyzed case-control study with reasonably good power. Potential confounders were controlled adequately, and interactions between diesel exhaust and other lung cancer risk factors were tested.

Of the seven bladder cancer case-control studies, four studies found increased risk in occupations with a high potential diesel exhaust exposure. A significantly increased risk of bladder cancer was found in Canadian railroad workers (RR = 9.0, 95 percent CI = 1.2 to 349.5; in Howe et al., 1980) truck drivers (OR = 2.9, Hoar and Hoover et al., 1985) and in Argentinean truck and railroad drivers (RR = 4.32; Iscovich et al., 1987). Significantly increased risks were observed with increasing duration of employment of ≥20 years in truck drivers (OR = 12) and railroad industry workers (OR = 2.21; Steenland and Burnett, 1987). No significant increased risk was found for any diesel-related occupations in studies by Wynder et al. (1985), Iyer et al. (1990), and Steinbeck et al. (1990). All these studies had several limitations including inadequate characterization of diesel exhaust exposure, lack of validation of surrogate measures of exposure, and presence of other confounding factors (urinary retention, concentrated smoke within the truck cab, etc.); most

of them had small sample sizes, and none presented any latency analysis.

In summary, in regard to lung cancer which is the endpoint used for the EPA unit risk, an excess risk of lung cancer was observed in three out of seven cohort studies and six out of seven case-control studies. Of these studies, two cohort and two case-control studies observed a dose-response relationship using duration of employment as a surrogate for dose. However, because of the lack of actual data on exposure to diesel exhaust in these studies and other methodologic limitations such as lack of latency analysis, the evidence of carcinogenicity in humans is considered to be limited for diesel exhaust exposure.

9.6.1.2 Weight-of-Evidence Judgement of Data and EPA Classification

Based upon the inductions of lung tumors in the three F344 rat studies, as well as the other research mentioned above and supported by positive results for mutagenicity, the evidence for carcinogenicity of diesel exhaust in animals is considered to be sufficient based on U.S. EPA cancer assessment guidelines.

Collectively, the epidemiological studies show a positive association between diesel exhaust exposure and lung cancer. However, because of the uncertainties due to limited exposure data and low relative risk ratios in these populations, the evidence for carcinogenicity of diesel engine emissions in humans is considered to be limited. This means that a causal interpretation is credible, but alternative explanations such as chance, bias, or confounding factors cannot be ruled out.

On the basis of limited evidence for carcinogenicity of diesel engine emissions in humans, supported by sufficient evidence in animals and positive mutagenicity data, diesel engine emissions are considered to best fit the weight-of-evidence category B1. Agents

classified into this category are considered to be probable human carcinogens.

9.6.1.3 Data Sets Used for Unit Risk Estimates

The most critical of the above mentioned animal studies are those that involve a chronic inhalation exposure of diesel particulate matter. To actually determine the unit risk of this particle, only three of the rat inhalation studies are selected for risk calculations because each study consists of multiple exposure groups and thus is more appropriate for risk calculations. The three studies used are Mauderly et al. (1987), Ishinishi et al. (1986), and Brightwell et al. (1986). These studies are summarized in Table 9-8.

The EPA also attempted to use two epidemiological studies, the Garshick et al. studies published in 1987 and 1988 to develop a unit risk estimate. These are summarized in Table 9-9. Though a relationship exists between diesel exhaust exposure and the incidence of lung cancer, both of these studies have strengths and weaknesses. These studies give a large sample size in a relatively stable workforce, and also take into account the confounding factors of smoking and asbestos exposure. The major weaknesses at this time are the limited qualitative and quantitative data on the exposure of these individuals and a short follow-up period. The number of years of exposure to diesel exhaust was used as a substitute for an actual dose so it is difficult to accurately assess the amount of diesel exhaust they were exposed to.

9.6.1.4 Dose-Response Model Used

The linearized multistage model is used to calculate unit risk estimates using various dose equivalence assumptions. All unit risk estimates that currently exist for diesel particulate matter are based exclusively on animal data.

9.6.1.5 Unit Risk Estimates

The approach that has been adopted by EPA in determining the unit risk from diesel particulate matter is the one that attributes the carcinogenicity to that of the particle itself rather than the
organics. The methodology used to develop the most recent EPA quantitative risk estimate differs from other chronic bioassay based estimates in several ways. Unlike earlier estimates, the present one uses a sophisticated dosimetry model to extrapolate lung burdens of particulate matter from animal exposures to humans. This model accounts for species differences in deposition efficiency, respiration rates, normal particle clearance rates, particle transport to lung associated lymph nodes, and effect of particle overload upon clearance rates.

TABLE 9-8. Animal Data Used for EPA's Unit Risk Estimates.

REPORT	ANIMAL	PARTICLE EXPOSURE CONCENTRATION AND TIME OF EXPOSURE	ENGINE TYPE AND CYCLE	MAJOR RESULTS
Mauderly, et al. (1987) (Lovelace)	F344/Crl rats, male and female	0.35, 3.5, and 7.0 mg/m^3 7h/d, 5d/week, for 30 mo.	1980 model 5.7L Oldsmobile V8 run at FTP hot start certification cycle	Control and low level exposure shows no statistical increase in lung tumors (0.9% and 1.3% increase in tumors respectively) Medium and high level exposure shows that the lung tumor incidence was statistically higher (3.6% and 12.8% increase in tumors respectively)
Ishinishi, et al. (1986) (JARI)	F344/Jcl rats, male and female	0, 0.1, 0.4, 1.0, or 2.0 mg/m^3 from light duty engine 0, 0.4, 2.0, or 4.0 mg/m^3 from heavy duty engine 16h/d, 6d/week, for 30 mo.	light duty engines were 1.8L-4 cylinder, swirl chamber operated at 1200 rpm heavy duty engines were 11L-6 cylinder, direct injection operated at 1700 rpm	Light duty engine exposure: carcinomas were dose-dependent with the highest incidence in the 1.0 mg/m^3 exposure (4.1% increase), there were no significant changes between groups Heavy duty engine exposure: carcinomas were dose-dependent with the highest incidence in the 4.0 mg/m^3 exposure (6.5% increase), significant changes were found between the 0 and 4.0 mg/m^3 exposure groups
Brightwell, et al.(1986) (Battelle-Geneva)	F344 rats, male and female pretreated 3d prior to exposure with tumor promoter	0.7, 2.2, or 6.6 mg/m^3, filtered and unfiltered 5-16h periods/wk over 2 yrs.	1.5L engine (no manufacturer given) using U.S. 72 (FTP) cycle	No significant increase was found in either group at low or control exposure Medium, unfiltered exposure 4-15% increase, and high, unfiltered exposure 23-54% increase in lung tumors Dose-dependent, promoter had no statistical effect

Table 9-9. Epidemiological Data Used for EPA's Unit Risk Estimates.

STUDY	TYPE AND SUBJECTS	EFFECTIVE START	PARAMETERS OBSERVED	MAJOR RESULTS

Garshick, et al. (1987)	Case-Control study on male railroad workers each cancer death (case) was matched to two randomly selected deceased workers (within 31d of death) with no evidence of cancer (control)	1959 (95% of locomotives were diesel)	Death from lung cancer between March 1, 1981 through February 28, 1982	Relative odds for lung cancer is 1.5 for the highest exposure category (low odds but 95% confidence interval is narrow) and it has been adjusted for smoking and asbestos exposure. This study supports the hypothesis that occupational exposure to diesel exhaust increases lung cancer.
Garshick, et al. (1988)	Cohort study on male railroad workers (a group of individuals having a statistical factor in common in an epidemiological study [i.e. diesel exposure])	same as above	Deaths due to lung cancer from 1959 to 1980	Relative risk for lung cancer is 1.5 (modest risk) and it has been adjusted for smoking and asbestos exposure. This study shows a positive association between occupational diesel exhaust exposure and a modest increase in lung cancer

A important feature of the dosimetry model is that it accounts for high dose inhibition of particle clearance. If this adjustment is not made, lung burden of particulate matter will be overestimated during extrapolation to low doses with an accompanying overestimation of cancer potency. Since most of the organics desorb from the particle surface even with normal clearance rates, inhibition of particle clearance will affect the concentration of organics only slightly. Cancer potency estimates may

therefore differ depending on whether they are based upon lung burden of particles or organics.

There are two reasons for using a particle based risk assessment. First of all, the concentration of PAH's on the particles is quite small. The quantity present at the exposure levels used in the chronic bioassays is unlikely to be great enough to produce the tumor response seen. B[a]P is present at a concentration of about 1 µg/gm particulate matter. Secondly, insoluble biochemically inert particles such as titanium dioxide (Lee et al., 1986) or activated carbon (Kawabata et al., 1986) can induce lung cancer at very high concentrations. Even more significant were the findings of Mauderly et al. (1991) and Heinrich et al. (1991) (see Section 9.6.3.2) that carbon black, which is very similar to the carbon core of the diesel particle, but contains essentially no adsorbed organics, induced lung cancer at the same concentrations as diesel exhaust. Additional support for the predominance of the particle effects was also contained in the report by Heinrich et al. (1991). In this report, pyrolyzed pitch condensate, which does not have an insoluble particle core, but contains about 1000 fold greater concentration of PAHs than diesel particles, is not that much more potent than diesel exhaust in the induction of lung cancer.

While this method is an improvement over previous ones, an important uncertainty remains. Particles deposited in the alveolar regions are ingested by macrophages, which are then induced to secrete a variety of cytokines, oxidants, and proteolytic enzymes. Some combination of these are thought to act upon adjacent alveolar cells to induce tumor formation. It is uncertain if very low macrophage particle burdens will induce release of these factors, or if there is a threshold for their effects. Use of a linearized multistage model to extrapolate to low doses could result in an overestimate of risk. Data, however, are presently inadequate to prove or disprove this possibility. Thus, EPA still employs the conservative linearized low-dose extrapolation model.

The unit risks based on the long term rat inhalation studies of Mauderly et al., (1987), Ishinishi et al., (1986), and Brightwell et al., (1986) were calculated by EPA using the carbon core only. The availability of preliminary data from studies discussed in Section 9.6.3.2 conducted on animals exposed to carbon black, though not used in the risk calculations, did influence the methodology. A geometric mean of the three unit risks was then

determined to be 1.7×10^{-5} $(\mu g/m^3)^{-1}$. This unit risk was presented at the Air and Waste Management Association meeting in October, 1991 (Pepelko and Ris, 1991d). This unit risk is also presented in the latest EPA diesel draft document (unpublished). It has yet to undergo Science Advisory Board (SAB) review and thus is subject to change. EPA is also in the process of developing a unit risk estimate that attempts to account for both particle and organic effects.

Pepelko and Ris (1991d) also discussed the attempt to develop a unit risk estimate for lung cancer based on human epidemiological data using Garshick et al., (1988). Using data from this study, the 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.

9.6.2 Other Views and Unit Risk Estimates

This section presents alternative views and/or risk assessments for diesel exhaust particulate matter. These alternative risk assessments are summarized in Table 9-10.

<u>Comparative Potency Method</u>

The comparative potency method is a method developed by EPA to predict human cancer risk from mutagenicity and animal bioassay data. The comparative potency method was developed because of a lack of chronic animal bioassays and a need to develop a potency estimate in the early 1980's. It has been applied to the polycyclic organic matter (POM) from selected emission sources, including diesel vehicles (Albert et al., 1983; Lades, 1991). POM is a general term referring to a complex mixture of polycyclic aromatic compounds generally associated with the particles or soot of emissions, and derived from the combustion of fossil fuels, vegetative matter, and synthetic chemicals.

In this comparative potency method, the risk of diesel particulate matter is estimated by comparison of diesel particulate matter bioassay potencies to the bioassay potencies of known human carcinogens (coke oven, roofing tar, cigarette smoke) according to the following equation:

Table 9-10. Comparison of Diesel Exhaust Particulate Matter
Inhalation Unit Risk Estimates.

Source	Method	Cancer Unit Risk Estimate $(\mu g/m^3)^{-1}$ Upper Bound[a]
Albert et al. (1983) Lewtas (1991)	Comparative potency method using extracted organics from one light-duty (LD) diesel engine	3.5×10^{-5}
Albert et al. (1983) Lewtas (1991)	same as above, using an average of three LD engines	2.6×10^{-5}
Harris (1983)	Comparative potency method[b]	2.9×10^{-4}
Cuddihy et al. (1984)	Comparative potency method[b,c]	7.0×10^{-5}
Albert and Chen (1986)	Multistage model, lung cancer in rats[d]	1.2×10^{-5}
Pott and Heinrich (1987)	Straight line extrapolation, lung cancer in rats[e]	$6.0-12.0 \times 10^{-5}$
McClellan et al. (1989)	Logistic regression, lung cancer in rats[f]	8.0×10^{-5}
Smith and Stayner (1990)	Time-to-tumor model, lung cancer in rats[d]	$1.5-3.0 \times 10^{-5}$
Harris (1983)	Epidemiological analysis, London Transport Study (Waller, 1981)	4.1×10^{-3}
McClellan et al. (1989)	Epidemiological analysis, Railroad workers (Garshick et al. 1987)	$0.6-2.0 \times 10^{-3}$

[a]Estimated upper bound of lifetime risk of continuous exposure to 1 $\mu g/m^3$ diesel exhaust particulate matter.
[b]Used data from studies by Albert et al. (1983).
[c]Used data from studies by Harris (1983).
[d]Used data from studies by Mauderly et al. (1987).
[e]Used data from studies by Brightwell et al. (1986), Heinrich et al. (1986a), and Mauderly et al. (1987).
[f]Used data from studies by Brightwell et al. (1986), Ishinishi et al. (1986), Iwai et al. (1986), and Mauderly et al. (1987).

$$unit\ risk\ (diesel) = unit\ risk\ (known\ carc.) \left[\frac{bioassay\ potency\ (diesel)}{bioassay\ potency\ (known\ carc.)} \right]$$

The term in brackets is the ratio of the slopes of the dose responses from the same bioassay, and is referred to as the relative potency. The underlying assumption of the comparative potency method is that the relative potency is constant across different bioassay systems. The equation above was applied using the extract of a light duty Nissan engine in the mouse skin tumor initiation bioassay to estimate the risk of diesel particulate matter. The mouse skin tumor initiation bioassay was chosen because the relative potencies of the known human carcinogens obtained with this bioassay correlated well with the relative potencies obtained with the human data. The mouse skin tumor test was also used because it gave a strong dose-response in the Nissan engine.

Extracts from particle samples from three light-duty diesel vehicles and one heavy-duty diesel engine were used. The unit risk estimates for two other light duty engines and a heavy duty engine were derived by comparing their potencies with that of the Nissan engine using three short-term tests. The average lifetime risk from the three light-duty diesel samples across the three comparative human carcinogens was $2.3 \times 10^{-4} (\mu g$ organic matter$/m^3)^{-1}$ or $2.6 \times 10^{-5} (\mu g$ particles$/m^3)^{-1}$. The lifetime cancer risk$/\mu g$ particles$/m^3$ ranged from 1.8×10^{-6} for the heavy duty engine, to 3.5×10^{-5} for the most potent light duty diesel engine (Albert et al., 1983; Lewtas, 1991).

The comparative potency method predicted a human lung cancer unit risk very similar to the unit risk estimate for diesel particulate matter that has been recently extrapolated from three rodent inhalation studies. The lifetime unit risk for the rodent studies is the same one cited earlier, $1.7 \times 10^{-5} (\mu g/m^3)^{-1}$. This compares to $2.6 \times 10^{-5} (\mu g/m^3)^{-1}$ by the comparative potency method. This demonstrates that these two independent approaches to cancer risk from diesel emissions result in very similar cancer unit risk estimates.

Harris (1983) developed comparative potency estimates for the same four engines used by Albert et al., (1983) but used only two epidemiological based potency estimates, those for coke ovens emissions and for roofing tar. Harris (1983) also used preliminary data for three of the same assays as did Albert et al., (1983). After making adjustments to adjust for lifetime exposure the Harris (1983) overall estimated unit risk value was $2.9 \times 10^{-4} (\mu g/m^3)^{-1}$ for the three light-duty engines.

Cuddihy et al. (1984) reported a unit risk of about 7.0×10^{-5} $(\mu g/m^3)^{-1}$ using a comparative potency method similar to those reported in the preceding paragraphs. The data base was similar to that used by Albert et al. (1983) and Harris (1983).

The comparative potency method suffers from two major uncertainties. The first being that mutagenicity is not a reliable predictor of carcinogenicity. Secondly, the relative cancer potency of diesel to the other agents used may be much different than relative potency in short-term.

Alternate Risk Estimates Derived From Rat Data

With the availability of chronic cancer bioassays, more recent assessments were based on lung tumor induction in rats. Albert and Chen (1986) reported a risk estimate based upon the chronic rat bioassay conducted by Mauderly et al. (1987). Using a multistage model and assuming equivalent deposition efficiency in humans and rats, they derived an upper bound for a lifetime risk of 1.2×10^{-5} $(\mu g/m^3)^{-1}$. Pott and Heinrich (1987) used a linear extrapolation, including data reported by Brightwell et al. (1986), Heinrich et al. (1986a), and Mauderly et al. (1987). They reported risk estimates of 6.0×10^{-5} to 12.0×10^{-5} $(\mu g/m^3)^{-1}$. Most recently, Smith and Stayner (1990), using a time-to-tumor model based on the data of Mauderly et al. (1987), derived and upper bound of 1.5×10^{-5} to 3.0×10^{-5} $(\mu g/m^3)^{-1}$. In McClellan et al. (1989) a logistic regression was used with the data from Brightwell et al. (1986), Ishinishi et al. (1986), Iwai et al. (1986), and Mauderly et al. (1987) to derived a unit risk estimate of 8.0×10^{-5} $(\mu g/m^3)^{-1}$.

Alternate Risk Estimates Derived From Epidemiological Data

Harris (1983) also assessed the risk of exposure to diesel engine emissions using data from the London Transport Worker Study by Waller (1981). Five groups of employees from the study were used (one high exposure, two intermediate exposure, and two with no exposure. Harris (1983) compared the exposed groups with internal controls. He merged the three exposed groups and compared them with the two groups considered to be unexposed. An adjustment was made for the greatest exposure groups. Using this method, the relative risk of the exposed groups was greater than 1, but was statistically significant for only the highest exposure groups from 1959 to 1960.

Harris (1983) identified a variety of uncertainties in the assessment. Taking the uncertainties into account, he derived a maximum likelihood estimate of 1.0×10^{-3} $(\mu g/m^3)^{-1}$ and a upper bound of 4.1×10^{-3} $(\mu g/m^3)^{-1}$.

McClellan et al. (1989) developed risk estimates based on the Garshick et al. (1987) study in which lung cancer in railroad workers was evaluated. Using a proportional risk model the values
of a lifetime risk of exposure to diesel exhaust ranged from 0.6×10^{-3} to 2.0×10^{-3} $(\mu g/m^3)^{-1}$.

Environ

The Environ report (Environ, 1987), prepared for the Motor Vehicle Manufacturers Association, was in response to the EPA report on air toxics (Carey, 1987). In its report, EPA uses a range of potency estimates, 2.0 x 10^{-5} to 1.0 x $10^{-4}(\mu g/m^3)^{-1}$, for estimating the lung cancer risk from diesel exhaust. The range of potencies all involved use of the comparative potency method, described above. This Environ (1987) report as well as Carey (1987) were completed while animal inhalation studies were still in progress, so neither report evaluates more recent data.

Environ, for several reasons, questions the validity of the comparative potency approach. Although there is some evidence that activity in short-term genotoxicity tests may be indicative of carcinogenic potential, Environ cites studies that show the correlation between genotoxicity and carcinogenicity was just 60%. Also, quantitative correlations have not been established for any individual carcinogens let alone a complex mixture such as diesel particles. Environ believes this renders the procedure scientifically unsound.

Environ raises further questions regarding the validity of the procedure by challenging the fact that it is based solely on extracts of diesel particles and ignores substances in the emissions that are not associated with the particle or are not extractable. Environ also states that organics adsorbed onto the particle may not be bioavailable so using extract overestimates the potency.

Environ states that these factors may increase the uncertainty associated with the Carey (1987) risk estimates for diesel emissions. They then offer no new or additional data analysis to support their claims or a unit risk estimate of their own.

International Agency for Research on Cancer (IARC)

IARC (IARC, 1989), does not estimate potencies for carcinogens, but has classified diesel engine exhaust into cancer weight-of-evidence Category 2A. Agents classified into Category 2A are considered to be *probable* human carcinogens. This classification is based on limited evidence for carcinogenicity in humans. This is supported by sufficient evidence for carcinogenicity in animals with whole diesel engine exhaust and in animals with extracts of the diesel engine exhaust particles. IARC considers the evidence for the carcinogenicity in animals of the gas-phase of diesel engine exhaust (with particles removed) to be inadequate. EPA did not develop separate weight-of-evidence evaluations for the gaseous and particle phases of diesel exhaust.

National Institute for Occupational Health and Safety (NIOSH)

In 1986, NIOSH evaluated the health effects of diesel exhaust in Evaluation of the Potential Health Effects of

<u>Occupational Exposure to Diesel Exhaust in Underground Coal Mines</u>. This document describes the short-term effects as well as stating that there is a causal association between exposure to whole diesel exhaust and cancer.

In a later publication, NIOSH (1988), the recent animal studies in rats and mice discussed previously in Section 9.6.1 (Brightwell et al. 1986; Heinrich et al. 1986; Ishinishi et al. 1986; Iwai et al. 1986; Mauderly et al. 1987) are used to confirm an association between the induction of cancer and exposure to whole exhaust. The lung is the primary site identified with carcinogenic or tumorigenic responses following inhalation exposures. Limited epidemiological evidence (Edling et al. 1987; Garshick et al. 1987, 1988) suggests an association between occupational exposure to diesel engine emissions and lung cancer. The consistency of these toxicologic and epidemiologic findings suggests that a potential occupational carcinogenic hazard exists in human exposure to diesel exhaust.

9.6.3 Recent and Ongoing Research

9.6.3.1 Metabolism and Pharmacokinetics

Much of the information in this section is summarized from the more detailed report, the Draft <u>Health Assessment Document for Diesel Emissions</u> (EPA 1990b). To examine this information further please refer to the document mentioned above.

Several studies affirm the bioavailability from inhaled diesel exhaust particles of compounds such as B[a]P and 1-nitropyrene (1-NP) which are known to be carcinogenic or mutagenic. Biotransformation of B[a]P, 1-NP, and some of the dinitropyrenes to reactive intermediates following inhalation of diesel exhaust particles has been verified. Furthermore, several reports have provided data indicating the formation of DNA adducts, considered an underlying mechanism of carcinogenicity, following administration of these compounds. The development of lung tumors in experimental laboratory animals following chronic exposures to particulate diesel exhaust occurs under conditions in which alveolar macrophage-mediated particle clearance from the lung is compromised. Although tumors have also been found to develop with other types of particles (e.g., titanium oxide) when this clearance mechanism is diminished, tumors developing in the lungs of diesel emissions-exposed rats with smaller lung mass or comparatively less volume burden of diesel particles suggest that the carcinogenic response is not exclusively related to an overabundance of the particles in the lungs *per se*. Therefore, the organic components on diesel particles may be importantly involved in the development

of lung tumors. The lung's pulmonary macrophages, which phagocytize deposited diesel particles, probably participate in the gradual *in situ* extraction and metabolism of procarcinogens associated with the diesel particles. Additionally, the normal tumoricidal activities of the pulmonary macrophages may be

compromised upon interaction with excessive numbers of diesel particles, and diesel particle-macrophage interactions could lead to the generation of reactive oxygen species that have been shown to be at least mutagenic. Alternatively, there is evidence that particles with a very large surface area/unit volume, such as diesel particles or carbon black, can stimulate production of harmful products by macrophages at much lower lung burdens, perhaps even at lung burdens insufficient to inhibit clearance. Processes and potential mechanisms discussed herein have largely been derived from animal data, and further research is required to determine how the activities of human pulmonary macrophages in response to particulate diesel exhaust compare with pulmonary macrophages from experimental animals. Most importantly, valid dosimetry for the human condition will require the elucidation of the underlying mechanisms involved in the development of lung tumors following chronic exposure to whole diesel exhaust.

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 is multiphasic and involves several processes including a relatively rapid mucociliary transport and slower 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 B[a]P and 1-NP. Experimental data also indicate alveolar macrophage-mediated metabolism and phagolysosomal solubilization of particle-adsorbed components. Although macromolecular binding of diesel exhaust particle-derived PAH 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.

In addition to the aforementioned points, one must also consider the fact that other compounds (e.g., gas-phase chemical irritants) may alter respiratory rate and, therefore the actual inhaled dose of potentially toxic components. Moreover, a better knowledge of particle dissolution rate and particle removal rate is necessary for more accurately assessing bioavailability of potentially carcinogenic components of diesel exhaust.

9.6.3.2 Carcinogenicity - Animal Studies

In a recent study by the Fraunhofer Institute (Heinrich et al. 1991) female Wistar rats were exposed to carbon black (CB), tar/pitch condensate (yielding PAH in the form of benzo(a)pyrene [BaP]), and mixtures of these compounds to assess their carcinogenic effect. Exposure time for all groups was 17 hrs/day, 5 days/week, for 10 and 20 month periods. A 17% increase in lung tumors incidences was reported following exposure of rats to carbon black particles for 10 months at a concentration of 6 mg/m^3, then clean air for the remainder of their lifetime. This is very close to the TLV value. The tar/pitch condensates (BaP) at 20 or 50 µg/m^3 gave increases in lung tumors of 4% and 39% at 10 months and 33% and 97% at 20 months, respectively. The mixture (2 or 6 mg/m^3 CB and 50 µg/m^3 BaP) test results were only for the 10 month period, and the increases ranged from 72 to 89%. In the abstract submitted, the authors claim carbon black in this experiment induced almost the same lung tumor rates in rats as diesel soot did in Heinrich et al. (1986a). No inferences were made concerning the greater increases in tumor incidences with pitch plus carbon black than with carbon black alone. This study shows that particles alone, devoid of organics are capable of inducing lung cancer. The study also shows that the effects of particles are enhanced by the presence of BaP. It should be noted, however, BaP concentrations in this study were much greater than those present on diesel particles.

In a study by Pott et al. (1991), also from the Fraunhofer Institute, lung tumors were observed in female Wistar rats intratracheally instilled with various non-fibrous and fibrous dusts. The percent of rats with primary lung tumors ranged from a 60 to 66% increase following exposure to 30 to 60 mg diesel soot (two types) or carbon black. The rats exposed to 45 mg of one type of diesel soot and those exposed to 45 mg of carbon black gave identical rates of 65%. This appears to support the contention that the carbon particle itself is the carcinogen. The abstract does not detail the differences in the two diesel soot types tested, nor does it provide data regarding the amounts of types of particle bound organics.

In a recent and as yet uncompleted Health Effects Institute study by Mauderly et al. (1991) of the Inhalation Toxicology Research Institute (ITRI), there was a direct comparison of carbon black particles and diesel exhaust. The study exposed F344/N rats 16 hours/day, 5 days/week for 24 months to carbon black (2.5 or 6.6 mg/m^3) or diesel exhaust (2.4 or 6.4 mg/m^3). They were sacrificed at 3, 6, 12, 18 and 23 months with remaining rats held for post-exposure observation. The results at this time are interim, but the responses to diesel exhaust and carbon black were qualitatively similar. Diesel exhaust caused a greater response than carbon black in lung weight (increased), lung burden of retained particles, and lung inflammation and cytotoxicity. Diesel exhaust and carbon black caused approximately similar responses in body weight (decreased), lymph node burden of retained particles and mortality. The numbers of

lung tumors observed grossly at necropsy were nearly identical
for diesel exhaust and carbon black. Observations to date do not
suggest that there is a difference between diesel exhaust and
carbon black in lung tumor type, multiplicity or growth in nude
mice. Mauderly et al. (1991), in the interim, concludes that the
information at this time suggest that soot-associated organic
compounds do not play a significant role in the pulmonary
carcinogenicity of diesel exhaust in rats.

Dr. Werner Stober of the Fraunhofer Institute of Technology
and Aerosol Research, whose research is funded in part by the
German automobile manufacturers, states his position in several
papers. This position is outlined in Stober (1987, 1989) and
more recently by Stober (1991), in which the present
epidemiological data, comparative potency method, and animal
inhalation studies are evaluated.

A search of the scientific literature for data on the health
effects (especially carcinogenic risk) of inhaled diesel
emissions was performed. Dr. Stober states that this search
provides some very weak and disputed epidemiological evidence
suggesting that, at certain occupational exposures, there may
have been a health hazard for certain workers. The major
confounding factor in all of these investigations is the
influence of the cancer statistics of cigarette smoking. He
states that it is most likely the residual effects which are
attributed to occupational diesel exposure are due to surrogate
and incomplete information about the smoking habits of the
cohorts. He finds it interesting that the supporters of
occupational risk from diesel emissions do not propose a risk to
the general populations at the present level of diesel emissions.

Stober (1989) also states that the labeling of diesel
exhaust as a potential or probable carcinogen by Germany, the
World Health Organization (IARC), and the U.S. EPA were made
without any reference to the evaluation of risk. He states that
the risk determined by the epidemiological studies should not be
used for the general public. The risk for the public at large,
at present levels of diesel emissions, is actually non-existent,
according to Dr. Stober. He compares the lifetime carcinogenic
risk from 1 $\mu g/m^3$ diesel emissions to the risk of being struck by
lightning in Germany (a lifetime risk of 2:100,000 or 2.0×10^{-5}).

Stober (1989) does concede that if a genotoxic mechanism can
be shown to play a significant role in experimental tumor
induction, then a small residual risk may be assumed to have been
obscured by the uncertainties of past epidemiological studies.
In that case, proper development and implementation of target
control strategies is advisable to lower the cancer risk.

Stober (1989) states further that he does not imply that it is unnecessary to regulate diesel exhaust emissions today. The future growth of unregulated diesel-powered vehicles would degrade the present particle levels, and the general public will resent this deterioration. But there is only a very low probability, if any, that this issue involves more than an insignificantly small residual risk of a health effect.

In Dr. Stober's Air and Waste Management Association presentation in October, 1991 (Stober 1991), he mentions several points regarding the limitations of the rat studies. At present, the rat data suggest that a threshold may exist for the exposure to diesel particulate matter and the appearance of tumors. He goes on to further state that the particle overload at the two highest levels where the tumors occurred is also an issue. Using the preliminary data from recent ITRI and Fraunhofer research (see Section 9.6.3 for details), he states that it shows qualitatively similar results or more pronounced responses from carbon black than diesel particulate matter. He proposes a possible epigenetic mechanism of tumor induction (i.e., the tumors have not been caused by a metabolic degradation of organic matter associated with the particles, but by the particle deposits itself).

9.7 Carcinogenic Risk

Urban and rural diesel particulate matter carcinogenic risks, expressed as annual cancer deaths, were calculated following the methodology discussed in Section 4.1, based on the HAPEM-MS exposure estimates from Section 9.5.1 and the EPA unit risk estimate given in Section 9.6.1.5. The resultant urban, rural, and total cancer deaths are given in Table 9-11. These cancer incidences are upper bound estimates and the risk may be less, but is unlikely to be more.

Table 9-11. Diesel Particulate Matter Cancer Deaths.[a,b]

Year	Urban	Rural	Total
1990	92	17	109
1995	56	10	66
2000	33	6	39
2010	23	4	27

[a]Projections have inherent uncertainties in emission estimates, dose-response, and exposure.
[b]Cancer deaths are based on the EPA 1991 draft unit risk, determined using animal data. This unit risk has not been peer reviewed and is subject to change.

9.8 Non-carcinogenic Effects of Inhalation Exposure to Diesel Particulate Matter

Since the focus of this report is on the carcinogenic potential of the various compounds, the noncancer information will be dealt with in a more cursory fashion. No attempt has been made to synthesize and analyze the data encompassed below. Also, no attempt has been made to accord more importance to one type of noncancer effect over another. The objective is to research all existing data, describe the noncancer effects observed, and refrain from any subjective analysis of the data.

Diesel Particulate Matter

The symptoms of acute (short) exposure to high levels (i.e. above ambient) of diesel exhaust have been detailed through the study of occupationally exposed workers. These workers include underground miners, bus garage workers, dock workers, and locomotive repairmen. The symptoms may be manifested as one or more of the following: mucous membrane and eye irritation, headache, light-headedness, nausea, vomiting, heartburn, weakness, numbness and tingling in extremities, chest tightness, and wheezing. The odors associated with diesel exhaust emissions also cause some effects, such as nausea, headache, and loss of appetite.

Even though this appears to be a formidable list of symptoms, the effects of a short-term diesel exhaust exposure are dissipated as soon as the exposure stops or the subject leaves the area. Any of the changes in respiratory symptoms and pulmonary function over the course of a workshift were generally found to be minimal.

The chronic (long-term) exposure to diesel exhaust emissions have also been followed in occupationally exposed workers, but the data are insufficient to make a correlation between the effects and the exposure experienced. Most of the chronic exposure data are derived from the use of animal studies.

Many of the changes observed in rats and other small animals exposed to diesel exhaust affect the cellular and structural make-up of the lung. These effects include: accumulation of particles in the lungs, increased lung weight, tissue inflammation, increased macrophages and leukocytes (white blood cells), macrophage aggregation, hyperplasia (excess cell formation) in the alveolar and bronchiolar epithelium (surface cell layer), and a thickening of alveolar septa (partitions). In some studies, a reduction in the growth of animals is also observed at 2 mg/m^3 for 16h/day, and alterations of pulmonary function parameters were seen at 2 to 6 mg/m^3.

All of these changes appear to be dependent on the concentration of the exhaust particulate matter, the pulmonary deposition of the particle, and the ability of the lung to clear the particulate matter.

Reference Concentration for Chronic Inhalation Exposure (RfC)

The reference concentration for chronic inhalation exposure (RfC) for diesel particulate matter has recently been established (EPA, 1993). This RfC was determined to be 5.0×10^{-3} mg/m^3 per day, over a lifetime. 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 two critical studies used in determining the diesel particulate RfC are chronic rat inhalation studies by Mauderly et al. (1998) and Ishinishi et al. (1988). These two studies observed various non-cancer endpoints and at various time points.

In Mauderly et al. (1988), rats and mice were exposed to target diesel particulate matter concentrations of 0, 0.35, 3.5, or 7.0 mg/m^3 for 7 hours/day, 5 days/week for up to 30 months for the rats or 24 months for the mice. Endpoints examined in this study include carcinogenicity, respiratory tract histopathology and morphometric analysis, particle clearance, lung burden of diesel particulate matter, pulmonary function testing, lung biochemistry, lung lavage biochemistry and cytology, immune function, and lung cell labeling index. Aggregates of particle-laden macrophages were seen after 6 months in rats exposed to 7.0 mg/m^3 target concentrations and, after 1 year of exposure, histological changes were seen including focal areas of epithelial metaplasia. Fibrosis and metaplasia increased with increasing duration of exposure and were observable in the 3.5 and 7.0 mg/m^3 group of rats at 24 months. In the 0.35 mg/m^3 group of rats, there was no inflammation or fibrosis.

A NOAEL of 0.353 mg/m^3, based on inflammatory, histological, and biochemical changes in the lung and impaired particle clearance was established. A LOAEL of 3.47 mg/m^3 based on the chronic rat inhalation study described above was also developed. To establish a human equivalent NOAEL and LOAEL for this study a conversion factor was applied. The human equivalent concentrations (HEC) were estimated from the experimental conditions using the particle retention model of Yu and Yoon (1990) assuming a continuous human exposure and using mass of diesel particle carbon core per unit of surface area in the pulmonary region as the dose expression. Using this conversion factor the NOAEL(HEC) = 0.042 mg/m^3 and the LOAEL = 0.36 mg/m^3.

In Ishinishi et al. (1986, 1988) both light-duty and heavy-duty diesel engines were operated under constant velocity and load conditions. Particle concentrations were 0.11, 0.41, 1.18, and 2.32 mg/m^3 for the light duty engine and 0.46, 0.96, 1.84, and 3.72 mg/m^3 for the heavy duty engines. Fischer 344 rats were exposed for 16 hours/day, 6 days/week, for 30 months. No histopathological changes were observed in the lungs of rats exposed to 0.4 mg/m^3 particulate matter or less. At concentrations above 0.4 mg/m^3 particulate matter, accumulation of particle-laden macrophages was observed. Hyperplastic lesions were reported at a lowest observed adverse effect level (LOAEL)

in chronically exposed rats at 1.18 mg/m^3 for light duty and 0.96 mg/m^3 for the heavy duty series.

A NOAEL of 0.46 mg/m^3, based on histological changes in the rat lung exposed to heavy duty diesel emissions was established. A LOAEL of 0.96 mg/m^3 based on the chronic rat inhalation study described above was also developed from the heavy duty emissions exposure. As described previously, to establish a human equivalent NOAEL and LOAEL for this study a HEC conversion factor must be applied. Using this conversion factor the NOAEL(HEC) = 0.155 mg/m^3 and the LOAEL = 0.30 mg/m^3.

To develop an RfC, the no-observable-adverse-effect level (NOAEL) of 0.46 mg/m^3 from the Ishinishi et al. (1986, 1988) study was converted to continuous lifetime exposure conditions [0.46 mg/m^3 × 16/24 hours/day × 6/7 days/week = 0.26 mg/m^3 NOAEL(ADJ). The NOAEL(ADJ) from the heavy duty emissions was adjusted based on the retention model Yu and Yoon (1990) to achieve a NOAEL(HEC) of 0.155 mg/m^3. The NOAEL(HEC) of 0.155 mg/m^3 had an uncertainty factor of 30 that was then applied to derive the RfC of 5.0×10^{-3} mg/m^3. The factor of 30 reflects a factor of 10 to protect sensitive individuals and 3 to adjust for interspecies extrapolation because dosimetric adjustments based on a particle deposition and retention model were applied (Yu and Yoon, 1990). The confidence in the RfC is high. The research programs performed were well conducted chronic studies with adequate numbers of animals and identified LOAELs and NOAELs at levels which are consistent across studies. The full data base contains 10 chronic studies as well as developmental and reproductive studies, resulting in high confidence.

PM$_{10}$

The following studies cited from the Federal Register (EPA, 1987) are those that were utilized to establish a final National Ambient Air Quality Standard (NAAQS) for PM$_{10}$ (particulate matter less than 10 microns in diameter) with a substantial margin of safety. These non-cancer inhalation human studies apply to PM$_{10}$ in general and are applicable to both diesel and gasoline particles.

To evaluate the short term effects of PM$_{10}$, the studies of Dockery et al. (1982) and Dassen et al. (1986) were useful. The Dockery study observed physiologically small but statistically significant decreases in lung function in a group of children exposed to peak PM$_{10}$ levels of 140-250 µg/m^3. The decrements persisted for 2-3 weeks following the exposures. The study also suggested the possibility of larger responses in a subset of the children, including those with existing respiratory symptoms. The Dassen study recorded similar decrements in children in the Netherlands following exposure to PM$_{10}$ levels estimated at 200-250 µg/m^3, but no observable effects two days after exposure to PM$_{10}$ levels estimated at 125 µg/m^3.

Long term examination of respiratory health in the same community studied by Dockery et al. (1982) suggests that the

children in that community have a higher incidence of respiratory illness and symptoms than children in communities with lower particle levels.

The most important study of long-term effects is an examination of six U.S. cities (Ware et al., 1986). The study indicates the possibility of increased respiratory symptoms and illnesses in children at multi-year levels across a range of 40 to over 58 $\mu g/m^3$ as PM_{10}, but found no evidence of reduced lung function at such concentrations. This study did not find similar gradients in symptoms and illness within some of the cities, which had somewhat smaller localized pollution gradients. The results of a separate series of studies on long and intermediate term (2 to 6 weeks) exposures in a number of U.S. metropolitan areas (Ostro, 1987; Hausman et al., 1984) are more supportive of the possibility of effects within cities (respiratory related activity restrictions in adults) at comparable U.S. exposure levels. The results of these more recent studies are generally consistent with the earlier U.S. studies. In particular, the findings of symptomatic responses in children with no change in lung function (Ware et al., 1986) is consistent with similar findings in adults (Bouhuys et al., 1978) at estimated long-term PM_{10} levels down to 50 $\mu g/m^3$. However, the information available to support the existence of significant adverse effects at annual PM_{10} levels below 50 $\mu g/m^3$, especially when 24 hour levels are maintained below 150 $\mu g/m^3$, is quite limited and uncertain.

To add some perspective to the levels of PM_{10} that are produced by various vehicle types, automobile with diesel engines can emit up to 100 times greater volume of particulate matter per mile than gasoline engine cars burning unleaded gasoline (Lewtas, 1991). When directly comparing the mutagenic emission rates for a number of certification vehicles, it was found that the diesel vehicles emitted 45 to 800 times as much mutagenic activity per mile as the gasoline catalyst-equipped vehicles (Claxton and Kohan, 1981). It should be noted, however, while gasoline engine automobiles produce a much smaller volume of particulate matter, they greatly outnumber diesel vehicles.

Since the establishment of the PM_{10} standard, a number of new epidemiological studies seem to indicate that particulate matter might influence daily mortality rates at concentrations lower than the ranges encountered in the earlier studies. In particular, studies that examined particulate matter pollution in Philadelphia, PA (Schwartz and Dockery, 1992a), Steubenville, OH (Schwartz and Dockery, 1992b), and Detroit, MI (Schwartz, 1991), found that in each case the relative risk of daily mortality increases in a generally linear fashion with increasing concentrations of particulate matter. In some cities, the association was seen between particulate matter and mortality even when particle levels never violate the current standard. All the studies (Schwartz and Dockery, 1992a,b; Schwartz, 1991) emphasize the lack of an apparent threshold, and that particulate matter may be influencing mortality even at levels well below the current standard of 150 $\mu g/m^3$.

Determining the specific causes of death which seen to be exacerbated by particulate matter pollution is more difficult, but the Philadelphia study found significant increases in death from chronic obstructive pulmonary disease and cardiovascular disease, with the large majority of death occurring in individuals aged 65 and older.

In two studies of a Western city, Provo, UT, it was also found that an increase in PM_{10} correlated with specific health indicators (Ransom and Pope, 1991) and school absenteeism (Pope et al., 1991). Particulate matter pollution in Provo, UT has been found to be associated with increased daily elementary school absenteeism, increased hospital admissions for respiratory disease, increased respiratory symptoms, and increased medicine used by asthmatics. The impact of particulate matter on the endpoints mentioned above occurred at levels well below the current 24 hour NAAQS standard of 150 $\mu g/m^3$.

9.9 References for Chapter 9

Albert, R.E., J. Lewtas, S. Nesnow, T.W. Thorslund, and E. Anderson. 1983. Comparative potency method for cancer risk assessment: Application to diesel particulate emissions. Risk Analysis. 3: 101-117.

Albert, R.E., and C. Chen. 1986. U.S. EPA diesel studies on inhalation hazards. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds. Carcinogenic and Mutagenic Effects of Diesel Engine Exhaust. Amsterdam: Elsevier, pp. 411-419.

American Public Transport Association. 1990. 1990 Transit Fact Book. September 1990.

Barfnecht, T.R., R.A. Hites, E.L. Cavaliers, and W.G. Thilly. 1982. Human cell mutagenicity of polycyclic aromatic hydrocarbon companies of diesel emissions. In: Lewtas, J., ed. Toxicological Effects of Emissions from Diesel Engines. New York: Elsevier, pp. 277-294.

Barnhart, M.L., S. Chen, S.O. Salley, and H. Puro. 1981. Ultrastructure and morphometry of the alveolar lung of guinea pigs chronically exposed to diesel engine exhaust: Six months experience. J. Appl. Toxicol. 1: 88-103.

Behymer, T.D. and R.A. Hites. 1985. Photolysis of polycyclic aromatic hydrocarbons adsorbed on simulated atmospheric particulates. Environ. Sci. Technol., 19: 1004-1006.

Benhamou S., E. Benhamou, and R. Flamant. 1988. Occupational risk factors of lung cancer in a French case-control study. Br. J. Ind. Med. 45: 231-233.

Boffetta, P. and S.D. Stellman. 1988. Association between diesel exhaust exposure and multiple myeloma an example of confounding. Prev. Med. 17: 236-247.

Bond, J.A., N.F. Johnson, M.B. Snipes, and J.L. Mauderly. 1990. DNA adduct formation in rat alveolar type II cells: cells potentially at risk for inhaled diesel exhaust. Environ. Molec. Mutagen. 16: 64-69.

Bouhuys, A., Beck, G.J., and Schoenberg, J.B. 1978. Do present levels of air pollution outdoors affect respiratory health? Nature 276: 466-471.

Brightwell, J., X. Fouillet, A.L. Cassano-Zoppi, R. Gatz, and F. Duchosal. 1986. Neoplastic and functional changes in rodents after chronic inhalation of engine exhaust emissions. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds. Carcinogenic and Mutagenic Effects of Diesel Engine Exhaust. Amsterdam: Elsevier, pp. 471-485.

Brooks, A.L., A.P. Li, J.S. Dutcher, C.R. Clark, S.J. Rothenberg, R. Kiyoura, W.E. Bechtold, R.O. McClellan. 1984. A comparison of genotoxicity of automobile exhaust particles form laboratory and environmental sources. Environ. Mutagen. 6: 651-668.

Butler, J.D. and P. Crossley. 1981. Reactivity of polycyclic aromatic hydrocarbons adsorbed on soot particles. Atmos. Environ., 15: 91-94.

Carey, P.M. 1987. Air toxic emissions from motor vehicles. Ann Arbor, MI: U.S. Environmental Protection Agency, Office of Mobile Sources, EPA Report no. EPA-AA-TSS-PA-86-5.

Casto, B.C., G.G. Hatch, S.L. Huang, J.L. Huisingh, S. Nesnow, and M.D. Waters. 1981. Mutagenic and carcinogenic potency of extracts of diesel and related environmental emissions: *In vitro* mutagenesis and oncogenic transformation. Environ. Int. 5: 403-409.

Chescheir, G.M. III, N.E. Garrett, J.D. Shelburne, J.L. Huisingh, and M.D. Waters. 1981. Mutagenic effects of environmental particulates in the CHO/HGPRT system. In: Waters, M.D. Sandhu, S.S., Huisingh. J.L., Claxton, L.C., Nesnow, S., eds. Application of short-term bioassays in the fractionation and analysis of complex environmental mixtures. New York: Plenum Press, pp. 337-350.

Claxton, L.D. 1981. Mutagenic and carcinogenic potency of diesel and related environmental emissions: Salmonella bioassay. Environ. Int. 5: 389-391.

Claxton, L.D. 1983. Characterization of automotive emissions by bacterial mutagenesis bioassay: A review. Environ. Mutagen. 5: 609-631.

Claxton, L.C. and M. Kohan. 1981. Bacterial mutagenesis and the evaluation of mobile-source emissions. In: Waters, M.D. Sandhu, S.S., Huisingh. J.L., Claxton, L.C., Nesnow, S., eds. Application of short-term bioassays in the fractionation and analysis of complex environmental mixtures. New York: Plenum Press, pp. 299-317.

Cuddihy, R.G., W.C. Griffith, and R.O. McClellan. 1981. Health risks from light-duty diesel vehicles. Environ. Sci. Technol. 18: 14-21a.

Curren, R.D., R.E. Kouri, D.M. Kim, and L.M. Schectman. 1981. Mutagenic and carcinogenic potency of extracts from diesel related environmental emissions: Simultaneous morphological

transformation and mutagenesis in BALB/c 3T3 cell. Environ. Int. 5: 411-415.

Damber, L.A., and L.G. Larsson. 1987. Occupation and male lung cancer: A case-control study in northern Sweden. Br. J. Ind. Med. 44:446-453.

Dassen, W., Brunekreff, B., Hoek, G., Hofschreuder, P., Staatsen, B., deGrout, H., Schouten, E., and Biersteker, K. 1986. Decline in children's pulmonary function during an air pollution episode. J. Air. Pollut. Control Assoc. 36: 1223-1227.

Depass L.R., K.C. Chen, and L.G. Peterson. 1982. Dermal carcinogenesis bioassays of diesel particulates and dichloromethane extract of diesel particulates in C3H mice. In: Lewtas, J., ed. Toxicological Effects of Emissions from Diesel Engines. Amsterdam: Elsevier. pp. 321-327.

Dlugi, R. and H. Güsten. 1983. The catalytic and photocatalytic activity of coal fly ashes. Atmos. Environ., 17: 1765-1771.

Dockery, D.W., Ware, J.H., Ferris Jr., B.G., Speizer, F.E., Cook, N.R., and Herman, S.M. 1982. Change in pulmonary function in children associated with air pollution episodes. J. Air. Pollut. Contrl. Assoc. 32: 937-942.

Dukovich, M., R.E. Yasbin, S.S. Lestz, T.H. Risby, and R.B. Zweidinger. 1981. The mutagenic and SOS-inducing potential of the soluble organic fraction collected form diesel particulate emissions. Environ. Mutagen. 3: 253-264.

Edling, O., C.G. Anjou, O. Axelson, and H. Kling. 1987. Mortality among personnel exposed to diesel exhaust. Int. Arch. Occup. Environ. Health 59: 559-565.

Environ Corporation. 1987. Risk assessment issues in EPA's technical report "Air Toxics Emissions from Motor Vehicles". Prepared for the Motor Vehicle Manufacturers Association.

EPA. 1987. Federal Register, 40 CFR Part 50, July 1, 1987, pages 24634-24669.

EPA. 1989. Analysis of Air Toxics Emissions, Exposures, Cancer Risks and Controllability in Five Urban Areas: Volume I: Base Year Analysis and Results. Office of Air Quality Planning and Standards, Research Triangle Park, NC. July, 1989. EPA Report No. EPA-450/2-89-012a.

EPA. 1990a. Regulation of Fuels and Fuel Additives: Fuel Quality Regulations for Highway Diesel Fuel Sold in 1993 and Later Calendar Years; Final Rule. Federal Register 55 (162): 34120-34151.

EPA. 1990b. Health assessment document for diesel emissions; workshop review draft. Office of Research and Development, Office of Health and Environmental Assessment, Washington, DC. EPA-600/8-90/057A.

EPA. 1991a. Compilation of Air Pollutant Emission Factors -- Volume II: Mobile Sources. Ann Arbor, Michigan: Office of Mobile Sources. Publication no. AP-42, Supplement A.

EPA. 1991b. National Air Pollutant Emission Estimates 1940-1990. Research Triangle Park, North Carolina: Office of Air Quality Planning and Standards. Publication no. EPA-450/4-91-026.

EPA. 1991c. National Air Quality and Emissions Trends Report, 1990. Research Triangle Park, North Carolina: Office of Air Quality Planning and Standards. Publication no. EPA-450/4-91-023.

EPA. 1991d. Presentation at Air and Waste Management's "Air Toxics from Mobile Sources" conference by Dr. William Pepelko, October 16-18, 1991.

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

EPA. 1993. Integrated Risk Information System. U.S. Environmental Protection Agency. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

Fox, M. A. and S. Olive. 1979. Photooxidation of anthracene on atmospheric particulate matter. Science, 205: 582-583.

Garshick, E., M.B. Schenker, A. Munoz, M. Segal, T.J. Smith, S.R. Woskie, S.K. Hammond, and F.E. Speizer. 1987. A case control study of lung cancer and diesel exhaust exposure in railroad workers. Am. Rev. Respir. Dis. 135: 1242-1248.

Garshick, E., M.B. Schenker, A. Munoz, M. Segal, T.J. Smith, S.R. Woskie, S.K. Hammond, and F.E. Speizer. 1988. A retrospective cohort study of lung cancer and diesel exhaust exposure in railroad workers. Am. Rev. Respir. Dis. 124: 820-825.

Grimmer, G., H. Brune, R. Deutsch-Wenzel, G. Dettbarn, J. Jacob, K.W. Naujack, U. Mohr, and H. Ernst. 1987. Contribution of polycyclic aromatic hydrocarbons and nitro-derivatives to the carcinogenic impact of diesel exhaust condensate evaluated by implantation into the lungs of rats. Cancer Lett. 37: 173-180.

Grosjean, D., K. Fung, and J. Harrison. 1983. Interactions of polycyclic aromatic hydrocarbons with atmospheric pollutants. Environ. Sci. Technol., 17: 673-679.

Guerrero, R.R., D.E. Rounds, and J. Orthoefer. 1981. Sister chromatid exchange analysis of Syrian hamster lung cells treated *in vivo* with diesel exhaust particulates. Environ. Int. 5: 445-454.

Hall, N.E., and E. Wynder. 1984. Diesel exhaust exposure and lung cancer. Environ. Res. 34: 77-86.

Harris, J.E. 1983. Diesel emissions and lung cancer. Risk Analysis. 3: 83-100.

Hausman, J.A., Ostro, B.D., and Wise, D.A. 1984. Air pollution and lost work. Cambridge, MA: National Bureau of Economic Research, NBER Working Paper no. 1263.

Hayes, R.B., T. Thomas, D.T. Silverman, et al. 1989. Lung cancer in motor exhaust occupations. Am. J. Ind. Med. 16: 685-695.

Heinrich, U., H. Muhle, S. Takenaka, H. Ernst, R. Fuhst, U. Mohr, F. Pott, and W. Stober. 1986a. Chronic effects on the respiratory tract of hamsters, mice, and rats after long-term inhalation of high concentrations of filtered and unfiltered diesel engine emissions. J. Appl. Toxicol. 6: 383-395.

Heinrich, U., F. Pott, and S. Rittinghausen. 1986b. Comparison of chronic inhalation effects in rodents after long-term exposure to either coal oven flue gas mixed with pyrolized pitch or diesel engine exhaust. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds. Carcinogenic and Mutagenic Effects of Diesel Engine Exhaust. Amsterdam: Elsevier. pp. 441-457.

Heinrich, U., D. Dungworth, F. Pott, A. Schulte, L. Peters, C. Dasenbrock, K. Levsen, W. Koch and O. Creutzenburg. (1991). The carcinogenic effects of carbon black particles and tar/pitch condensation aerosol after inhalation exposure of rats. Seventh International Symposium on Inhaled Particles, Edinburgh, September 16-20, 1991.

Hoar, S., and R. Hoover. 1985. Truck driving and bladder cancer mortality in rural New England. J. Natl. Cancer Int. 74: 771-774.

Howe, G.R., J. D. Burch, and A.B. Miller. 1980. Tobacco use, occupation, coffee, various nutrients, and bladder cancer. J. Natl. Cancer Inst. 64: 701-713.

Howe, G.R., D. Fraser, J. Lindsay, et al. 1983. Cancer mortality (1965-77) in relation to diesel fume and coal exposure in a cohort of retired railway workers. J. Natl. Cancer Inst. 3NCI 70: 1013-1019.

Huisingh, J., R. Bradow, R. Jungers, L. Claxton, R. Zweidinger, S. Tejada, J. Bumgarner, F. Duffeild, M. Waters, V.F. Simmon, C. Hare, C. Rodrigues, and L. Snow. 1978. Application of bioassay to the characterization of diesel particle emissions. In: Waters, M.D. Sandhu, S.S., Huisingh. J.L., Claxton, L.C., Nesnow, S., eds. Application of short-term bioassays in the fractionation and analysis of complex environmental mixtures. New York: Plenum Press, pp. 381-418.

Ingham, M. C., and R. B. Warden. 1987. Cost-effectiveness of diesel fuel modifications for particulate control. SAE Paper 870556.

International Agency for Research on Cancer. 1989. IARC monographs on the evaluation of carcinogenic risks to humans; diesel and gasoline engine exhausts and some nitroarenes, Volume 46.

Iscovich, J., R. Castelleto, J. Esteve, et al. 1987. Tobacco smoking, occupational, exposure, and bladder cancer in Argentina. Int. J. Cancer 40: 734-740.

Ishinishi, N., N. Kuwabara, S. Nagase, T. Suzuki, S. Ishiwata, and T. Kohno. 1986. Long-term inhalation studies on effects of exhaust from heavy and light duty diesel engines on F344 rats. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds. Carcinogenicity and mutagenicity of diesel engine exhaust. Amsterdam: Elsevier, pp. 329-348.

Ishinishi, N., N. Kuwabara, Y. Takaki, et al. 1988. Long term inhalation experiments on diesel exhaust. In: Diesel Exhaust and Health Risk. Results of the HERP Studies. Entire text of discussion, Research Committee for HERP Studies. Japan Automobile Research Institute, Inc. Tsukuba, Ibaraki 305, Japan.

Iwai, K., T. Udagawa, M. Yamagishi, and H. Yamada. 1986. Long-term inhalation studies of diesel exhaust on F344 SPF rats. Incidence of lung cancer and lymphoma. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds. Carcinogenicity and mutagenicity of diesel engine exhaust. Amsterdam: Elsevier, pp. 349-360.

Iyer et al. 1990. Diesel exhaust exposure and bladder cancer risk. Eur. J. Epidemiol. 6: 49-54.

Johnson, J.H. 1988. Automotive emissions. In: Air pollution, the automobile, and public health. Health Effects Institute, National Academy Press, Washington, D.C.

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.

Kaplan, H.L., W.F. Mackenzie, K.J. Springer, R.M. Schreck, and J.J. Vostal. 1982. A subchronic study of the effects of exposure of three species of rodents to diesel exhaust. In: Lewtas, J., ed. Toxicological Effects of Emissions from Diesel Engines. New York: Elsevier Biomedical, pp. 161-220.

Karagianes, R.F., R.F. Palmer, and R.H. Busch. 1981. Effects of inhaled diesel emissions and coal dust in rats. Am. Ind. Hyg. Assoc. J. 42: 382-391.

Kawataba, Y., K. Iwai, T. Udagawa, K. Tukagoshi, and K. Higuchi. 1986. Effects of diesel soot on unscheduled DNA synthesis of tracheal epithelium and lung tumor formation. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds. Carcinogenicity and Mutagenicity of Diesel Engine Exhaust. Amsterdam: Elsevier, pp. 213-222.

Kitchen, M. and W. Damico. 1992. Development of Conversion Factors for Heavy Duty Bus Engines g/bhp-hr to g/mile. Ann Arbor, MI: U.S. Environmental Protection Agency, Office of Mobile Sources, EPA Report no. EPA-AA-EVRB-92-01.

Korfmacher, W.A., G. Mamantov, E.L. Wehry, D.F.S. Natusch, and T. Mauney. 1981. Nonphotochemical decomposition of fluorene vapor-adsorbed on coal fly ash. Envirn. Sci. Technol., 15: 1370-1375.

Korfmacher, W.A., D.F.S. Natusch, D.R. Taylor, G. Mamantov, and E.L. Wehry. 1980a. Oxidative transformations of polycyclic aromatic hydrocarbons adsorbed on coal fly ash. Science, 207: 763-765.

Korfmacher, W.A., E.L. Wehry, G. Mamantov, and D.F.S. Natusch. 1980b. Resistance to photochemical decomposition of polycyclic aromatic hydrocarbons vapor-adsorbed on coal fly ash. Environ. Sci. Technol., 14: 1094-1099.

Kotin P., H. Falk, and M. Thomas. 1955. Aromatic hydrocarbons. III. Presence in the particulate phase of diesel engine exhausts and the carcinogenicity of exhaust extracts. Arch. Ind. Health 11: 113-120.

Kunitake, E., A. Imase, K. Shimamura, N. Ishinishi, A. Hisanaga, and A. Tanaka. 1988. Skin application and subcutaneous injection experiments of diesel particulate extracts using ICR mice and nude mice. In: Diesel Exhaust and Health Risks. Research Committee for HERP Studies, Ibaraki, Japan. pp. 217-225.

Lee, K.P., N.W. Henry III, H.J. Trochimowicz, and C.F. Reinhardt. 1986. Pulmonary response to impaired lung clearance in rats

following excessive TiO$_2$ dust deposition. Environ. Res. 41: 144-167.

Lerchen M.L., C.L. Wiggins, and J.M. Samet. 1987. Lung cancer and occupation in New Mexico. J. Natl. Cancer Inst. 79: 639-645.

Lewis, T.R., F.H. Green, W.J. Moorman, J.A. Burg, and D.W. Lynch. 1986. A chronic inhalation toxicity study of diesel engine emissions and coal dust alone and combined. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds. Carcinogenicity and mutagenicity of diesel engine exhaust. Amsterdam: Elsevier, pp. 361-380.

Lewtas, J. 1982. Mutagenic activity of diesel emissions. In: Lewtas J. ed. Toxicological Effects of Emissions from Diesel Engines, New York: Elsevier; pp.243-264.

Lewtas, J. 1983. Evaluation of the mutagenicity and carcinogenicity of motor vehicle emissions in short-term bioassays. Environ. Health Perspect. 47: 141-152.

Lewtas, J. 1991. Carcinogenic risks of polycyclic organic matter (POM) from selected emission sources. Health Effects Research Laboratory, Research Triangle Park, NC. Deliverable report no. 3128.

Li, A.P., and R.E. Royer. 1982. Diesel-exhaust-particle extract enhancement of chemical-induced mutagenesis in cultured Chinese hamster ovary cells: possible interaction of diesel exhaust with environmental chemicals. Mutat. Res. 103: 349-355.

Liber, H.L., B.M. Andon, R.A. Hites and W.G. Thilly. 1981. Diesel soot: Mutation measurements in bacterial and human cells. Environ. Int. 5: 281-284.

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

Ligocki, M. P., R. R. Schulhof, R. E. Jackson, M. M. Jimenez, G. Z. Whitten, G. M. Wilson, T. C. Meyers, and J. L. Fieber. 1992. Modeling the effects of reformulated gasolines on ozone and toxics concentrations in the Baltimore and Houston areas. Systems Applications International, San Rafael, California (SYSAPP-92/127).

Machiele, P. A. 1988. Heavy-Duty Vehicle Emission Conversion Factors II, 1962-2000. Ann Arbor, Michigan: U.S. Environmental Protection Agency, Office of Mobile Sources. Publication no. EPA-AA-SDSB-89-01.

Matsushita, H., S. Gotto, O. Endo, J. Lee, and A. Kawai. 1986. Mutagenicity of diesel exhaust and related chemicals. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds.

Carcinogenicity and mutagenicity of diesel engine exhaust. New York: Elsevier, pp. 103-118.

Mauderly, J.L., R.K. Jones, W.C. Griffith, R.F. Henderson, and R.O. McClellan. 1987. Diesel exhaust is a pulmonary carcinogen in rats exposed chronically by inhalation. Fundam. Appl. Toxicol. 9: 208-221.

Mauderly, J.L., Rr.K. Jones, R.F. Henderson, R.K. Wolff, J.A. Pickrell, R.O. McClellan, and N.A. Gillett. 1988. Relationship of lung structural and functional changes to accumulation of diesel exhaust particles. Ann. Occup. Hyg. 32: 659-669.

Mauderly, J.L., M.B. Snipes, E.B. Barr, W.E. Bechtold, S.A. Belinsky, R.F. Henderson, C.E. Mitchell, K.J. Nikula, and D.G. Thomassen. 1991. Influence of particle-associated organic compounds on carcinogenicity of diesel exhaust. Seventh International Symposium on Inhaled Particles, Edinburgh, September 16-20, 1991.

McClellan, R.O., R.G. Cuddihy, W.C. Griffith, and J.L. Mauderly. 1989. Integrating diverse data sets to assess the risks of airborne pollutants. In: Mohr, U., ed Assessment of Inhalation Hazards. Springer Verlag: New York, pp. 3-22.

Mitchell, A.D., E.L. Evans, M.M. Lotz, E.S. Riccio, K.E. Mortelmans, and V.F. Simmon. 1981. Mutagenic and carcinogenic potency of extracts of diesel and related environmental emissions: *In vitro* mutagenesis and DNA damage. Environ. Int. 5: 393-401.

Morimoto, K., H. Kondo, M. Kitamura, and A. Koizumi. 1986. Genotoxicity of diesel exhaust emissions in *in vitro* short-term assays. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds. Carcinogenicity and Mutagenicity of Diesel Engine Exhaust. New York: Elsevier, pp. 85-102.

Motor Vehicle Manufacturers Association. 1991. MVMA Motor Vehicle Facts and Figures '91.

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.

National Academy of Sciences. 1972. Particulate polycyclic organic matter. National Academy of Sciences, National Academy Press, Washington, D.C.

National Research Council. 1982. Impacts of diesel-powered light-duty vehicles: diesel technology. National Academy Press, Washington, D.C.

Nesnow, S., C. Evans, A. Stead, J. Creason, T.J. Slaga, and L.L. Triplett. 1982. Skin carcinogenesis studies of emission extracts. In: Lewtas, J., ed. Toxicological Effects of Emissions from Diesel Engines. Amsterdam: Elsevier, pp. 295-320.

NIOSH. (1988). Carcinogenic effects of exposure to diesel exhaust. Current Intelligence Bulletin 50, August 1988.

Orthoefer J.G., W. Moore, and D. Kraemer. 1981. Carcinogenicity of diesel exhaust as tested in strain A mice. Environ. Int. 5: 461-471.

Ostro, B.D. 1987. Air pollution and morbidity revisited: a specification test. J. Environ. Econ. Manage. 14(1): 87-98.

Pepelko, W.E., and W.B. Peirano. 1983. Health effects of exposure to diesel engine emissions. J. Amer. Coll. Toxicol. 2: 253-306.

Pepelko, W.E. and C.H. Ris. 1991. Update on U.S. Environmental Protection Agency Activities in the Assessment of Mobile Source Air Toxics. Proceedings from the Air and Waste Management Association's specialty conference "Air Toxics Pollutants from Mobile Sources: Emissions and Health Effects." October 16-18, 1991.

Pereira, M.A., T.H. Conner, J. Meyne, and M.S. Legator. 1981a. Metaphase analysis, micronucleus assay and urinary mutagenicity assay of mice exposed to diesel emissions. Environ. Int. 5: 435-438.

Pereira, M.A., P.S. Sabharwal, L. Gordon, and A.J. Wyrobek. 1981b. The effect of diesel exhaust on sperm-shape abnormalities in mice. Environ. Int. 5: 459-460.

Pereira, M.A., L. McMillan, P. Kaur, D.K. Gulati, and P.S. Sabharwal. 1982. Effect if diesel exhaust emissions, particulates, and extract on sister chromatid exchange in transplacentally exposed fetal hamster liver. Environ. Mutagen. 4: 215-220.

Pereira, M.A., P.S. Sabharwal, P. Kaur, C.B. Ross, A. Choi, and T. Dixon. 1981c. *In vitro* detection of mutagenic effects of diesel exhaust by short-term mammalian bioassays. Environ. Int. 5: 439-443.

Pistikpopulos, P., H.M. Worthwm, L. Gomes, S. Masclet-Beyne, E.B. Nguyen, P.A. Masclet, and G. Mouvier. 1990. Mechanisms of formation of particulate polycyclic aromatic hydrocarbons in relation to the particle size distribution; effects on the meso-scale transport. Atmos. Environ., 24A: 2573-2584.

Pope, C.A. III, D.W. Dockery, J.D. Spengler, and M.E. Raizenne. 1991. Respiratory health and PM_{10} Pollution; a daily time series analysis. Am. Rev. Respir. Dis. 144:668-674.

Pott, F., D.L. Dungworth, U. Heinrich, H. Muhle, K. Kamino, P.G. German, M. Roller, and R.M. Rippe. (1991). Lung tumors in rats after intratracheal instillation of dusts. Seventh International Symposium on Inhaled Particles, Edinburgh, September 16-20, 1991.

Pott, F. and U. Heinrich. 1987. Dieselmotorabgas und Lungenk auf die Gefanfrdung des Menschen. In: Umwelthygiene, Vol. 19. Med. Institut. F. Umwelthygiene, Annual Report 1986/87. Dusseldorf, F.R.G., pp. 130-167.

Ransom, M.R. and C.A. Pope III. 1992. Elementary school absences and PM_{10} pollution in Utah valley. Environ. Res. 58:204-219.

Rushton, L., M.R. Alderson, and C.R. Nagarajah. 1983. Epidemiological survey of maintenance workers in London Transport executive bus garages at Chiswick works. Br. J. Ind. Med. 40: 340-345.

Russell, L.B., W.M. Generoso, E.F. Oakberg, W.L. Russell, J.W. Bangham, and K.F. Stelzner. 1980. Tests for heritable effects induced by diesel exhaust in the mouse. ORNL-5685. Martin Marietta Energy Systems, Inc. Oak Ridge Natl. Lab.

Schreck, A.M., S.C. Collins, L. Dooley, A. Fiks, J.L. Graf, and M.M. Preache. 1982. Experimental conditions in GMR chronic inhalation studies of diesel exhaust. J. Appl. Toxicol. 1: 68-76.
Schuler, R.L., and R.W. Niemeier. 1981. A study of diesel emissions on *Drosophila*. Environ. Int. 5: 431-434.

Schwartz, J. 1991. Particulate air pollution and daily mortality in Detroit. Environ. Res. 56:204-213.

Schwartz, J. and D.W. Dockery. 1992a. Increased mortality in Philadelphia associated with daily air pollution concentrations. Am. Rev. Respir. Dis. 145:600-604.

Schwartz, J. and D.W. Dockery. 1992b. Particulate air pollution and daily mortality in Steubenville, Ohio. Am. J. Epidemiol. 135:12-19.

Siak, J.S., T.L. Chan, and P.S. Lees. 1981. Diesel particulate extracts in bacterial test systems. Environ. Int. 5: 243-248.

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. Seinicki, Navistar International Transportation Corporation, to Phil Lorang, Environmental Protection Agency, January 6, 1992.

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

Smith, R.A., and L. Stayner. 1990. An exploratory assessment of the risk of lung cancer associated with exposure to diesel exhaust based on a study with rats. Final Report. Division of Standards Development and technology transfer. NIOSH. Cincinnati, OH.

Steenland, K. et al. 1990. Case-control study of lung cancer and truck driving in the Teamster Union. Am. J. Public Health 80: 670-674.

Steenland, K., C. Burnett, and A.M. Osoria. 1987. A case-control study of bladder cancer using city directions as a source of occupational data. Am. J. Epidemiol. 126: 247-257.

Steinbeck et al. 1990. Increased risk of urothelial cancer in Stockholm during 1985-87 after exposure to benzene and exhausts. Int. J. Cancer 45: 1012-1017.

Stober, W. 1986. Experimental induction of tumors in hamsters, mice, and rats after ling-term inhalation of filtered and unfiltered diesel engine exhaust. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds. Carcinogenicity and Mutagenicity of Diesel Engine Exhaust. New York: Elsevier, pp. 421-439.

Stober, W. 1987. On the health hazards of particulate diesel engine exhaust emissions. SAE Technical Paper 871988.

Stober, W. 1989. Estimates of health hazards from exposures to diesel exhaust. Air and Waste Management Association Paper 89-34A.2.

Stober, W. 1991. Health Hazards Associated with Public Exposure to Diesel Exhuast-An Experimental Data Update. Proceedings from the Air and Waste Management Association's specialty conference "Air Toxics Pollutants from Mobile Sources: Emissions and Health Effects." October 16-18, 1991.

Takaki Y., S. Kitamura, N. Kuwabara, and Y. Fukuda. 1989. Long-term inhalation studies of exhaust from diesel engine in F344

rats: The quantitative relationship between pulmonary hyperplasia and anthracosis. Exp. Pathol. 37: 56-62. Report of poster presentation.

Takemoto, K., H. Yoshimura, and H. Katayama. 1986. Effects of chronic inhalation studies exposure to diesel exhaust on the development of lung tumors in di-isopropanol-nitrosamine-treated F344 rats and newborn C57BL and ICR mice. In: Ishinishi, N., Koizumi, A., McClellan, R., Stober, W., eds. Carcinogenicity and mutagenicity of diesel engine exhaust. New York: Elsevier, pp. 311-327.

Takemoto, K., H. Katayama, T. Kuwabara,. and M. Hasumi. 1988. Carcinogenicity by subcutaneous administration of diesel particulate extracts in mice. In: Diesel Exhaust and Health Risks. Research Committee for HERP Studies, Ibaraki, Japan. pp. 227-234.

Tucker, J.D., J. Xu, J. Stewart, P.C. Baciu, and T. Ong. 1986. Detection of sister chromatid exchanges induced by volatile genotoxicants. Teratogen. Carcinogen. Mutagen. 6: 15-21.

Ullman, T. L. 1989. Investigation of the Effects of Fuel Composition and Injection and Combustion System Type on Heavy-Duty Diesel Exhaust Emissions. Final Report, CRC Project VE-1. March, 1989.

University of Michigan Transportation Research Institute. 1988. Analysis of Heavy-Duty Truck Use in Urban Areas. Ann Arbor, Michigan: University of Michigan. Publication no. UMTRI-88-31.

U.S. Department of Energy. 1991. National Energy Stategy: Powerful Ideas for America. First Edition 1991/1992. Washington, D. C. February, 1991.

Van Vaeck, L. and K. Van Cauwenberghe. 1978. Cascade impactor measurements of the size distribution of the major classes of organic pollutants in atmospheric particulate matter. Atmos. Environ., 12: 2229-2239.

Van Vaeck, L. and K. Van Cauwenberghe. 1984. Conversion of polycyclic aromatic hydrocarbons on diesel particulate matter upon exposure to ppm levels of ozone. Atmos. Environ., 18: 323-328.

Waller, R.E. 1981. Trends in lung cancer in London in relation to exposure to diesel fumes. Environ. Int. 5: 479-483.

Ware, J.H., Ferris, Jr., B.G., Dockery, D.W., Spengler, J.D., Stram, D.O., and Speizer, F.E. 1986. Effects of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. Am. Rev. Respir. Dis. 133: 834-842.

Waxweiler, R.J., J.K. Wagoner, and V.E. Archer. 1973. Mortality of potash workers. J. Occup. Med. 15: 486-489.

Williams, R.R., N.L. Stegens, and J.R. Goldsmith. 1977. Associations of cancer site and type with occupation and industry from the Third National Cancer Survey interview. J. Natl. Cancer Inst. 59: 1147-1185.

Wong, D., C.E. Mitchell, R.K. Wolf, J.L. Mauderly, and A.M. Jeffrey. 1986. Identification of DNA damage as a result of exposure of rats to diesel engine exhaust. Carcinogenesis 7: 1595-1597.

Wong, O., R.W. Morgan, L. Kheyts, S.R. Larson, and M.D. Whorton. 1985. Mortality among members of a heavy construction equipment operators' union with potential exposure to diesel exhaust emissions. Br. J. Ind. Med. 42: 435-448.

Wong, P.O., R.E. Gregory, and A.L. Brooks. 1983. *In vitro* evaluation of the tumor-promoting potential of diesel-exhaust-particle extracts. J. Toxicol. Environ. Health 11: 188-197.

Wynder, L.E., G. Dieck, and N.E. Hall. 1985. A case control study of diesel exhaust exposure and bladder cancer. Environ. Res. 34: 475-489.

Yu, C.P. and K.J. Yoon. 1990. Retention modeling of diesel exhaust particles in rats and humans. Research Report No. 40. Health Effects Institute, Cambridge, MA.

10.0 GASOLINE PARTICULATE MATTER

10.1 Chemical and Physical Properties

Gasoline particulate exhaust consists of a solid core composed mainly of carbon, a soluble organic fraction, sulfates, and trace elements. The gasoline particulate matter ranges from 0.01 to 0.1 µm in diameter with the peak at around 0.02 µm while the majority of the diesel particulate matter ranges from 0.1 to 1.0 µm with a peak at around 0.15 µm. When a particle is less than 1 micron (µm) in diameter it is small enough to be inhaled deeply into the lungs.

Light-duty diesel engines emit from 30 to 100 times more particles than comparable catalyst-equipped gasoline vehicles (NRC, 1982). This is offset to some degree by the greater number of catalyst-equipped gasoline vehicles on the road, relative to diesel vehicles.

The remaining chemical and physical properties of gasoline particulate matter are very similar to those of diesel particulate matter. Please consult Section 9.1 concerning diesel particulate matter for further information. No mention of the gasoline exhaust phase will be attempted here since this chapter discusses gasoline particulate matter exclusively. Motor vehicle emissions have been shown to substantially increase the mutagenic activity of bacterial strains following irradiation of the gas phase rather than the particulate phase of mixtures. These findings are discussed further in Section 12.4.3.

10.2 Formation and Control Technology

Gasoline particulate matter is formed as a result of incomplete combustion of gasoline. Lubricating oil and other fuel hydrocarbons may also contribute. It consists of a carbon core with various organic compounds associated with it. The sulfate particles experienced by gasoline engines is mostly from catalyst equipped vehicles utilizing unleaded gasoline (EPA, 1985). At present, there are no controls 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.

EPA's Five City Study (EPA, 1989) determined that POM contributed to 27% of the average excess aggregate cancer incidence in the five cities. Of this 27%, gasoline particulate matter accounted for 32%.

10.3 **Emissions**

10.3.1 **Emission Factors for Baseline Scenarios**

Gasoline particulate matter is emitted at the mg/mile level. Because it 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.

For this study, the available emissions data were summarized (Hammerle, et al., 1992; Lewtas, 1991; Carey, 1987; Lang, et al., 1981; EPA, 1985; Volkswagen, 1991; Smith, 1981; Urban, 1980a, 1980b, 1980c; CARB, 1986). A summary table is provided in Appendix H. Gasoline particulate emissions are given in mg/mile and expressed as total hydrocarbons (THC), as measured by the FID. The gasoline particulate mass emissions data vary considerably, but appear more consistent when expressed as a percentage of THC. In general, gasoline particulate emissions tend to increase as THC emissions increase.

Since this report is meant to provide a prospective look at emissions, the data from Hammerle, et al. (1992), the only study which includes post-1986 model year vehicles, were used solely. Data from the other studies were used as support for the apparent correlation between gasoline particulate matter and THC. Use of the Hammerle, et al. (1992) data offers several advantages. First, data were collected from current 1991 model year Escorts and Explorers equipped with three-way and dual three-way catalysts, respectively. Second, data were collected at various mileage intervals up to 105,000 miles. Data from this study indicate that the mass of gasoline particulate matter is roughly 1.1% of the mass of THC as measured by the FID. This percentage was used as input to MOBTOX and applied to all gasoline vehicle categories.

10.4 **Atmospheric Reactivity and Residence Times**

The atmospheric reactivity and residence times are very similar to those discussed previously for diesel particulate matter and POM in Section 9.4.

10.4.1 **Urban Airshed Modeling of Reformulated Gasoline Impact on Ambient POM**

Treatment of POM in the UAM-Tox model is discussed in Section 9.4.5.

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.

In addition, motor vehicle-related POM concentrations with federal reformulated gasoline use decreased more in winter than in summer, ranging from 4 to 8 percent. Simulations for the summer Houston episode predicted larger decreases than in the Baltimore-Washington area with the use of reformulated gasoline.

10.5 Exposure Estimation

The data presented in Table 10-1 represent the results determined by HAPEM-MS modeling that was described previously in Section 4.1.1. These numbers have been adjusted to represent the increase in VMT expected in future years.

Table 10-1.   Annual Average HAPEM-MS Exposure Projections for Gasoline Particulate Matter.

Year-Scenario	Exposure ($\mu g/m^3$)		
	Urban	Rural	Nationwide
1990 Base Control	0.58	0.31	0.51
1995 Base Control	0.32	0.17	0.29
1995 Expanded Reformulated Fuel Use	0.31	0.17	0.28
2000 Base Control	0.23	0.12	0.20
2000 Expanded Reformulated Fuel Use	0.22	0.12	0.19
2000 Expanded Adoption of California Standards	0.22	0.12	0.19
2010 Base Control	0.19	0.10	0.17
2010 Expanded Reformulated Fuel Use	0.18	0.09	0.16
2010 Expanded Adoption of California Standards	0.17	0.09	0.15

10.6 Carcinogenicity of Gasoline Particulate Matter and Unit Risk
Estimates

10.6.1 Most Recent EPA Assessment

At this time there exists no official EPA document detailing the carcinogenicity evidence relating to gasoline particulate matter. Much of the information in this section will be taken from several sources, some relating to particles in general and others focusing on the organic compounds associated with gasoline particulate matter.

10.6.1.1 Description of Available Carcinogenicity Data

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 a 1977 Mustang II-302 with a V8 engine, catalyst and EGR, running on unleaded fuel. Also tested was a 6 cylinder Ford van without a catalyst, running on leaded fuel. The organic material was extracted from the particles and five to seven doses or concentrations were used in the bioassays discussed below (Lewtas, 1991).

There were four *in vitro* bioassays, *Salmonella typhimurium* (Ames test), L5178Y mouse lymphoma, BALB/c 3T3, and Chinese hamster ovary assays, conducted to determine the possibility of gene mutation. The organics from the gasoline particles were found to be mutagenic in the *S. typhimurium*, L5178Y mouse lymphoma, and BALB/c 3T3 assays, with the Chinese hamster ovary assay showing a relatively weak to negative response. In the four *in vitro* bioassays conducted to determine DNA damage (recombination, chromatid exchanges, unscheduled DNA repair, and sister chromatid exchanges) the gasoline particle 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.

10.6.1.2 Weight-of-Evidence Judgement of Data and EPA
          Classification

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.

10.6.1.3 Data Sets Used for Unit Risk Estimate

     The unit risk available for gasoline particulate matter is based on the comparative potency method. This method, as described previously under diesel particulate matter in Section 9.6.2, utilizes epidemiological data from coke oven emissions, roofing tar emissions, and cigarette smoke and develops a correlation with the gasoline particle organics based on the relative potencies in the mouse skin tumor initiation assay. This process then determines the unit risk. See Section 9.6.2 for a more complete description of the comparative potency method.

     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.

10.6.1.4 Dose-Response Model Used

     Since the comparative potency method is being used to determine the unit risk, no single dose-response model was used. In this comparative potency estimate, gasoline exhaust particulate matter was compared with coke oven emissions, roofing tar, and cigarette smoke condensate using skin papilloma multiplicity data (papillomas/mouse at 1 mg dosage).

10.6.1.5 Unit Risk Estimate

     The lifetime unit risks developed for gasoline particulate matter use the response of the organics associated with the particles in the comparative potency method. 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)$^{-1}$ (Lewtas, 1991). Maximum likelihood estimates have not been developed.

10.6.2 Other Views and Risk Estimates

International Agency for Research on Cancer (IARC)

IARC (IARC, 1989) has not developed a potency for gasoline engine exhaust but has classified gasoline engine exhaust into cancer weight-of-evidence Group 2B. Agents classified into this category are considered to be *possible* human carcinogens. This classification is based on inadequate evidence for carcinogenicity of gasoline engine exhaust in humans. There is also inadequate evidence in animals with whole gasoline engine exhaust, but sufficient evidence exists for carcinogenicity in animals using condensate/extracts of gasoline engine exhaust.

The condensate/extract of gasoline engine exhaust has been tested by skin painting, subcutaneous injection, intratracheal instillation, or implantation into the lungs in mice, rats, and Syrian hamsters. There were excess skin tumors, lung tumors, and tumors at the injection site in the studies cited.

No consistent increase in lung tumors could be detected following exposure of either animals or humans to whole gasoline engine exhaust.

10.6.3 Recent and Ongoing Research

None are available at this time.

10.7 Pro Forma Carcinogenic Risk

The cancer incidences calculated below are based on 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 below should be considered *pro forma*.

Table 10-2 summarizes annual *pro forma* cancer incidences for all the scenarios. For estimating *pro forma* annual cancer incidences, the gasoline unit risk for catalyst vehicles based on particulate matter was used. 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 emissions are higher, particularly in the out years. The projected increase in both population and vehicle miles traveled (VMT) from 2000 to 2010

Table 10-2. Annual Pro Forma Cancer Incidence Projections for Gasoline Particulate Matter.[a,b]

Year-Scenario	Emission Factor g/mile	Urban Cancer Cases	Rural Cancer Cases	Total Cancer Cases	Percent Reduction from 1990	
					EF	Cancer
1990 Base Control	0.0198	79	14	93	–	–
1995 Base Control	0.0110	46	8	54	44	42
1995 Expanded Reformulated Fuel Use	0.0107	45	8	53	46	43
2000 Base Control	0.0078	33	6	39	61	58
2000 Expanded Reformulated Fuel Use	0.0073	32	6	38	63	59
2000 Expanded Adoption of California Standards	0.0075	32	6	38	62	59
2010 Base Control	0.0062	29	5	34	69	63
2010 Expanded Reformulated Fuel Use	0.0060	27	5	32	70	66
2010 Expanded Adoption of California Standards	0.0056	26	5	31	72	67

[a]Projections have inherent uncertainties in emission estimates, dose-response, and exposure.
[b]Cancer incidence estimates are based on upper bound estimates of an unofficial EPA unit risk. This unit risk was determined from animal studies using the comparative potency method.

appears to offset some of the gains in emissions achieved through fuel and vehicle modifications.

Please note that the *pro forma* cancer unit risk estimates for gasoline particulate matter are derived using the comparative potency method and are considered an upper bound estimate for human risk. True human cancer risk may be as low as zero, but are unlikely to be greater.

10.8 Non-carcinogenic Effects of Inhalation Exposure to Gasoline Particulate Matter

No studies exist that specifically address gasoline particulate matter. The studies cited previously in Section 9.8 relating to PM_{10} in general are applicable to both diesel and gasoline particulate matter. These studies are from the Federal Register (EPA, 1987) and were those that were utilized to establish a final National Ambient Air Quality Standard (NAAQS) for PM_{10} (particulate matter less than 10 microns in diameter) with a substantial margin of safety. Please refer to Section 9.8 for this information.

References for Chapter 10

California Air Resources Board. 1986. Particulate Exhaust Emissions: Gasoline Powered Vehicles. Emissions Studies Section, El Monte, CA. Project no. 2R8618.

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.

Environmental Protection Agency. 1985. Size Specific Total Particulate Emission Factors for Mobile Sources. Office of Mobile Sources, Emission Control Technology Division, Ann Arbor, MI. August, 1985. EPA Report No. EPA-460/3-85-005.

Environmental Protection Agency. 1987. Federal Register, 40 CFR Part 50, July 1, 1987, pages 24634-24669.

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. Office of Air Quality Planning and Standards, Research Triangle Park, NC. July, 1989. EPA Report No. EPA-450/2-89-012a.

Hammerle R. H., T. J. Korniski, J. E. Weir, E. Chladek, C. A. Gierczak, R. E. Chase, and R. G. Hurley. 1992. Effect of Mileage Accumulation on Particulate Emissions from Vehicles Using Gasoline with Methylcyclopentadienyl Manganese Tricarbonyl. SAE Paper 920131.

International Agency for Research on Cancer. 1989. IARC monographs on the evaluation of carcinogenic risks to humans; diesel and gasoline engine exhausts and some nitroarenes, Volume 46.

Lang, J. M., L. Snow, R. Carlson, F. Black, R. Zweidinger, and S. Tejeda. 1981. Characterization of Particulate Emissions from In-Use Gasoline-Fueled Motor Vehicles. SAE Paper 811186.

Lewtas, J. 1991. Carcinogenic risks of polycyclic organic matter (POM) from selected emission sources. Health Effects Research Laboratory, Research Triangle Park, NC. Deliverable report no. 3128.

National Research Council. 1982. Impacts of diesel-powered light-duty vehicles: diesel technology. National Academy Press, Washington, D.C.

Smith, L. R. 1981. Characterization of Exhaust Emissions from High-Mileage Catalyst-Equipped Automobiles. Ann Arbor, Michigan: U.S. Environmental Protection Agency, Office of Mobile Sources. Publication no. EPA-460/3-81-024.

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.

Volkswagen. 1991. Unregulated Motor Vehicle Exhaust Gas Components. Volkswagen AG, Research and Development, Wolfsburg, Germany.

11.0 GASOLINE VAPORS

11.1 Chemical and Physical Properties

Unleaded gasoline is a refined product of crude oil (petroleum) composed of a complex mixture of hydrocarbons, additives, and blending agents. This mixture of hydrocarbons that distills within the range of 100 to 400°F is comprised of paraffins (alkanes), olefins (alkenes), and aromatics. Compounds containing sulfur, nitrogen, and oxygen are also present in the gasoline refinery streams. Different refinery streams are blended to achieve industry specifications for physical properties, including boiling point range and vapor pressure, and desired seasonal performance standards, such as octane rating, to minimize pre-ignition or knock. Additives and blending agents are added to improve the performance and stability of gasoline. Blending agents, such as tert-butyl alcohol, methyl tert-butyl ether (MTBE), and other alcohols and ethers are used in unleaded gasoline as replacements for organometallic anti-knock agents such as tetraethyl lead. Trace concentrations of various elements including manganese, chromium, zinc, copper, iron, boron, magnesium, lead, and sulfur are also present in most gasolines. For the most part, these metals are native to the crude oil prior to refining.

Gasoline exists in two phases, liquid and vapor, with the hydrocarbon compositions being different. Liquid gasoline consists principally of 66 to 69 percent paraffins (alkanes), 24 to 27 percent aromatics, and 6 to 8 percent olefins (alkenes) (Battelle, 1985). Unleaded gasoline contains higher concentrations of isoparaffins and aromatics than does leaded gasoline. Typical gasoline product lines may contain more than 150 separate compounds. Gasoline vapors consist mainly of short-chained and iso- alkanes and their percent in gasoline vapors, as determined by four separate studies, are detailed in Table 11-1.

Table 11-1. Vapor Composition of Gasoline (Volume %).

	McDermott	Halder	Battelle	Runion	Liquid Phase
Alkanes	84	93	92	92	66-69
Alkenes	6	2	5	8	6-8
Aromatics	3	4	5	1	24-27

SOURCE: McDermott and Killiany, 1978; Halder et al., 1984; Battelle, 1985; Runion, 1975.

11.2 Exposure Estimation

Emissions of gasoline vapors to the atmosphere occur throughout the entire process of fuel handling and marketing. This process begins at the refinery and continues through bulk loading, transport, and unloading operations, then down to the service stations where vehicle refueling occurs. Gasoline vapors are also released from the vehicle itself through evaporative and tailpipe emissions. Vapors released into the atmosphere are subject to the processes of transport, dilution, and dispersion, thus, spreading the vapor over a wide area. Due to the differences in the partial pressure of various hydrocarbons, gasoline vapors emitted in the manner described above, consist of relatively more of the lighter compounds (e.g., alkanes) and less of the heavier ones (e.g., branched alkanes) than liquid fuel.

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 principle exposure pathways are from the ambient air, gasoline migration into the basements of homes, and the ingestion of gasoline contaminated groundwater (NESCAUM, 1989). 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.

NESCAUM (1989) attempted to estimate the occupational and the general population exposure to gasoline vapors based on six separate scenarios. This information is summarized from NESCAUM (1989) in Table 11-2. The exposure information is based upon existing monitoring studies and limited case-study information.

11.3 Carcinogenicity of Gasoline Vapors and Unit Risk Estimates

11.3.1 Most Recent EPA Assessment

The information presented here was obtained from EPA's Evaluation of the Carcinogenicity of Unleaded Gasoline (EPA, 1987a). In the development of this document, the scientific literature has been reviewed through 1985. New issues concerning the carcinogenicity of gasoline vapors will be presented in Section 11.3.3, which summarizes recent and ongoing research not included in the 1987 EPA evaluation.

11.3.1.1 Description of Available Carcinogenicity Data

Genotoxicity

Mutagenesis tests of unleaded gasoline have been carried out in *Salmonella* (Litton Bionetics, 1977), yeast (Litton Bionetics,

Table 11-2. Summary of Ambient Concentrations and Exposure Doses Associated with Exposure to Gasoline and Benzene (NESCAUM, 1989).

Scenario	Ambient Concentrations (mg/m^3)		Estimated Exposure Doses based on alveolar ventilation (mg/kg/day)	
	Mean	Maximum	Mean	Maximum
Scenario 1: Self-service customer at gas station exposed via inhalation.				
Gasoline	369.8	1882.3	9.4×10^{-3}	1.0×10^{-1}
Benzene	2.9	13.4	7.3×10^{-5}	7.2×10^{-4}
Scenario 2: Gas station attendant exposed via inhalation.				
Gasoline	54.6	–	1.8	–
Benzene	0.6	4.1	$21. \times 10^{-2}$	1.4×10^{-1}
Scenario 3: Resident living downwind of gas station exposed via inhalation.				
Gasoline	1.5×10^{-2}	7.7×10^{-2}	3.1×10^{-3}	1.6×10^{-2}
Benzene	1.3×10^{-4}	5.1×10^{-4}	2.6×10^{-5}	1.1×10^{-4}
Scenario 4: Resident inhaling vapors from nearby leaking underground storage tank.				
Gasoline	–	–	–	–
Benzene	1.8	9.3	3.6×10^{-1}	1.9
Scenario 5: Resident exposed to gasoline via ingestion of contaminated well water.				
Gasoline	16	276	1.0×10^{-1}	2.9
Benzene	1.6	7.3	1.4×10^{-2}	7.0×10^{-2}
Scenario 6: Resident exposed via inhalation and dermal contact during showering.				
Gasoline	NA	NA	1.7×10^{-1}	3.4×10^{-1}
Benzene	NA	NA	1.4×10^{-2}	2.8×10^{-2}

1977), and mouse lymphoma *in vivo* cytogenetics (Phillips Petroleum, 1984; Litton Bionetics, 1977) to determine mutation frequency. These studies, when taken collectively, indicate no increase in the mutational frequency of unleaded gasoline. A study by Phillips Petroleum (1984) to determine sister chromatid exchanges in Chinese hamster ovary cells was also negative except at the highest dose with metabolic activation.

In a series of *in vivo* studies conducted by Litton Bionetics (1977), rats received acute and subacute intraperitoneal injections of unleaded gasoline. The rate of mutation in the rat bone marrow cells showed aberrations at the intermediate dose levels, but no dose-response was observed, and the subchronic studies showed no increase in chromosomal aberrations. Litton Bionetics (1977) also administered unleaded gasoline orally for 5 days at three concentrations which yielded no significant increases in chromosomal aberrations.

Fregda et al. (1979) evaluated chromosomal aberrations among 65 males occupationally exposed to gasoline and benzene through fuel handling. There was a significant increase in frequency of chromosomal aberrations in all road tanker drivers but it may not have been primarily due to benzene exposure since both those delivering gasoline and milk had the same incidence of chromosome aberrations. Fregda et al. (1982) also investigated the incidence of chromosomal changes in men occupationally exposed to automobile fuels and exhaust gases. The chromosomal analysis showed that smokers exposed to either gasoline or diesel fuel had a higher frequency of sister chromatid exchanges (SCE) than did the non-smokers. However, the authors were unable to determine an association between the work environment and frequency of chromosomal changes.

Taken collectively, the results of these assays have not met the criteria for positive responses for genotoxicity.

Animal Data

A lifetime inhalation bioassay of aerosolized whole unleaded gasoline (zero, 67, 292, and 2056 ppm exposures) in Fischer 344 rats and B6C3F1 mice (International Research and Development Corporation (IRDC), 1982; MacFarland et al., 1984) induced a significant increase in renal carcinomas in the kidney cortex of male rats and a larger, also significant increase in hepatocellular (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 (2056 ppm). However, if renal adenomas, carcinomas, and sarcomas are combined, the increase was significant at both the high and intermediate dose levels. In mice, the increase in the incidence of liver carcinomas alone and adenoma and carcinomas combined was statistically significant only in the highest dose group. Moderate decrements in body weight gain in the high-dose groups indicate that the maximum tolerated dose was reached.

Glomerulonephrosis occurred in nearly all male rats, and mineralization of the pelvis was correlated with dose. However, there was no correlation between animals with tumors and those with mineralization.

MacFarland (1983) conducted a 90-day inhalation exposure study of the toxicity of unleaded gasoline vapor in Sprague-Dawley rats and squirrel monkeys as a prechronic test in preparation for the carcinogenicity study with unleaded gasoline in rats and mice. Rats and monkeys were exposed 6 hours/day, 5 days/week for 13 weeks to totally vaporized unleaded EPA reference gasoline (exposures of 0, 384, and 1552 ppm) and a leaded commercial gasoline (0, 103, 374 ppm). The animals were examined for mortality, body-weight, food consumption, toxic signs, hematological changes, urinary changes, tissue lead levels, pathology, and pulmonary tests (monkeys only). Many changes were observed in the above mentioned areas in both the rats and monkeys. Most changes were decrements in various organ weights, hematological components, and respiratory functions at the highest exposure level for both gasolines.

The initial pathological examinations showed no treatment related effects. Histopathologic reexamination of tissue sections showed subtle but discernible changes in the kidneys of the high exposure, unleaded gasoline group of male rats.

The acute and subchronic renal toxicity of decalin (Alden et al., 1983), a volatile hydrocarbon of the same general type as those contained in gasoline, is confined to male rats and does not occur in female rats or in mice, dogs, or guinea pigs. In a series of 21-day inhalation exposures of male rats to a variety of chemical fractions of gasoline (Halder et al., 1984), renal toxicity was correlated with the paraffin components, specifically, those having C6-C9 carbons with one or more branches, and not with the aromatic compounds in the mixture. In a four week study conducted by Tegeris Laboratories (1985), rats were administered by oral gavage a variety of C8 and C10 branched hydrocarbons. It was determined that two C8 branched compounds produced severe to moderate effects in the induction of kidney toxicity.

The same pattern of renal toxicity as well as positive renal tumor response occurs in response to chronic inhalation of two synthetic fuels (RJ-5 and JP-10) (MacHaughton and Uddin, 1983). Chronic inhalation studies with the jet fuels used by the Air Force and Navy (JP-4 and JP-5) have shown the same nephrotoxic lesions, but information on the carcinoma response is not available.

The Universities Associated for Research and Education in Pathology, Inc. (UAREP, 1983) analyzed the toxicology and carcinogenicity of unleaded gasoline and other hydrocarbons. This group agrees that a renal toxicity pattern is observed with exposure to hydrocarbon mixtures in Fischer 344 and Sprague-Dawley rats, involving protein accumulation in renal tubules, but

it is clearly different than the kidney lesions occurring spontaneously in old rats. The lesions do not occur in females of these strains or in mice or monkeys. Old-rat nephropathy was morphologically different from preneoplastic lesions in the API carcinogenicity study (IRDC, 1982; MacFarland et al., 1984) of unleaded gasoline vapor. However, a possible causal relationship between old-rat nephropathy and toxic lesions from exposure to unleaded gasoline vapor cannot be ruled out.

Human Data

There were fifty-five studies reviewed by the EPA in its evaluation of the carcinogenicity of unleaded gasoline (EPA, 1987a) to determine if there is any epidemiological evidence for an association between gasoline exposure and cancer risk. Only a cursory review of these studies will be included in this chapter; for more detail, consult EPA (1987a). Since unleaded gasoline was only introduced in the mid-1970's, many epidemiological studies are not likely to show an unleaded gasoline effect because of the long latency period generally associated with cancer. Therefore, the EPA review was not limited to unleaded gasoline exposure, but addressed any potential gasoline exposure. None of the studies reviewed provided qualitative as well as quantitative estimates of gasoline exposure (EPA, 1987a).

Seven studies were identified that evaluated the association between employment in the gasoline service industry and cancer risks; the industry here includes gasoline service station owners and attendants, garage workers, gasoline and fuel truck drivers, and these who reported working with gasoline. The study by Stemhagen et al. (1983) provided some evidence of an association between gasoline service station employment and risk of primary liver cancer. The remaining six studies were judged inadequate.

Twenty-five studies were reviewed that evaluated the association between employment in a petroleum refinery (a work environment with potential gasoline exposure) and cancer risk. Judged individually, these studies provided inadequate evidence of an association. However, judged collectively these studies provide suggestive evidence of an association between employment in a petroleum refinery and risk of stomach cancer, respiratory system cancer (i.e., lung, pleura, nasal cavity and sinuses), and cancer of the lymphatic and hematopoietic tissues.

Nineteen case-control studies were reviewed which evaluated employment in the petroleum industry as a cancer risk factor. The study by Howe et al. (1980) provided limited evidence of an association between petroleum industry employment and risk of bladder cancer.

Overall, these epidemiological studies provide limited evidence that employment in the petroleum industry is associated with certain types of cancer. However, the epidemiologic evidence for evaluating gasoline as a potential carcinogen is considered inadequate.

11.3.1.2 Weight-of-Evidence Judgement and EPA Classification

EPA classifies gasoline vapor as a Group B2, probable human carcinogen. This is based on sufficient evidence in animals (IRDC, 1982; MacFarland et al., 1984) that inhalation of wholly vaporized gasoline is carcinogenic. Although employment in the petroleum refineries is possibly associated with cancers of the stomach, respiratory system, and lymphopoietic and hematopoietic tissues, exposure to gasoline cannot be implicated as a causative agent because of confounding exposure to other chemicals and inadequate information on gasoline exposure. Gasoline vapors from vehicle refueling might be less carcinogenic than indicated by animal experiments using wholly aerosolized gasoline, since it is the less volatile components that apparently contribute to the carcinogenic response.

11.3.1.3 Data Sets Used for Unit Risk Estimate

The chronic inhalation study of unleaded gasoline vapor conducted by the International Research and Development Corporation (IRDC, 1982; MacFarland et al., 1984) and sponsored by the American Petroleum Institute (API) is the only study that can be used to derive the carcinogenic potency of aerosolized whole unleaded gasoline. This study is summarized in Section 11.3.1.1.

11.3.1.4 Dose-Response Model Used

The linearized multistage model is used to calculate the unit risk estimate for wholly vaporized gasoline. The unit risk estimate that currently exists for gasoline vapors is based exclusively on animal data.

11.3.1.5 Unit Risk Estimate

In the calculation of unit risk, ppm in air is assumed to be equivalent between animals and humans. The data from the highest dose group have been excluded from the calculation because the model does not fit well if these data are included and the data seem to indicate a toxic effect in the highest dose group. The results of the calculation are presented in Table 11-3 (EPA, 1987a). Both the upper bound estimate and the maximum likelihood estimate are given. The kidney data in rats and the combined hepatocellular adenoma/carcinoma data in mice are similar, spanning a range from $2.1 \times 10^{-3} (ppm)^{-1}$ to $3.5 \times 10^{-3} (ppm)^{-1}$.

**Table 11-3.   Estimates of Carcinogenic Potency Due to Exposure
to 1 ppm of Leaded Gasoline Vapor (EPA,1987a).**

Data Base	Upper Bound Estimate	Maximum Likelihood Estimate
(1) Kidney tumors in male rats	3.5×10^{-3}	2.0×10^{-3}
(2) Hepatocellular carcinoma/adenoma in female mice	2.1×10^{-3}	1.4×10^{-3}
Hepatocellular carcinoma in female mice	1.4×10^{-3}	8.5×10^{-4}
Geometric Mean of (1) and (2)	2.7×10^{-3}	1.7×10^{-3}

11.3.2 Other Views and Unit Risk Estimates

IIT Research Institute

According to the results of subchronic studies sponsored by
API, the 0° to 145°F distillate fraction (lighter, more volatile)
of unleaded gasoline as well as C4-C5 (short-chain) olefinic
(alkene) hydrocarbons, which are the primary components of this
fraction, did not cause detectable nephrotoxicity in rats (IIT
Research Institute, 1985a,b). It was further shown that the 145°
to 280°F fractions contained most of the nephrotoxic activity and
that the specific compounds responsible for most of the toxicity
were branched-chain olefins (alkene) 6 to 9 carbons in length.
Since the 0° to 145° distillate fraction is much more volatile at
ambient temperatures than the heavier distillates, the vapor
fraction would be expected to contain a much greater percentage
of short-chain hydrocarbons. Thus, the composition of whole
aerosolized gasoline used in chronic cancer assessments and
gasoline vapors at ambient temperatures results in a degree of
uncertainty. There are no data to indicate which fraction is
responsible for induction of liver tumors in mice.

Cancer Risk Attributable to Benzene Content in Gasoline Vapor

EPA (1987a) estimates the cancer incidence which could be
attributed to the benzene content of gasoline vapor. To
accomplish this, two assumptions were made; the tumor response to
benzene is additive to the response produced by other components
of gasoline and a particular air concentration produces the same
effect in animals and humans. While benzene in gasoline may be
additive with other components in terms of total tumor response,
it is unlikely to be additive for the types of tumors detected.
Gasoline exposure induced kidney and liver tumors, whereas,
benzene is known to induce other tumors such as leukemia. The
benzene component was calculated to account for about 20% of the

total response. This small incidence was determined to be undetectable, since 20% of the kidney cancer incidence (16%) at the high dose (MacFarland et al., 1984) is only 0.2×16% = 3%, which is only about one animal out of 45 in that group.

There is no evidence from the benzene or gasoline literature to support or deny the additivity assumption. There is abundant evidence that a carcinogen or noncarcinogen could modify (enhance or inhibit) the carcinogenic action of another compound. Since gasoline vapor contains more than one chemical compound, such interactive effects are possible.

Health Effects Institute 1985 and 1988

The Motor Vehicle Manufacturers Association (MVMA) and the EPA requested that the Health Effects Institute (HEI) undertake a review of the issues concerning potential adverse health effects of exposure to vapors of unleaded gasoline (HEI, 1985). On the basis of the study by MacFarland et al., (1984), HEI concluded that wholly vaporized gasoline is an animal carcinogen and a presumptive human carcinogen. Taken collectively, the epidemiological studies were considered to provide weak evidence, but no proof of an association between exposure to petroleum vapors and an increase in kidney cancer. Thus, the available epidemiological evidence can neither confirm or deny the association between gasoline vapor and possible human carcinogenicity.

HEI considers the development of a realistic quantitative risk assessment too difficult to resolve or decide due to several uncertainties. Uncertainties remain regarding the difference in composition of gasoline vapors to which humans are exposed and the wholly vaporized gasoline used in the animal studies and because of the different patterns of exposure in humans and experimental animals. On the basis of the available animal studies and established guidelines, HEI agreed that the existing data can be used to calculate an upper-bound risk factor for gasoline vapors as EPA has done. HEI cautioned that the health hazard, if any, cannot be established without additional data, and that the actual risk may be anywhere between zero and the upper bound.

In January of 1988, HEI issued an update (HEI, 1988) on the issue of gasoline vapor and human cancer, evaluating the scientific information published between 1985 and 1987. This information will be summarized in the following section on recent and ongoing research. HEI concluded that the research conducted since 1985 has reduced some of the uncertainty surrounding the issues that HEI detailed in 1985. The new research lends additional support to the cautious approach adopted at that time: that the information is not available to draw accurate conclusions concerning the degree of human risk that results from exposure to gasoline vapors.

Northeast States for Coordinated Air Use Management (NESCAUM)

In its 1989 report (NESCAUM, 1989), NESCAUM concluded that gasoline and at least one of its major constituents, benzene, are presumed human carcinogens. Exposure to gasoline and its components is also associated with other adverse health effects such as toxicity to the hematopoietic, kidney, liver, reproductive/developmental, and nervous systems. NESCAUM agrees that many uncertainties still exist. The major uncertainties being that the vapor composition in MacFarland et al., (1984) animal study is different than ambient human exposure and that the kidney tumors observed in male rats may be the result of a mechanism specific to the male rat and not female rats of other species.

Potential individual lifetime (70 years) cancer risks associated with exposure to unleaded gasoline and benzene are presented in Table 11-4. These cancer risks are based on a cancer potency value of 0.0035 per mg/kg/day for gasoline and 0.026 mg/kg/day for benzene. The exposure doses corresponding to one in a million cancer risk for gasoline and benzene are estimated to be 2.8×10^{-4} mg/kg/day and 3.8×10^{-5} mg/kg/day, respectively. NESCAUM uses a variety of research studies, case studies (most specific to the northeast states), and computer models for each of the six scenarios to derive the estimated lifetime cancer risk. Several uncertainties exist, including the following: estimates may exclude gasoline components of potential concern; inaccuracies in the assumptions about the intensity and duration of exposure; lack of information on interactive effects among constituents in the complex mixture; and uncertainties associated with exposure of sensitive individuals. These and other uncertainties cause NESCAUM to develop a conservative approach, when possible.

Motor Vehicle Manufacturers Association (MVMA)

The MVMA petitioned the EPA (MVMA, 1991) to reexamine the evidence for gasoline vapor carcinogenicity and, in light of new information, to reclassify the category of gasoline vapors from Group B2 (probable) to C (possible human carcinogen). MVMA feels this reclassification is essential so that the need for public protection from the risk of cancer due to exposure to evaporated gasoline can be properly assessed and any proposed regulatory action involving vapor can be appropriately updated. MVMA states that the animal tumors are of questionable relevance and there is inadequate epidemiological evidence for predicting the true human risk. MVMA cites HEI (1985;1988), EPA (1987a), EPA (1991b) discussed in Section 11.3.3.1, and the SAB concurrence with that document to support their position. MVMA states that this new

Table 11-4. Potential Cancer Risks Associated with Exposure to Gasoline and Benzene (NESCAUM, 1989).

Exposure Scenario	Mean Lifetime Cancer Risk-Gasoline (risk/person/lifetime)	Mean Lifetime Cancer Risk-Benzene (risk/person/lifetime)
Self-service customer at gas station exposed via inhalation.	3.3×10^{-5}	1.9×10^{-6}
Gas station attendant exposed via inhalation.	6.3×10^{-3}	5.5×10^{-4}
Resident living downwind of gas station exposed to inhalation.	1.1×10^{-5}	6.8×10^{-7}
Resident inhaling vapors from nearby leaking underground storage tank.	--	9.4×10^{-3}
Resident exposed to gasoline via ingestion of contaminated well water.	6.0×10^{-4}	3.6×10^{-4}
Resident exposed via inhalation and dermal contact during showering.	6.0×10^{-4}	3.6×10^{-4}

information indicates that gasoline vapor exposure does not pose significant risks to humans.

EPA Response to MVMA Petition Letter

In response to MVMA's letter (EPA, 1991a) the Agency has not initiated any specific effort to re-examine the weight-of-evidence for gasoline vapors based on the new tumor evaluation criteria (EPA, 1991b). 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.

11.3.3 Recent and Ongoing Research

The information contained below summarizes the information released since EPA (1987a). This document reviewed the literature available through 1985.

11.3.3.1 Alpha$_{2u}$-globulin: Association with Chemically Induced Renal Toxicity and Neoplasia in the Male Rat

EPA's Risk Assessment Forum report, <u>Alpha$_{2u}$-globulin: Association with Chemically Induced Renal Toxicity and Neoplasia in the Male Rat</u> (EPA, 1991b), provides Agency wide guidelines for evaluating renal tubule tumors in the male rat. According to this report, risk assessment approaches generally assume that a chemical producing tumors in laboratory animals is a potential hazard to humans. For most chemicals, including many rodent kidney carcinogens, this assumption remains suitable. The report describes to EPA risk assessors the scientific conditions under which the information on certain renal tubule tumors or nephrotoxicity should not be used to assess human risk. In this situation, the chemical in question induces accumulation of the protein alpha$_{2u}$-globulin in the proximal tubule of the male rat kidney. This initiates a sequence of events, specific to the male rat kidney, that appears to lead to renal tubule tumors. This EPA policy is an important change in EPA's general approach to cancer risk assessment.

The Forum report stresses the need for a full review of a substantial data set to determine when it is reasonable to presume that renal tumors in male rats are linked to a process involving alpha$_{2u}$-globulin accumulation. Only then can the appropriate procedures be selected to estimate human risks under such

circumstances. Complete details of this analysis can be found by referring to the Risk Assessment Forum report (EPA, 1991b).

11.3.3.2 Genotoxicity

Dooley et al. (1987) tested unleaded gasoline, a DMSO (dimethylsulfoxide) extraction of unleaded gasoline, and an evaporative residue of unleaded gasoline for their ability to induce mutation in a modified Ames bioassay performed with and without activation. No increase in reverent colonies were observed for unleaded gasoline or the unleaded gasoline extraction. A reduction in the revertant colonies at the highest doses indicated that the unleaded gasoline extraction was toxic, but not mutagenic. The evaporative residue induced a less than two-fold increase in the mutant colonies, and, therefore, was not considered mutagenic in the assay.

Dooley et al. (1987) also tested unleaded gasoline, extraction of unleaded gasoline, and an evaporative residue for increases in the mutational frequency in the mouse lymphoma cells assay. Dooley et al. (1987) reported that at concentrations of unleaded gasoline yielding 10 percent or greater cell survival, no appreciable increase in mutation frequency with or without activation was observed. There was a greater mutational frequency observed at concentrations yielding less than 10 percent growth, but this was not considered mutagenic. It is suggested by the authors that unleaded gasoline may contain weakly mutagenic components that are masked by the toxicity of the total mixture.

Richardson et al. (1986) reported no significant increases in the mutational frequency or sister chromatid exchange frequency in human lymphoblast tested with unleaded gasoline, 2,2,4-trimethylpentane (branched C5 alkane found in gasoline), and a volatile fraction of unleaded gasoline.

Loury et al. (1986) conducted a series of *in vivo* and *in vitro* assays to assess the ability of unleaded gasoline and 2,2,4-trimethylpentane to induce unscheduled and replicative DNA synthesis in Fischer 344 rats, B6C3F1 mice, and human hepatocytes. Unscheduled DNA synthesis is a measurement of DNA repair, and therefore, an indicator of genotoxic activity. Replicative DNA synthesis is an indirect measure of cell proliferation activity.

In the unscheduled DNA synthesis assays, there are many results. In the assays involving *in vivo*/*in vitro* hepatocytes exposed to unleaded gasoline by gavage, the rat hepatocytes showed no increase, whereas the mouse hepatocytes exhibited a statistically significant increase in activity in male and female mice 12 hours after treatment. In the assays that were exclusively *in vitro*, unleaded gasoline produced statistically significant increases in DNA activity in the male mouse hepatocytes and a dose-dependent increase in activity in the rat hepatocytes. The highest doses which produced unscheduled DNA

synthesis in rat hepatocytes were toxic to mouse and human cells. Trimethylpentane yielded no positive results.

In the second part of the experiments, both unleaded gasoline and trimethylpentane were tested for their effects on replicative DNA synthesis. *In vitro/in vivo* exposure of both rat and mouse hepatocytes to unleaded gasoline produced no statistically significant results. When both were exposed to 2,2,4-trimethylpentane, the replicative DNA synthesis was significantly increased.

Loury et al. (1987) also assessed the ability of unleaded gasoline to induce unscheduled DNA synthesis and replicative DNA synthesis in kidney cells of Fischer 344 rats exposed *in vivo* and *in vitro* by gavage and inhalation. The purpose of the study was to determine whether the induction of kidney tumors by unleaded gasoline is related to genotoxicity or cell proliferative effects. The *in vivo/in vitro* assay for unscheduled DNA synthesis produced no detectable unscheduled DNA synthesis by either gavage or inhalation. There was a significant increase in replicative DNA synthesis activity in male kidneys but not female rat kidneys.

Dooley et al. (1987) also performed the dominant lethal assay of sperm cells of CD-1 mice exposed to unleaded gasoline by inhalation. These experiments produced only a non-significant increase in pre- and post-implantation loss of embryos as compared to controls.

11.3.3.3 Metabolism and Pharmacokinetics

Overall, there is not an abundance of information on the action of unleaded gasoline in the human body. What is known is that, once absorbed in to the blood, the gasoline components partition themselves between the plasma and serum. Binding to serum proteins reduces the amount of free compounds available for exchange with air and tissues. Bound and unbound gasoline components are transported through the blood to various tissues.

Tissue clearance is accomplished by the metabolism of the parent hydrocarbons into more polar compounds, which can be excreted more efficiently. Many hydrocarbons are metabolized by enzyme systems in the liver, although metabolism may occur in other organs as well. Metabolic efficiency is dependent on several variables, including the tissue concentration, the affinity of the specific compound for the enzyme system, the enzyme interactions with other gasoline components, the nutritional status of the individual, exposure to other chemicals, the influence of metabolism (e.g., ethanol), and the presence or absence of tissue injury.

Much, but not all, of the pharmacokinetic data that has been accomplished since the publication of EPA (1987a) has been devoted to trying to determine the mechanism involved in the

development of the chemically-induced kidney tumors observed in the male rat. The brief summary that follows is excerpted from a Chemical Industry Institute of Toxicology technical report (CIIT, 1991) which also contains the qualitative and quantitative evidence used to support the position. Other mechanistic information from 1986 and 1987 is summarized in HEI (1988).

Alpha$_{2u}$-globulin is synthesized and secreted at a rapid rate from the liver into the blood of the male, but not female rats. Approximately 50% of the protein filtered by the kidney is resorbed by the proximal tubule cells (PTC) of the kidney. Chemical treatment does not appear to increase the synthesis of alpha$_{2u}$-globulin in the liver or change the amount that is resorbed by the PTC. The most plausible explanation for the accumulation of alpha$_{2u}$-globulin in the kidney cells is that the rate of hydrolysis of alpha$_{2u}$-globulin in the kidney is decreased when the protein is bound to the chemical. This then results in protein droplet accumulation and increased cell proliferation in the PTC. This has been observed in male rats exposed to unleaded gasoline or 2,2,4-trimethylpentane.

Chemical-mediated accumulation of alpha$_{2u}$-globulin is thought to be responsible for killing cells, which in turn stimulates cell division as the kidney attempts to repair itself. With prolonged exposure, repeated cycles of cell death and reparative replication are proposed to be responsible for the observed tumorigenic response.

11.3.3.4 Carcinogenicity - Animal Studies

Studies have been accomplished linking other chemicals to the production of lesions in the male rat kidney. Similar findings have been reported for decalin (Kanerva et al., 1987a), d-limonene (Kanerva et al., 1987b), 1,4-dichlorobenzene (Charbonneau et al., 1988), and 2,2,4-trimethylpentane (Stonard et al., 1986). Some of these compounds also cause a sex-specific increase in renal adenomas and/or carcinomas in rats. The three compounds decalin, d-limonene, and trimethylpentane have been used as model compounds for studying the mechanisms of renal toxicity because of their ability to produce the characteristic lesions.

Two studies were conducted that evaluated the renal toxicity associated with the light hydrocarbons. These hydrocarbons are more characteristic of the major fraction of ambient gasoline vapors. These two inhalation experiments (Halder et al., 1986; Aranyi et al., 1986) used Sprague-Dawley rats exposed to vapor mixtures representative of ambient gasoline vapors. In both studies, there was no evidence of nephrotoxicity in rats of either sex after either 3 or 13 weeks of exposure.

All the rest of the animal research has been conducted to further the understanding of the mechanisms involved in the toxicity and potential carcinogenicity of hydrocarbons in the male rat kidney due to gasoline vapor exposure. The results of

this body of work were summarized in Section 11.3.3.3. Little
research has been directed toward furthering a better
understanding of the role of hydrocarbons in the induction of
female mouse liver tumors observed in MacFarland et al. (1984).

11.3.3.5 Carcinogenicity - Epidemiological Studies

In a literature survey by Harrington (1987), 22 cohort
studies and 19 case-control studies of the health experience of
workers in the petroleum manufacturing and distribution industry
published between 1972 and 1986 were reviewed. The standard
mortality ratio for kidney cancer was elevated for exposed
workers in two studies, decreased in four studies, and unchanged
in three studies. Taken collectively, the epidemiological
studies observed do not provide support for and association
between exposure to petroleum hydrocarbons and renal cancer.

In a study not reviewed by Harrington (1987), McLaughlin et
al. (1985) examined renal cell carcinoma in relation to
employment in the petroleum industry. This study was based on
data collected in a case-control study of renal carcinoma in the
Minneapolis-St. Paul area (McLaughlin et al., 1984). In this
analysis of the 1984 study, using only male data, no evidence was
produced to support a positive association between kidney cancer
and employment in the petroleum industry.

Schwartz (1987) conducted a proportionate mortality ratio
analysis of automobile mechanics and gasoline service station
workers in New Hampshire. There were significant increases in the
association between workplace and the incidence of suicide and
leukemia, but no significant increases in other tumors.

11.4 Pro Forma Carcinogenic Risk

The cancer incidences in Table 11-5 below are based on
uncertain exposure estimations and an unofficial EPA unit risk
estimate. The unit risk estimate, as mentioned above, is based
on wholly vaporized gasoline and may not be representative of
actual exposure. Furthermore, when considering the other views
and the recent and ongoing research summarized in Sections 11.3.2
and 11.3.3, it is reasonable to assume that the gasoline vapor
risk estimates are conservative and more highly uncertain than
the risk estimates for the other pollutants examined in this
report. Due to these factors, the cancer incidences should be
considered *pro forma*.

Table 11-5. Summary of Annual Average Baseline Risks (1988 to 2020) of Exposure to Whole Gasoline Vapor (EPA, 1987b).[a,b]

Facility Category	Annual Average Incidence	
	Benzene	Gasoline Vapors
Bulk Terminals	0.1	3.5
Bulk Plants	0.05	1.4
Service Stations		
1) Community Exposure		
a) Stage I	0.1	3
b) Stage II	0.4	10
2) Self-Service	4.4	33
Total Public Incidence	5.1	51
Occupational (Service Stations)	1.7	17
Total Incidence for Gasoline Marketing Source Category	6.8	68

[a]Projections have inherent uncertainties in emission estimates, dose-response, and exposure.
[b]Cancer incidence estimates are based on upper bound estimates of an unofficial EPA unit risk. This unit risk was determined from animal studies based on wholly vaporized gasoline and may not be representative of actual exposure.

Table 11-5 contains the baseline average annual cancer incidence from high exposure to gasoline vapor that was conducted by EPA (1987b) in a draft regulatory impact analysis. The aerosolized whole unleaded gasoline risk values are based on the upper bound estimate of the API sponsored rat studies (IRDC, 1982; MacFarland et al., 1984). The values presented in Table 11-5 are the average annual values for the study period of 1988 to 2020. The cancer cases attributed to benzene have already been accounted for by considering benzene evaporative emissions in Chapter 5.

11.5 Non-carcinogenic Effects of Inhalation Exposure to Gasoline Vapors

Since the focus of this report is on the carcinogenic potential of the various compounds, the noncancer information will be dealt with in a more cursory fashion. No attempt has been made to synthesize and analyze the data encompassed below. Also, no attempt has been made to accord more importance to one type of noncancer effect over another. The objective is to research all existing data, describe the noncancer effects observed, and refrain from any subjective analysis of the data.

Exposure to gasoline vapors through inhalation at low concentrations and/or acute exposure may cause a variety of symptoms including respiratory tract irritation and burning with cough and sore throat, and central nervous system depression with headache, nausea, and mental confusion. Higher concentrations may cause respiratory difficulty, pulmonary edema (accumulation of fluid in the lungs) or bronchial pneumonia with fever. Heart damage and further central nervous system depression may also occur with muscular incoordination, blurred vision, unconsciousness, or convulsions. Even brief exposure to high concentrations may cause unconsciousness, coma, or death from severe central nervous system depression resulting in respiratory failure (MSDS, 1985;1987;1989).

Chronic exposure to gasoline vapors may cause dizziness, weakness, nervousness, limb pain, peripheral numbness, or other abnormalities in sensation. Other effects that may develop include anorexia, weight loss, pallor, fatigue, confusion, or anemia (MSDS, 1985). An important hazard associated with chronic gasoline inhalation (of even low concentrations which are the most common) is exposure to aromatic hydrocarbons, especially benzene. Severe exposure can result in irreversible effects such as encephalopathy (any disease of the brain); aplasticanemia (low oxygen carrying capacity due to bone marrow failure); and leukemia (EPA, 1980).

The threshold limit value (TLV), or the level which is considered to be safe by NIOSH for exposure for an eight hour work day, is set at 300 ppm for gasoline. The short term exposure limit (STEL), or the level considered to be safe for a period of 15 minutes, is set at 500 ppm.

11.6 References for Chapter 11

Alden, C.L., R.L. Kanerva, G. Ridder, and L.C. Stone. 1983. The pathogenesis of nephrotoxicity of volatile hydrocarbons in the male rat. G.P. Hemstreet III, J.J. Thorpe, and M.L. Kane, eds. Proceedings of the Workshop on the Kidney Effects if Hydrocarbons, Boston, MA July 18-20. American Petroleum Institute, pp. 154-170.

Aranyi, C., W.J. O'Shea, C.A. Halder, C.E. Holdsworth, and B.Y. Cockrell. 1986. Absence of hydrocarbon-induced nephropathy in rats exposed subchronically to volatile hydrocarbon mixtures pertinent to gasoline. Toxicology and Industrial Health, 2:85-98.

Battelle. 1985. Literature evaluation of gasoline vapor toxicology. Prepared for Health Effects Institute. Richland, MI: Battelle Pacific Northwest Laboratories.

Charbonneau, M., J. Strasser, E.A. Lock, M.J. Turner, and J.A. Swenberg. 1988. 1,4-Dichlorobenzene-induced nephrotoxicity: similarity with unleaded gasoline-induced renal effects. In: Nephrotoxicity: Extrapolation from *in vitro* to *in vivo* and from animals to man, P. Bach and E.A. Lock ed.

CIIT. 1991. Protein Nephrotoxicity and Kidney Cancer in Male Rats: Qualitative and Quantitative Issues and Human Relevance. Vol. 11, No. 1, January 1991.

Dooley, J.F., M.J. Skinner, T.A. Roy, G.R. Blackbuar, C.A. Schreiner, and C.R. Mackerer. 1987. Evaluation of the genotoxicity of API reference unleaded gasoline. In: Polynuclear aromatic hydrocarbons. Proceedings of the tenth international symposium. Battelle Press.

EPA. 1980. Facts and issues associated with the need for a hydrocarbon criteria document. Office of Research and Development, Environmental Criteria and Assessment Office, Internal Document, February 1980.

EPA. 1987a. Evaluation of the Carcinogenicity of Unleaded Gasoline. EPA Report No. 600/6-87/001, April 1987.

EPA. 1987b. Draft Regulatory Impact Analysis: Proposed Refueling Emission Regulations for Gasoline-Fueled Motor Vehicles. Volume I: Analysis of Gasoline Marketing Regulatory Stategies. EPA Report No. 450/3-87-001a, July 1987.

EPA. 1991a. Letter from William Farland of EPA to Thomas Carr of MVMA, October 28, 1991.

EPA. 1991b. Alpha$_{2u}$-Globulin: Association with Chemically Induced Renal Toxicity and Neoplasia in the Male Rat. Risk Assessment Forum, Washington, D.C. EPA Report No. 625/3-91/019F, September 1991.

Fregda, K., J. Reitalu, and M. Berlin. 1979. Chromosome studies in workers exposed to benzene. In: Genetic Damage in Man Caused by Environmental Agents, Vol. 1, pp. 187-203. Edited by E.K. Berg. New York: Academic Press.

Fregda, K., L. Davring, M. Sunner, B.O. Bengtsson, C.G. Elinder, P. Sigtryggsson, and M. Berlin. 1982. Chromosome changes in workers (smokers and nonsmokers) exposed to automobile fuels and exhaust gases. Scand. J. Environ. Health 8:209-221.

Halder, C.A, T.M. Warne, and N.S. Hatoum. 1984. Renal toxicity of gasoline and related petroleum naphthas in male rats. In: Advances in Modern Environmental Toxicology, Volume VII. Renal Effects of Petroleum Hydrocarbons, p. 73-88. Edited by M.A. Mehlman, C.P. Hemstreet, J.J. Thorpe, and N.K. Weaver. Princeton: Princeton Scientific Publishers, Inc.

Halder, C.A., G.S. Van Gorp, N.S. Hatoum, and T.M. Warne. 1986. Gasoline vapor exposures: Part II. Evaluation of the nephrotoxicity of the major C4/C5 hydrocarbon components. Am. Ind. Hyg. Assoc. J. 47:173-175.

Harrington, J.M. 1987. Health experience of workers in the petroleum manufacturing and distribution industry: a review of the literature. Am. J. Indust. Med. 12:475-497.

HEI. 1985. Gasoline Vapor Exposure and Human Cancer: Evaluation of Existing Scientific Information and Recommendations for Future Research. Cambridge, MA, September 1985.

HEI. 1988. Supplement to: Gasoline Vapor Exposure and Human Cancer: Evaluation of Existing Scientific Information and Recommendations for Future Research. Cambridge, MA, January 1988.

Howe, G.R., J.D. Burch, A.B. Miller, G.M. Cook, J. Esteve, B. Morrison, P. Gordon, L.W. Chambers, G. Fodor, and G.M. Wilson. 1980. Tobacco use, occupation, coffee, various nutrients, and bladder cancer. J. Natl. Cancer Inst. 64:701-713.

IIT Research Institute. 1985a. Thirteen-week inhalation toxicity study of a 0 to 145°F gasoline distillate fraction in rats. Sponsored by the American Petroleum Institute. Chicago, IL: IIT Research Institute.

IIT Research Institute. 1985b. Thirteen-week inhalation toxicity study of C4/C5 hydrocarbon blends in rats. Sponsored by

the American Petroleum Institute. Chicago, IL: IIT Research Institute.

IRDC. 1982. Motor Fuel Chronic Inhalation Study. Six Volumes. Mattawan, MI: Sponsored by the American Petroleum Institute.

Kanerva, R.L., M.S. McCracken, C.L. Alden, and L.C. Stone. 1987a. Morphogenesis of decalin-induced renal alterations in the male rat. Fd. Chem. Toxic. 25:53-61.

Kanerva, R.L., G.M. Ridder, F,R, Lefever, and C.L. Alden. 1987b. Comparison of short-term renal effects due to oral administration of decalin or d-limonene in young adult male Fischer 344 rats. Fd. Chem. Toxicol. 25:345-353.

Litton Bionetics, Inc. 1977. Mutagenicity Evaluation of Unleaded Gasoline. Final Report. Submitted to the American Petroleum Institute, Washington, D.C.

Loury, D.J., T. Oliver-Smith, S. Strom, R. Jirtle, G. Michalopoulos, and B.E. Bitterworth. 1986. Assessment of unscheduled and replicative DNA synthesis in hepatocytes treated *in vivo* and *in vitro* with unleaded gasoline or 2,2,4-trimethylpentane. Toxicol. and App. Pharm. 85:11-23.

Loury, D.J., T. Oliver-Smith, and B.E. Butterworth. 1987. Assessment of unscheduled and replicative DNA synthesis in rat kidney cells exposed *in vitro* or *in vivo* to unleaded gasoline. Tox. and App. Pharm. 87:127-140.

MacFarland, H.N. 1983. Xenobiotic-induced kidney lesions: Hydrocarbon--the PS-6 90-day and 2-year gasoline studies. G.P. Hemstreet III, J.J. Thorpe, and M.L. Kane eds. Proceedings of the Workshop on the Kidney Effects of Hydrocarbons, Boston, MA, July 18-20. American Petroleum Institute, pp. 68-74.

MacFarland, H.N., C.E. Ulrich, C.E. Holdsworth, D.N. Kitchen, W.H. Walliwell, and S.C. Blum. 1984. A chronic inhalation study with unleaded gasoline vapor. J. Am. Coll. Toxicol. 3:231-248.

MacHaughton, M.G. and D.E. Uddin. 1983. Toxicology of mixed distillates and high-energy synthetic fuels. G.P. Hemstreet III, J.J. Thorpe, and M.L. Kane eds. Proceedings of the Workshop on the Kidney Effects of Hydrocarbons, Boston, MA, July 18-20. American Petroleum Institute, pp. 171-185.

McDermott, H.J. and S.E. Killiany. 1978. Quest for gasoline TLV. Amer. Indus. Hyg. Assoc. J. 39:110-117.

McLaughlin, J.K., J.S. Mandel, W.J. Blot, L.M. Schuman, E.S. Mehl, and J.F. Fraumeni. 1984. A population-based case-control study of renal cell carcinoma. JNCT 72:275-284.

McLaughlin, J.K., W.J. Blot, J.S. Mandel, E.S. Mehl, P.A. Stewart, F.S. Venable, and J.F. Fraumeni. 1985. Petroleum-related employment and renal cell cancer. J. Occup. Med. 27:9, 672-674.

MSDS. 1985. Material Safety Data Sheet: Gasoline/Automotive. Occupational Health Services, Inc., September 1985.

MSDS. 1987. Material Safety Data Sheet: Gasoline; Evaporated Exhaust Emissions Test Fuel. Howell Hydrocarbons, Inc., April 1987.

MSDS. 1989. Material Safety Data Sheet: Unleaded Premium Gasoline. Sun Oil Company. June 1989.

MVMA. 1991. Letter to William K. Reilly of EPA from Thomas J. Carr of MVMA. September 1991.

NESCAUM. 1989. Evaluation of the Health Effects From Exposure to Gasoline and Gasoline Vapors. Final Report. August 1989.

Phillips Petroleum Company. 1984. Toxicity study summary for unleaded gasoline. Submitted to the American Petroleum Institute in November 12, 1984.

Richardson, K. A., J.L. Wilmer, D. Smith-Simpson, and T.R. Skopek. 1986. Assessment of the genotoxic potential of unleaded gasoline and 2,2,4-trimethylpentane in human lymphoblasts *in vitro*. Toxicol. and Appl. Pharm. 82:315-322.

Runion, H.E. 1975. Benzene in Gasoline. Am. Ind. Hyg. Assoc. J. 36:338-350.

Schwartz, E. 1987. Proportionate mortality ratio analysis of automobile mechanics and gasoline service station workers in New Hampshire. New Hampshire Department of Health and Human Services, Division of Health Services. Amer. J. Indus. Med. 12:91-99.

Stemhagen, A., J. Slade, R. Altman, and J. Bill. 1983. Occupational risk factors and liver cancer. A retrospective case-control study of primary liver cancer in New Jersey. Am. J. Epidemiol. 177:443-454.

Stonard, M.D., P.G.N. Phillips, J.R. Foster, M.G. Simpson, and E.A. Lock. 1986. Alpha$_{2u}$-globulin: measurement in rat kidney following administration of 2,2,4-trimethylpentane. Toxicol. 41:161-168.

Tegeris Laboratories, Inc. 1985. Four-week oral nephrotoxicity screening study in male F334 rats. Temple Hills, MD. Sponsored by the American Petroleum Institute.

UAREP. 1983. Hydrocarbon toxicity: Acute, subchronic, and chronic effects in relation to unleaded gasoline exposure of rodents, with comments on the significance to human health. Prepared for the American Petroleum Institute.

12.0 EPA'S INTEGRATED AIR CANCER PROJECT

12.1 Background

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.

The Raleigh and Albuquerque efforts were essentially done to develop methodologies and validate them in the field. The Boise program was a much more extensive effort in a simple airshed with two main pollution sources -- residential wood combustion and motor vehicle emissions. The Roanoke program involves a more complex airshed, with pollution from residential oil combustion, woodsmoke, and motor vehicle emissions. The IACP program will conclude with the analysis of the Roanoke study. Although IACP studies have focused on air toxics from residential wood combustion, distillate oil combustion appliances, and motor vehicle emissions, this summary only includes information relevant to the subject of air toxics from motor vehicle emissions.

12.2 Methodology for Mutagenicity Apportionment

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. Typically, EOM accounts for about 60% of the fine particles collected at both the residential and mobile source sites (Highsmith et al., 1988). Some detailed characterization of the chemical content of collected samples was done using procedures such as gas chromatography, matrix isolation, and Fourier transform infrared spectrometry. 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.

The IACP has primarily used variations on a test known as the *Salmonella* mutagenicity assay to determine the mutagenicity of organic compounds (EPA, 1989). These findings have been validated by animal bioassays and human epidemiological data. The assay involves exposing colonies of the bacterium *Salmonella typhimurium* to single compounds, complex mixtures, or fractions of complex mixtures. This assay, unlike whole animal assays, can be interlaced with analytical chemistry methods to identify the major classes or specific compounds responsible for the mutagenicity. The *Salmonella* mutagenicity assay uses strains of *S. typhimurium* having specific genetic mutations which make them more sensitive to mutagenesis than "normal" *S. typhimurium*. The IACP studies used a strain designated TA98, with the addition of a rat liver homogenate, designated +S9. When exposed to a mutagenic compound or a mutagenic fraction from an air sample, cells from the TA98 strain can undergo another mutation which causes them to revert back to "normal." Thus, mutagenicity of an air sample is measured in terms of the number of revertants per cubic meter (rev/m^3). The potency of EOM collected from that sample can be expressed in terms of revertants per microgram EOM (rev/μg EOM).

To determine source apportionment of mutagenicity, the IACP has used what is referred to as the receptor-model approach (EPA, 1989). This approach is most effective where the number of sources are small and well-characterized and involves using an element or compound as a tracer to identify a given source (Lewtas and Cupitt, 1987). For instance, the element lead (Pb) has traditionally been used as a tracer for motor vehicle sources of emissions and is the primary tracer used in this study. However, since it is being phased out as a component of gasoline, other tracers have been suggested. Among these are the organic compounds xylene, methylhexane, methylcyclohexane, methylpentane and trimethylpentane (Zweidinger et al., 1990). Potassium (K) and Carbon-14 (^{14}C) are often used as tracers for residential wood burning.

In summary, the IACP approach involves the following series of steps for EOM:
1) Ambient air samples are collected on filters and the organic material is extracted. Detailed chemical characterization is done using gas chromatography and other techniques.
2) Mutagenicity is determined using the *Salmonella* mutagenicity assay.
3) Mutagenicity is 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.

12.3 Apportionment of Mutagenicity from Field Measurement Programs

To date, the IACP has completed two field measurement programs. The first involved sampling in two locations -- Raleigh, North Carolina and Albuquerque, New Mexico, while the second involved sampling in Boise, Idaho.

The first field measurement program, conducted in Winter, 1985, was designed primarily to assess proposed sampling and analytical methods, and to provide a field test of the methods available. The Raleigh sampling effort focused on assessing proposed sampling and analytical methods, while the Albuquerque effort focused on providing a field test of available methods in a simple airshed.

The Boise, Idaho field program was a much more extensive program than the sampling programs in Raleigh or Albuquerque. Boise was selected for an extensive sampling effort because of the predominance of residential wood combustion and motor vehicle emissions as pollution sources in the winter and because of the ready availability of good sampling sites. Because there is little industry in Boise, and because of meteorological conditions that result in retention of local emissions, Boise is a relatively simple airshed to study.

12.3.1 Raleigh, North Carolina

Samples were collected for 12 hour periods beginning at 7:00 A.M. and 7:00 P.M., at two ambient monitoring sites, one in a residential area and one in a rural area. The residential monitoring site was impacted by woodsmoke, while the rural site was chosen to evaluate regional source contributions to the airshed. Source sampling was conducted at three residences with wood stoves, to determine if mutagenicity of organics in the ambient air was higher than the wood stove source samples (Stevens, et al., 1990; Highsmith, et al., 1987). The field study was conducted from January through March 1985.

As previously mentioned, source apportionment of samples collected at ambient monitoring sites was done using the receptor-modeling approach. Receptor-modeling assumes that the ambient concentration of a pollutant can be represented as a sum of terms, with each term representing the contribution of a particular source category (EPA, 1989). These terms are the product of a chemical species tracer (e.g., Pb for motor vehicle emissions) and some coefficient determined by regression analysis of data obtained from ambient sampling. Regression analysis determines how the value of one variable affects another variable. Thus, this analysis could be used to determine what

pollutant concentration could be expected for a given concentration of a specific tracer.

This receptor-modeling approach was used to apportion EOM to sources. It was found that almost all EOM was from woodsmoke or motor vehicles (>99%). This verified that Raleigh was indeed a simple airshed with two major pollution sources. For both monitoring sites combined, approximately 93% of EOM was from woodsmoke and approximately 7% was from motor vehicles (Stevens, et al., 1990).

Mutagenicities and potencies of woodsmoke and motor vehicle emissions from ambient samples were determined using the results of the regression analysis (Stevens, et al., 1990). Results indicated that almost all mutagenic activity for samples could be attributed to wood burning and motor vehicles. Although only about 7% of EOM was from motor vehicles, it accounted for over 20% of the total mutagenicity. This was because the mutagenic potency for motor vehicle emissions was much higher than for woodsmoke (3.7 ± 1.5 rev/µg EOM for motor vehicle emissions, as determined from the Albuquerque work discussed later, versus 0.8 ± 0.1 rev/µg EOM for woodsmoke).

In addition, the mutagenic potency of organics in the ambient air (1.1-1.9 rev/µg EOM) was significantly higher than the mutagenicity of wood stove source samples (0.2-0.6 rev/µg EOM). This difference is likely due to the contribution of motor vehicle emissions to ambient air samples and the effects of atmospheric transformation.

^{14}C content was measured in a small number of samples to confirm the results obtained using K as a tracer for woodsmoke (Stevens et al., 1990). The ^{14}C isotope of carbon is found in emissions resulting from wood burning, in a characteristic ratio to the more common isotope, ^{12}C, but is absent from emissions resulting from combustion of fossil fuels. The ^{14}C approach provides a direct estimate of the fraction of carbon in an ambient air sample resulting from woodsmoke. Estimates of source apportionment using this approach corresponded well to estimates obtained using the receptor-modeling approach with K as a tracer for woodsmoke emissions and Pb as a tracer for motor vehicle emissions.

Analysis of source samples from three residences in Raleigh with wood stoves was also done to compare indoor and outdoor samples (EPA, 1989). It was found that, although the distribution of volatile organic compounds (VOCs) was similar both indoors and outdoors, the mass of fine particles was lower indoors than outdoors. In contrast, aldehyde concentrations were higher indoors than outdoors, because aldehyde concentrations were probably affected more by building materials, furnishings, and homeowner activities more than woodsmoke or motor vehicle emissions.

12.3.2 Albuquerque, New Mexico

As with the Raleigh sampling effort, samples were collected for 12 hour periods beginning at 7:00 A.M. and 7:00 P.M. Samples were collected at two ambient monitoring sites, one a residential site impacted by woodsmoke and the other a roadway site. The field study was conducted from December, 1984 to February, 1985.

In Albuquerque, as in Raleigh, most EOM was from woodsmoke and motor vehicles (94%) (Stevens et al., 1990, Lewis et al., 1988). However, in Albuquerque, motor vehicles contributed much more to the total EOM than in Raleigh (17% in Albuquerque versus 7% in Raleigh). Moreover, motor vehicles accounted for about 36% of the total mutagenicity. The measured mutagenic potency for motor vehicle emissions was 3.7 ± 1.5 rev/µg, which was also used in the Raleigh work. The identical motor vehicle potencies result from the same coefficient being used to express the relationship of Pb and motor vehicle emissions in both cities. In Raleigh, motor vehicle emissions made up such a small percentage of the total that the coefficient for that site could not accurately be determined as it was for Albuquerque. The potency of woodsmoke emissions in Albuquerque was 1.3 ± 0.3 rev/µg, comparable to the value of 0.8 ± 0.1 rev/µg for Raleigh. Once again, results using the ^{14}C approach to determine amount of emissions resulting from woodsmoke supported results using K as a tracer.

12.3.3 Boise, Idaho

The sampling effort in Boise took place in two phases (Cupitt and Fitz-Simons, 1988; Highsmith et al., 1988, 1992). The first phase occurred during August and September 1986 when ambient samples were collected for 12 hour periods beginning at 7:00 A.M. and 7:00 P.M. These samples were collected at two sites, one in a residential area impacted by wood combustion emissions and the other a background location not impacted by any local sources. The second phase was conducted during the November 1986 through February 1987 winter heating season, when extensive wood combustion takes place. Ambient samples in this phase were collected for 12 hour periods beginning at 7:00 A.M. and 7:00 P.M. at 7 sites: three primary sites and four auxiliary sites. The primary sites were a woodsmoke site, a roadway site, and a third site in a background location not impacted by any local sources. A residential monitoring program similar to the one in Raleigh was conducted in conjunction with the winter effort.

The first phase of the Boise study was designed to test existing IACP sampling, analysis, and data management procedures, and to train personnel. No source apportionment analyses were done on data collected in this phase.

Data collected from the second phase of the Boise study indicated that over 90% of EOM could be attributed to either woodsmoke or motor vehicles (Lewis et al., 1991). Although about 27% of ambient EOM was estimated to be from motor vehicles, based on tracer studies, it accounted for 56% of total mutagenicity.

The mutagenic potency for motor vehicle emissions was 3.0 ± 1.1 rev/µg and 0.84 ± 0.25 rev/µg for woodsmoke. It should be noted, however, that recent research (Kleindienst et al., 1991) indicates that mutagenicity of the gas phase is influenced by HC/NO_x ratio. In the Kleindienst et al. study, plates of *S. typhimurium* bacteria were exposed to a mixture of VOCs (similar to that which may be found in Boise) under controlled conditions, with a VOC/NO_x ratio of either 20 or 11. On average, observed mutagenic activity at a VOC/NO_x ratio of 11 was 24 rev/h, while at a ratio of 20 it was 58 rev/h. This work also states that the gas phase reaction products are far more mutagenic than the particle phase. As was the case with the Albuquerque analyses, results using the ^{14}C approach to determine the amount of emissions resulting from woodsmoke supported results using K as the tracer.

Across sampling periods, over 60% of the mutagenicity from ambient air samples could typically be attributable to EOM. Most of the remainder could be attributed to SVOCs, with a very small fraction attributable to VOCs (EPA, 1989). The contribution of SVOCs to total mutagenicity was greater indoors than outdoors. However, the significance of the SVOC contribution to mutagenicity is unknown, since the carcinogenicity of SVOCs has not been studied.

12.4 Other IACP Studies

12.4.1 Human Cancer Risk Estimates

The Boise effort, unlike the Raleigh and Albuquerque efforts, involved animal tumorigenicity studies of the collected ambient samples. This study was the first to quantitatively estimate tumor potency and human cancer risk from EOM in an urban airshed (Lewtas et al., 1991, 1992).

Skin tumorigenesis studies were conducted in female mice. An initiator was applied over a 1 to 4 day period to the skin. The initiator was either a woodsmoke dominated sample (89% woodsmoke, 11% motor vehicle emissions) or a motor vehicle emissions dominated sample (64% motor vehicle emissions, 36% woodsmoke) applied in several dose levels on 1, 2, or 4 successive days. A positive control group was given a single dose of benzo(a)pyrene (BaP), a polycyclic aromatic hydrocarbon (PAH) known to cause lung tumors in laboratory animals. A negative control group was given a single dose of acetone (the solvent for the samples and BaP). After application of the initiator and a one week rest period, a skin tumor promotor was applied twice a week for 26 weeks. After 26 weeks the woodsmoke dominated sample was found to have a tumor initiation potency of 0.0954 skin tumors/mouse/mg (maximum likelihood estimate) and the motor vehicle emissions dominated sample was found to have a tumor initiation potency of 0.215 skin tumors/mouse/mg. Tumor initiation potencies for the positive and negative controls were not given in Lewtas et al. (1991).

These tumor initiation potencies were converted into cancer unit risks using the comparative potency method. The tumor initiation potencies were compared to potencies of human carcinogens for which unit risks exist to determine relative potencies. These relative potencies were then multiplied by the known unit risks for the human carcinogens to get unit risk estimates for the woodsmoke and motor vehicle emissions dominated samples. The other known carcinogens used in the study were emissions from coke ovens, roofing tar, and cigarettes. The cancer unit risk estimates obtained using this approach were 0.57 x 10^{-4} lifetime risk/µg EOM/m^3 for woodsmoke dominated samples and 1.28 x 10^{-4} lifetime risk/µg EOM/m^3 for motor vehicle emissions dominated samples. This compares to combustion source EOM unit risks of 2.3 x 10^{-4} lifetime risk/µg EOM/m^3 for diesel vehicles and 1.2 x 10^{-4} lifetime risk/µg EOM/m^3 for a gasoline catalyst-equipped vehicle (Lewtas, 1991).

12.4.2 Human Exposure

In order to determine human exposure, the IACP measured indoor and outdoor exposure levels to various carcinogens, and applied these exposure levels to human activity patterns.

A variety of methods were used to measure levels of organic compounds. As described previously, extractable organic matter was collected from ambient air samples on filters using methylene chloride. VOCs were collected in canisters and identified by gas chromatography, and aldehydes were collected on 2,4-dinitrophenylhydrazine silica cartridges and eluted from the cartridges with acetonitrile. Elements were measured by X-ray fluorescence of fine fraction dichot filters; inorganic ions, acids and bases were determined using annular denuders and ion chromatography (Highsmith et al., 1991).

Human activity patterns were determined by having 43 residents complete daily diaries of their activities, estimating the fraction of time spent in different microenvironments (Glen et al., 1991). Exposure in microenvironments was based on ambient concentration and time spent in the microenvironment. Total exposure, then, was the sum of exposures in different microenvironments.

The exposure analysis indicated that while the ambient concentration of EOM in Boise during winter averaged 15.3 µg/m^3 from woodsmoke and 4.2 µg/m^3 from mobile sources, human exposure concentrations were 9.5 µg/m^3 from woodsmoke and 2.1 µg/m^3 from mobile sources. These winter exposures result in annual estimates of 3.4 µg/m^3 and 1.2 µg/m^3, respectively. Thus, mobile sources account for about 27% of the annual exposure to residential wood combustion and mobile sources (Cupitt et al., 1992).

12.4.3 Atmospheric Transformation

Although wintertime conditions in Boise, Idaho are not considered conducive to photochemical reactions, IACP analyses (Nishioka and Lewtas, 1992) indicate appreciable ambient concentrations of nitrous acid. Nitrous acid is readily oxidized to hydroxyl radicals, which can then initiate atmospheric transformation processes. Also, certain compounds which occur primarily in atmospheric transformation reactions, such as nitro-aromatic and hydroxy-nitro-aromatic species, were found in ambient Boise samples (Nishioka and Lewtas, 1992). Thus, data suggest that atmospheric transformations did occur in Boise, at least on sunny days, even in winter.

Clearly, atmospheric transformation must be considered throughout the year. In fact, smog chamber simulations done as part of the IACP program (Cupitt et al., 1988, Kleindienst et al., 1991) have demonstrated an important role of atmospheric transformation in the formation of mutagens. The Cupitt et al. (1988) smog chamber studies indicate that emissions from wood stoves and motor vehicles can give substantial increases in mutagenic activity of bacterial strains following irradiation. These substantial increases are found primarily in the gas phase rather than the particulate phase of the mixtures. Whereas most mutagenicity is associated with EOM and SVOCs before irradiation, after irradiation in a smog chamber 80-99% of mutagenicity is in the gas phase. The Kleindienst et al. (1991) smog chamber study shows similar results, indicating that surrogate hydrocarbon mixtures representative of urban atmospheres can give substantial increases in mutagenic activity of bacterial strains following irradiation. As previously mentioned, the Kleindienst et al. (1991) study also indicated greater mutagenicity of irradiated VOC mixtures at higher VOC/NO_x ratios.

12.5 Roanoke Field Study

A field study similar in size and duration to the Boise study was conducted in Roanoke, Virginia during the winter of 1988-1989 (EPA, 1989; Highsmith et al., 1991). The objectives and approach of the Roanoke study paralleled those of the Boise study, except that the Roanoke airshed contained emissions from residential distillate oil combustion appliances. Once again, ambient samples were collected for 12 hour periods, beginning at 7:00 A.M. and 7:00 P.M. at three primary and four auxiliary sites. A residential monitoring program was also conducted.

Analyses of samples collected in the Roanoke study are still being performed. Some preliminary analyses indicate that total NMHC at the primary mobile source site in Roanoke was 1.3 to 1.8 times the level measured at the primary residential site. This is similar to results from Boise. Furthermore, total carbonyls at the Roanoke primary mobile source site were 1.4 times higher than those at the primary residential site.

12.6 Implications

EPA's Updated Six-Month Study (EPA, 1990) indicated that roughly 50% of cancer exposure from airborne toxics may be from mobile sources. Results of the IACP study support this conclusion, with mobile sources accounting for 20% of the mutagenicity of EOM from ambient samples in Raleigh, 36% in Albuquerque, and 56% in Boise. In larger cities, where mobile sources would be expected to contribute more of the ambient EOM, this contribution to mutagenicity would be even higher. 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. Moreover, human exposure estimates from the Boise study indicate that mobile sources account for about 27% of the annual EOM exposure. Finally, atmospheric transformation may greatly exacerbate the risk from mobile sources, since the contributions of VOCs to mutagenicity of ambient samples increases dramatically following irradiation. Thus, efforts to control cancer incidence from airborne toxics must include mobile sources.

12.7 References for Chapter 12

Cupitt, L. and T. Fitz-Simons. 1988. The Integrated Air Cancer Project: Overview and Boise survey results. Published in: Proceedings of the 1988 EPA/APCA International Symposium on Measurement of Toxic and Related Air Pollutants. APCA Publication VIP-10; EPA Report No. 600/9-88-015; pp. 799-803.

Cupitt, L., L. D. Claxton, T. E. Kleindienst, D. F. Smith and P. B. Shepson. 1988. Transformation of Boise sources: The production and distribution of mutagenic compounds in wood smoke and auto exhaust. Published in: Proceedings of the 1988 EPA/APCA International Symposium on Measurement of Toxic and Related Air Pollutants. APCA Publication VIP-10; EPA Report No. 600/9-88-015; pp. 885-889.

Cupitt, L., G. Glenn and J. Lewtas. 1992. Exposure and risk from ambient particle bound pollution in an airshed dominated by residential wood combustion and mobile sources. Risk Analysis (submitted).

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. 1989. Integrated Air Cancer Project: Status Report. Research Triangle Park, North Carolina: Office of Research and Development. February 1989.

Glen, W. G., V. R. Highsmith and L. Cupitt. Development of an exposure model for application to wintertime Boise. Published in: Proceedings of the 1991 AWMA Annual Meeting, paper 91-131.7.

Highsmith, V. R., R. B. Zweidinger, C. W. Lewis, R. K. Stevens, L. D. Claxton and J. Wu. 1992. Characterization of the wintertime Boise, Idaho airshed. Atmospheric Environment (submitted).

Highsmith, V. R., A. J. Hoffman, R. B. Zweidinger, L. T. Cupitt and D. B. Walsh. 1991. The IACP: Overview of the Boise, Idaho, and the Roanoke, Virginia, field studies. Published in: Proceedings of the 1991 AWMA Annual Meeting, paper 91-131.1.

Highsmith, V. R., R. Zweidinger, J. Lewtas, A. Wisbith and R. Hardy. 1988. Published in: Proceedings of the 1988 EPA/APCA International Symposium on Measurement of Toxic and Related Air Pollutants. Impact of residential wood combustion and automotive emissions on the Boise, ID airshed. APCA Publication VIP-10; EPA/600/9-88-015; pp. 841-852.

Highsmith, V. R., R. E. Baumgardner, B. Harris, T. Lumkin, R. Grago, R. Zweidinger and R. Merrill. 1987. The collection of neighborhood air samples impacted by residential wood combustion in Raleigh, NC and Albuquerque, NM. Published in: Proceedings of the 1987 EPA/APCA Symposium on Measurement of Toxic and Related Air Pollutants, May 3-6, 1987. APCA Publication VIP-8; EPA Report No. 600/9-87-010; pp. 562-572.

Kleindienst, T. E., D. F. Smith, E. E. Hudgens, C. D. McIver, E. Perry, L. T. Cupitt, J. J. Bufalini and L. D. Claxton. 1991. The atmospheric transformation of combustion source emissions and the formation of mutagenic products. Published in: Proceedings of the 1991 AWMA Annual Meeting, paper 91-131.5.

Lewis, C. W., R. K. Stevens, R. B. Zweidinger, L. D. Claxton, D. Barraclough and G. A. Klouda. 1991. Source apportionment of mutagenic activity of fine particle organics in Boise, ID. Published in: Proceedings of the 1991 AWMA Annual Meeting, paper 91-131.3.

Lewis, C., R. Baumgardner, R. Stevens, L. Claxton and J. Lewtas. 1988. The contribution of wood smoke and motor vehicle emissions to ambient aerosol mutagenicity. Environ. Sci. Technol., 22:968-971.

Lewtas, J. 1991. Carcinogenic risks of polycyclic organic matter (POM) from selected emission sources: Development of a comparative potency method. U.S. Environmental Protection Agency, Health Effects Research Laboratory, Research Triangle Park, NC. Deliverable Report No. 3128.

Lewtas, J., M. Moore, C. T. Helms, and S. Nesnow. 1992. Comparative tumor-initiating activity of urban aerosol and source emissions from wood smoke and motor vehicles. Carcinogenesis (submitted).

Lewtas, J. R. B. Zweidinger and L. Cupitt. 1991. Mutagenicity, tumorigenicity and estimation of cancer risk from ambient aerosol and source emissions from woodsmoke and motor vehicles. Proceedings of the 1991 AWMA Annual Meeting, paper 91-131.6.

Lewtas, J. and L. Cupitt. 1987. Overview of the Integrated Air Cancer Project. Published in: Proceedings of the 1987 EPA/APCA Symposium on Measurement of Toxic and Related Air Pollutants, May 3-6, 1987. APCA Publication VIP-8; EPA Report No. 600/9-87-010; pp. 555-561.

Nishioka, M. G. and J. Lewtas. 1992. Quantification of nitro- and hydroxylated nitro-aromatic/polycyclic aromatic hydrocarbons in selected ambient air daytime winter samples. Atmospheric Environment (in press).

Stevens, R. K., C. W. Lewis, T. G. Dzubay, L. T. Cupitt and J. Lewtas. 1990. Sources of mutagenic activity in urban fine particles. Toxicol. and Indust. Health 6: 81-94.

Zweidinger, R., R. Stevens, C. Lewis and H. Westburg. 1990. Identification of volatile hydrocarbons as mobile source tracers of fine-particulate organics. Environ. Sci. Technol. 24: 538-542.

13.0   TOXICS ASPECTS OF ALTERNATIVE FUELS

The CAAA of 1990 provide for a centrally fueled clean fuel fleet program, covering fleets of 10 or more vehicles capable of central refueling. Under this program, beginning in 1998, 30 percent of new light duty vehicles and trucks purchased by centrally fueled fleets in certain ozone and CO nonattainment areas will be required to use clean fuels. There will be a separate requirement for heavy duty trucks. Rental vehicles, police vehicles, emergency vehicles, and nonroad vehicles are exempt from this requirement.

Clean fuels are defined simply by exhaust emission performance standards and result in tailpipe emissions substantially lower than those in place for general passenger cars. Vehicles could meet requirements of the program through the use of various alternative fuels and also reformulated gasolines. It is likely, however, that reformulated gasolines will capture most of the market. Initiation of the program may be delayed until 2001 if cars meeting these stringent tailpipe standards when running on clean fuels are not already being produced in California. Beginning with the model year 2001, the 50,000 mile emission certification standards for light duty clean fuel vehicles are 0.075 g/mi NMOG, 3.4 g/mi CO and 0.2 g/mi NO_x. If the program starts in 1998, the purchase requirement for clean fuel light duty vehicles and trucks will be 70 percent of the new vehicles and trucks purchased for the applicable fleet in the year 2000 and later.

The new California motor vehicle standards are intended to encourage the consideration of alternative fuels. Nevertheless, as previously mentioned, it is likely that most vehicles meeting California LEV standards will run on reformulated gasoline. It should be noted, however, that the ZEV portion of the California standards in effect require the development of electric vehicles, since electric vehicles are the only vehicles which could feasibly meet the California requirement for ZEVs at this time. The California standards are discussed in detail in Section 3.1.3.1.

Also, the Comprehensive National Energy Policy Act of 1992 requires non-gasoline alternative fueled vehicles in government and certain other fleets in the petroleum and alternative fuels industry beginning with 30 percent of new fleet purchases in 1996 and phasing up to 90 percent in 1999. The Act also requires alternative fueled vehicles in state fleets starting at 10% in 1996 and phasing up to 75% in the year 2000. In addition, the Department of Energy may extend the program to other fleets if it determines such a program is necessary.

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), or compressed natural gas (CNG), with a small number of electric vehicles produced to meet California's 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 fuel differences. This chapter gives a brief overview of toxics aspects from methanol, ethanol, and CNG fuel use. A brief discussion of liquid propane gas (LPG) as a motor vehicle fuel follows.

13.1 Methanol

A promising alternative fuel for motor vehicles appears to be methanol. Although methanol has about one-half the energy per gallon as gasoline, it is a more energy efficient fuel than gasoline. Methanol is liquid, high octane, and has a reasonable range, which makes it easily adaptable. Because of its lower volatility, methanol use would result in a reduction of collision related vehicle fires (Machiele, 1990). Moreover, combustion of methanol produces a slight increase in engine power. Methanol can be used in a combustion system which operates lean much of the time. In addition, methanol can be produced easily and economically from natural gas and a range of other feedstocks (EPA, 1989). This is done today all over the world. However, non-dedicated engines burning M100 are difficult to start at low temperatures. Also, combustion of M100 does not produce a visible flame, causing a potential safety hazard in those cases where only fuel is burning. Another potential safety hazard is the possibility of fuel tank explosions with methanol. This is not expected to be a frequent occurrence, but when it does occur, the explosion is typically minor and frequently contained by the fuel tank. Also, simple design modifications can drastically reduce this possibility. Moreover, methanol is odorless, colorless and tasteless, which combined with a high toxicity, means steps must be taken to assure it is not ingested (accidentally or otherwise) by humans. Denaturant type additives are needed to prevent ingestion. Because of these concerns, small quantities of gasoline are often added to methanol. One commonly used gasoline/methanol blend is M85, with 15% gasoline and 85% methanol. Nonetheless, preliminary data obtained in tests with both M85 and M100 suggest a much greater potential for reduction in the emission of ozone precursors exists with M100 usage (Gabele, 1990). Thus, it is desirable to solve problems related to usage of M100 fuel.

13.1.1   Health Effects of Toxic Emissions from Methanol Use

The two toxic emissions resulting from methanol use which are of the most concern are methanol itself and formaldehyde. The health effects of formaldehyde were discussed in Chapter 6. The Office of Toxic Substances is revising the formaldehyde

cancer risk assessment, which will assist in estimating impacts of ambient lifetime exposures.

A large percentage of total exhaust and evaporative organic emissions from motor vehicles running on either M100 or methanol blends is methanol itself (Gabele, 1990; California Air Resources Board, 1989, 1991; Auto/Oil, 1992a,b). The health effects of M85 evaporative and combustion emissions will be some combination of those associated with both methanol and gasoline. Because the noncancer health effects of neither emissions from unleaded gasoline nor methanol are adequately understood, more research on M85 is needed.

Also, the release of methanol in the atmosphere can potentially result in the increased formation of dimethyl sulfate under certain conditions. Dimethyl sulfate is a carcinogen, is acutely toxic to the nervous system, and is highly irritating to the eyes, respiratory system, and skin. The compound has been observed in ambient air downwind of power plants, presumably as a product of the reaction of hydrocarbons with sulfur dioxide on particles. Research is needed to determine whether dimethyl sulfate is also formed to any significant degree with methanol as the hydrocarbon and, if so, to assess its potential for increasing human cancer incidence, neurotoxicity, and/or respiratory effects in areas using methanol fuels.

13.1.1.1 Effects of Chronic and Acute Exposures -- Humans

To date, little research has been done dealing with chronic effects of long term low level inhalation exposure. Chronic exposure to low levels (less than 200 ppm) of methanol has not been observed to cause serious adverse health effects in the workplace (ACGIH, 1986). As a consequence it may be that methanol has no toxic effect at the anticipated low levels of exposure from automotive methanol vapor (Kavet and Nauss, 1990; Health Effects Institute, 1987). There are currently reports of possible health effects that require further consideration. These are discussed in more detail below. The Health Effects Institute report (HEI, 1987) summarized the health effects of methanol and concluded that methanol exposure from motor vehicles would not be significant compared to methanol exposure from the diet (e.g., from a number of fruits, vegetables, diet beverages). Additional research to verify this conclusion is currently underway.

Chronic or acute exposure to methanol vapor at low levels, however, can cause symptoms such as eye irritation, headache, dizziness, nausea, and blurred vision. In humans, methanol is absorbed following oral and inhalation exposure (HEI, 1987). Once ingested or inhaled, methanol is slowly eliminated from the body, hence chronic or repeated exposures result in increased concentrations in blood and tissues (Henderson and Haggard, 1943). A primary issue is microenvironmental exposure to methanol, which would increase if methanol were used as an alternative fuel. A recent pilot clinical study sponsored by the

Health Effects Institute (Cook et al., 1991) suggests there may be slight impairment of memory and concentration due to chronic exposure at low levels (192 ppm). These results are preliminary and need to be confirmed by more definitive studies.

Oral ingestion of methanol is toxic in doses as small as 18 ml or more (Machiele, 1990). NIOSH (1976) suggests that the critical effects most associated with relatively high acute methanol exposure are visual disturbances and metabolic acidosis. Acute exposure, via oral and inhalation routes, has frequently caused death or blindness (McNally, 1937; Jacobson et al., 1945). Following ingestion of methanol by 23 men in Korea, six men died, while the others experienced nausea, epigastric pain, vomiting, headache, dizziness, delirium, varying degrees of transitory blindness, acidosis, and acetonuria (Keeney and Mellinkoff, 1951). Similar effects have also been reported after occupational exposure to high levels (4,000 to 13,000 ppm[1] for 12 hours) of methanol vapor (Browning, 1965). Acute occupational exposure to 800 to 1,000 ppm methanol has been associated with frequent headaches and blurred vision (McNally 1937). Henson (1960) noted partial vision loss in one worker exposed to 1,200-8,000 ppm methanol for four years.

Formate, a toxic metabolite of methanol which is associated with acute methanol toxicity in humans, does not appear to accumulate in blood when methanol exposure concentrations are below 200 ppm (Lee et al., 1992). The key to methanol toxicity in folate-deficient (folic acid) animals is a diminished capacity to detoxify formate in the liver. This is due to the fact that a metabolite of folate is necessary in the detoxification pathway of formate. This pathway has been demonstrated in both rats and non-human primates. In these cases, the effect of dietary folate (folic acid) on formate metabolism was the direct link to altered sensitivity to methanol; slowing formate metabolism induced a methanol-sensitive state. The role of formate in toxicity resulting from repeated acute or chronic exposures is currently unclear.

13.1.1.2 Effects of Chronic and Acute Exposures -- Animal Studies

In animals, inhalation exposure to methanol caused alterations and degeneration in the ganglionic cells, retina, and choroid of the eye (Scott et al., 1933). Additionally, rats gavaged daily with methanol for 38-90 days experienced elevated levels of serum alkaline phosphatase (SAP) and serum glutamate pyruvic transaminase (SGPT), decreased brain weights, and statistically insignificant increases in liver weights (EPA, 1986). Increases in the levels of the serum enzymes (SAP and SGPT) indicate the possibility of liver damage.

---

[1]Conversion factors for methanol = 1 ppm = 1.31 mg/m^3; 1 mg/m^3 = 0.76 ppm

Inhalation exposure has also been shown to have reproductive effects on male rats. Mature rats were exposed to 260, 2,600, and 13,000 ppm methanol for 8 hours per day for 1 to 6 weeks; decreased circulating free testosterone was noted at the two lower levels (Cameron, et al., 1984). Moreover, another study by Cameron et al. (Cameron et al., 1985) also suggests that exposure to low levels of methanol could alter serum testosterone levels in rats. However, Lee et al., 1991 found that low level methanol exposure may not cause an inhibitory effect on testosterone synthesis. Rats exposed by inhalation to 200 ppm for 6 weeks (8 hr/d, 5d/wk) did not have reduced serum testosterone levels. The testes-to-body ratios of rats exposed to up to 800 ppm methanol for up to 13 weeks (20 hr/d, 7 d/wk) were not different from those of air-exposed rats. Methanol had no adverse effect on testicular morphology at the end of the 13 week exposure period at 800 ppm in either normal rats or folate-reduced methanol-sensitive rats at age 10 months. Even so, a greater incidence of testicular degeneration was noticed in the 18 month old folate-reduced rats exposed to 800 ppm for 13 weeks, suggesting that methanol may have a potential to accelerate the age-related degeneration of the testes.

Recent EPA studies show that inhalation of methanol causes birth defects in rodents. Pregnant mice exposed to 5,000 ppm of methanol, for 7 hours/day on days 6-15 of pregnancy, produced offspring with cleft palates and exencephaly (deformation of the brain where the brain forms outside of the cranium) (Rogers, et al., 1991). These data confirm developmental effects seen in other tests on rats exposed to higher concentrations of methanol (Nelson, et al., 1985).

Two recent EPA studies that are now in press, (Rogers et al., 1992; Andrews et al., 1992) continue to investigate reproductive and developmental toxicity resulting from methanol inhalation. Rogers et al., (1992) exposed pregnant CD-1 mice by inhalation to 1000, 2000, 5000, 7500, 10,000, and 15,000 ppm methanol for 7 hr/day on days 6-15 of gestation. Significant increases in exencephaly and cleft palate were observed at 5000 ppm and above, increased postimplantation mortality at 7500 ppm and above (including an increasing incidence of full-litter resorption), and reduced fetal weight at 10,000 ppm and above. A dose-related increase in skeletal abnormalities were significant at 2000 ppm and above. The No Observed Adverse Effect Level (NOAEL) for developmental toxicity in this study was 1000 ppm. The dose-response data were qualitatively modeled to estimate the added risk of developmental toxicity of inhaled methanol. The lowest maximum likelihood estimate for 5% added risk of developmental toxicity was in the dose range of the NOAEL. The results of this study indicate that inhaled methanol is developmentally toxic in the mouse at exposure levels which were not maternally toxic.

In Andrews et al., rat embryos were explanted and cultured in 0, 2, 4, 8, 12, or 16 mg MeOH/ml rat serum for 24 hours. Embryonic development of the 2 and 4 mg/ml MeOH exposure groups

were not significantly different than that of the control groups. Exposures at the higher concentration resulted in a dose related decrease in the embryos segment number and overall development. The 12 mg/ml exposure resulted in some embryo lethality as well as abnormal formation and differentiation of tissues and organs (dysmorphogenesis), while the 16 mg/ml MeOH exposure level was embryolethal. Methanol was dysmorphogenic *in vitro* to rat embryos at concentrations comparable to serum levels reported to be developmentally toxic after *in vivo* exposure. In the same study, mouse embryos were explanted and cultured in 0, 2, 4, 6, and 8 mg MeOH/ml culture medium. At all exposure levels the crown rump length and developmental score were significantly lower when compared to controls. The high dose group produced 80% lethality. Since the mouse exhibits dysmorphogenesis and embryotoxicity at lower methanol concentrations than the rat, it may be intrinsically more sensitive to methanol exposure. This species difference in sensitivity is consistent with the in vivo exposure results.

Based on these laboratory studies, inhalation of methanol vapors in excess of 1000 ppm can cause birth defects in both rats and mice. Thus, qualitatively, methanol would be called a developmental toxicant using EPA's developmental toxicity guidelines. The implications of these findings for humans, however, must await the results of further pharmacokinetics and health effects research. Pregnant women may be of special concern as a sensitive subpopulation because many have folate deficiency, which may increase susceptibility to birth defects in the developing fetus.

13.1.1.3 Health Based Criteria

An oral reference dose (RfD) of 0.5 mg/kg/day for methanol has been derived by EPA (1992). The value was based on a rat gavage subchronic study by EPA (1986) in which the increases in SAP and SGPT, and decreases in brain weights were evaluated. A no-observed-adverse-effect-level (NOAEL) of 500 mg/kg/day and an uncertainty factor of 1,000 were used to calculate the RfD. An uncertainty factor of 10 was used to account for interspecies extrapolation, 10 for range of sensitivity within the human population to xenobiotics and 10 to account for extrapolation from subchronic to chronic exposure. A reference concentration (RfC) for methanol is unavailable at this time. Methanol has not been evaluated by the EPA for evidence of human carcinogenicity potential.

The ACGIH Threshold Limit Value (TLV-TWA) is 200 ppm (260 mg/m^3) for methanol, with a short-term exposure limit (STEL) of 250 ppm (328 mg/m^3) (ACGIH, 1990). Methanol has a skin designation (ACGIH, 1990). The OSHA Permissible Exposure Limit (PEL) time-weighted average (TWA) and STEL values correspond with ACGIH's TLV-TWA and STEL for methanol, respectively (OSHA, 1989). These values were based on occupational studies in which the NOAEL was 300 ppm (Leaf and Zatman, 1952).

13.1.2   Effects of Methanol Use on Air Toxic Levels

Use of methanol in motor vehicles will result in substantial reductions or elimination of benzene, 1,3-butadiene, acetaldehyde, gasoline refueling vapors, and particulate. However, tailpipe emissions of formaldehyde (i.e. primary formaldehyde) will go up. Conversely, the use of methanol, with its lower hydrocarbon emissions, is likely to result in decreased levels of secondary formaldehyde, which is formed in the ambient air from photochemical oxidation of hydrocarbons. Projected changes in air toxics levels were given in a recent comparison of gasoline and M85 emissions from flexible fuel and variable fuel vehicles (Auto/Oil, 1992a), and toxics reductions for M100 and M85 from optimized FFVs were estimated in EPA's Methanol Special Report (EPA, 1989). Data from these sources are given in Table 13-1.

Table 13-1. Percent Change in Air Toxics Levels for M85 and M100 Relative to Gasoline.

	Auto/Oil Study (1992a)	EPA Methanol Special Report (1989)	
Pollutant/Source	M85	M85	M100
Exhaust Benzene	-84	-77	-97
Evaporative Benzene	-	-67	-100
Running Loss Benzene	-	-69	-100
Refueling Benzene	-	-14	-100
Other Gasoline Refueling Vapors	-	-14	-100
Exhaust 1,3-Butadiene	-93	-64	-99
Exhaust Gasoline POM	-	-72	-99
Primary Formaldehyde	+436	+600	+200
Secondary Formaldehyde	-	-43	-80

In addition, Auto/Oil (1992b) recently released a study comparing toxic emissions from three dedicated methanol vehicles, one running on M85 and two on M100, to toxic emissions from the FFV/VFV fleet in Auto/Oil (1992a). The dedicated methanol vehicle running on M85 was a Chevrolet Lumina designed to target California TLEV emission levels. The formaldehyde emission level for this vehicle was 5.7 g/mi (217% larger than the average formaldehyde emission level from dedicated gasoline vehicles running on baseline fuel). Benzene emissions were 1.0 g/mi (89% lower). The two dedicated methanol vehicles running on M100, a Nissan Sentra and Toyota Corolla, were designed to optimize fuel

economy, not emissions. While the Toyota Corolla's formaldehyde
emission level was 7.5 g/mi (317% higher than for dedicated
gasoline vehicles running on baseline fuel), the Nissan Sentra's
formaldehyde emission level was 27.2 g/mi (well over 1000%
higher). This disparity in emission levels is indicative that
dedicated methanol vehicles are still in early stages of
development, and thus a definitive assessment of toxic emissions
typical of such vehicles is premature. In fact, EPA tested the
same Nissan Sentra vehicle as Auto/Oil with a resistively heated
palladium:cerium catalyst (Hellman and Piotrowski, 1990), and
formaldehyde was measured at 0.20 g/mi.

As mentioned previously, combustion of M100 and methanol
blends produces more primary formaldehyde emissions than
combustion of gasoline. However, most of these formaldehyde
exhaust emissions occur during the cold start portion of
emissions testing (Gabele, 1990), which is reasonable since
formaldehyde is a partial combustion product of methanol. A
dedicated methanol vehicle (i.e., one using only methanol) burns
methanol more efficiently than a flexible fuel vehicle (which can
use various mixtures of gasoline and methanol ranging from 0% to
85%). Thus, it is likely that a dedicated methanol vehicle would
have lower primary formaldehyde emissions than an FFV operated on
M85. Also, as indicated by Hellman and Piotrowski (1990), it is
also likely that dedicated methanol vehicles could have catalysts
more effective in removing formaldehyde than catalysts designed
for gasoline vehicles, as gasoline catalysts must be designed to
reduce a wide variety of HC compounds. Also, placement of the
catalyst can be optimized. The durability of the emission
control systems in methanol fueled vehicles would also affect
formaldehyde levels. Clarification of whether the formaldehyde
fraction of methanol vehicle exhaust will increase with catalyst
deterioration is needed. If formaldehyde emissions rates are
observed to increase beyond acceptable levels, then new more
durable catalyst formulations may need to be developed.

Moreover, a study of formaldehyde emissions from one
variable gasoline/methanol fueled car, running on gasoline, M25,
M50, M85, and neat methanol, indicates that primary formaldehyde
emissions increase progressively for methanol blends, but are
lower for M100 than M85 (Gabele, 1990). In any case, secondary
formaldehyde emissions from combustion of neat methanol should be
lower than for gasoline, due to the relative decrease in reactive
hydrocarbons emitted (EPA, 1989). In fact, when improvement in
methanol engine and emission control technology are considered
along with secondary formaldehyde emissions reductions, EPA
projects no substantial increase in overall formaldehyde
emissions with use of M100 in dedicated vehicles (EPA, 1989).

Any possible increase in cancer incidence due to primary
formaldehyde exposure from methanol fuel combustion would be more
than offset by the dramatic reduction in 1,3-butadiene exposure,
with its much higher unit risk (2.8×10^{-4} for 1,3-butadiene
versus 1.3×10^{-5} for formaldehyde), as well as by reductions in
benzene, acetaldehyde, refueling vapors, and particulate

exposure. Overall, EPA's Methanol Special Report (EPA, 1989) estimates that neat methanol vehicles can be designed to have only 10% of the air toxics emissions of gasoline vehicles, and FFVs can be designed to have about half. Thus, use of neat methanol and methanol/gasoline blends should result in cancer incidence reductions, although reductions with the M85 vehicles are lower than with M100. Possible noncancer risks posed by exposure to methanol, particularly in microenvironments, is currently under evaluation and no definitive statements can be made at this time.

13.2 Ethanol

Low level ethanol mixtures (10% ethanol and 90% gasoline) are already widely used in the U.S., and account for about 6% of total fuel use nationwide (Section 3.1.3.2). Higher level ethanol mixtures (e.g., 85% ethanol, or E85) and neat ethanol may be used as an alternative fuel source in the future for vehicles specifically designed for them. Ethanol has a higher octane than gasoline; thus, vehicles could be designed for improved fuel efficiency. It also has a lower vapor pressure, which would result in lower evaporative emissions. Moreover, it can be produced from renewable resources, such as corn and other biomass. Its flammability is lower than gasoline, which should result in fewer vehicle fires. However, without subsidies, ethanol cannot presently compete with gasoline in price. Also, cold starting is more difficult than with gasoline.

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. According to EPA's Ethanol Special Report (EPA, 1990a), substantial reductions in benzene, 1,3-butadiene, refueling vapors and particulate 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 (Section 8.8). 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).

Although a large percentage of total emissions from motor vehicles running on either neat ethanol or ethanol blends is ethanol itself (California Air Resources Board, 1989, 1991), ethanol is not considered a toxic pollutant at the low levels likely to be inhaled due to its use as a motor fuel.

13.3 Compressed Natural Gas

Worldwide, compressed natural gas (CNG) has been used extensively as a motor vehicle fuel, and is currently used in the U.S. to power a wide range of vehicles and equipment, including some light duty trucks and some urban buses. Generally, most CNG use has involved retrofitting existing engines to run on either gasoline or CNG. However, the greatest benefits from using CNG can be realized with dedicated vehicles, optimized to make use of the specific combustion properties of CNG.

Based on an energy equivalent price comparison in EPA's Compressed Natural Gas Special Report (EPA, 1990b), CNG can be comparable to gasoline in cost. There are several drawbacks to CNG use, however, including decreased driving range, safety hazards due to carrying a fuel under pressure, and the effect of large, heavy storage tanks on vehicle range, performance, and efficiency. Nevertheless, substantial environmental benefits may be achieved from the use of CNG as an alternative fuel, due to the low reactivity of exhaust emissions from CNG fueled vehicles relative to gasoline.

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 (California Air Resources Board, 1989, 1991). Formaldehyde and acetaldehyde exhaust emissions are roughly the same as for gasoline. Thus, the air toxics benefits are greater than those with M100.

Since methane fueled vehicles emit large quantities of methane, the health effects and global climate change of methane emissions should also be considered. Methane is present at low levels in the atmosphere from natural sources (e.g., decaying vegetation). Methane is generally not thought to have adverse health effects at low levels, although high levels can cause asphyxiation. However, methane can contribute significantly to the potential for global climate change. This aspect must be thoroughly evaluated.

13.4 Liquid Propane Gas

Liquid propane gas (LPG) is another possible alternative fuel for motor vehicles. As for CNG, most LPG use has involved retrofitting existing engines to run on either gasoline or CNG. However, the greatest benefits from using LPG would be realized with dedicated vehicles, optimized to make use of the specific combustion properties of the fuel.

LPG would be expected to have very little evaporative emissions. The California Air Resources Board has speciated exhaust emissions from several LPG vehicles (CARB, 1989, 1991).

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.

13.5 References for Chapter 13.0

ACGIH. 1986. Documentation of the threshold limit values. 5th ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

ACGIH. 1990. Threshold limit values and biological exposure indices for 1990-1991. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

Andrews, J.E., M. Ebron-McCoy, L.L. Mole, T.R. Logsdon, R.J. Kavlock, and J.M. Rogers. 1992. Developmental toxicity of methanol in whole embryo culture: a comparative study with mouse and rat embryos. Teratology, in press.

Auto/Oil Air Quality Improvement Research Program. 1992a. Technical Bulletin No. 7: Emissions and Air Quality Modeling Results from Methanol/Gasoline Blends in Prototype Flexible/Variable Fuel Vehicles.

Auto/Oil Air Quality Improvement Research Program. 1992b. Technical Bulletin No. 10: Emissions of Three Dedicated-Methanol Vehicles.

Browning, E. 1965. Toxicity and metabolism of organic solvents. Elsevier, Amsterdam.

California Air Resources Board. 1989. Definition of a Low-Emission Motor Vehicle in Compliance with the Mandates of Health and Safety Code Section 39037.05 (Assembly Bill 234, Leonard, 1987). El Monte, California: Mobile Source Division.

California Air Resources Board. 1991. Proposed Reactivity Adjustment Factors for Transitional Low-Emission Vehicles: Technical Support Document. El Monte, California: Mobile Source Division. Updated Version, November 13, 1991.

Cameron, A.M., Nilsen, O.G., Haug, E., and Eik-Nes, K.B. 1984. Circulating concentrations of testosterone, luteinizing hormone, and follicle stimulating hormone in male rats after inhalation of methanol. Arch. Toxicol., Suppl., 7:441-443.

Cameron, A. M., K. Zahlsen, E. Haug, O. G. Nilsen, and K. B. Eik-Nes. 1985. Circulating steroids in male rats following inhalation of n-alcohols. Arch. Toxicol., Suppl. 8: 422-424.

Cook, M. R., F. J. Bergman, H. D. Cohen, M. M. Gerkovich, C. Graham, R. K. Harris, and L. G. Siemann. 1991. Effects of Methanol Vapor on Human Neurobehavioral Measures. Health Effects Institute Research Report No. 42.

Davis, M. and L. D. Grant. 1991. Health Effects Issues Associated with Methanol Fuels. Presented at: AWMA Specialty Conference -- Toxic Air Pollutants from Mobile Sources: Emissions and Health Effects, Detroit, Michigan, October, 1991.

Environmental Protection Agency. 1986. Rat Oral Subchronic Toxicity study with Methanol. Washington, DC: Environmental Protection Agency, Office of Solid Waste.

Environmental Protection Agency. 1989. 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. 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. 1992. Integrated Risk Information System. U.S. Environmental Protection Agency. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

Gabele, P. A. 1990. Characterization of emissions from a variable gasoline/methanol fueled car. J. Air Waste Manage. Assoc. 40: 296-304.

Health Effects Institute. 1987. Automotive Methanol Vapors and Human Health: An Evaluation of Existing Scientific Information and Issues for Future Research. Report of HEI Health Research Committee, May, 1987.

Hellman, K. H., and G. K. Piotrowski. 1990. Recent results from prototype vehicle and emission control technology evaluation using methanol fuel. SAE Technical Paper 901112.

Henderson Y. and H.W. Haggard. 1943. Noxious Gases. Reinhold Publishing Corp., New York. 218.

Henson, E.V. 1960. J. Occup. Med. 2:497.

Jacobson, B.M., H.K. Russell, J.J. Grimm, and E.C. Fox. 1945. U.S. Naval Med. Bull. 44:1099.

Kavet, R., and K. M. Nauss. 1990. The toxicity of inhaled methanol vapors. Critical Reviews in Toxicology 21: 21-50.

Keeney, A.H. and S.M. Mellinkoff. 1951. Ann. Int. Med. 34: 331.

Leaf, G. and L.J. Zatman. 1952. Brit. J. Ind. Med. 9: 19.

Lee, E.W., T.S. Terzo, J.B. D'Arcy, K.B. Gross, and R.M. Schreck. 1992. Lack of blood formate accumulation in humans following exposure to methanol vapor at the current permissible exposure limit of 200 ppm. Am. Ind. Hyg. Assoc. J. 53:99-104.

Lee, E.W., A.N. Brady, M.J. Brabec, and T. Fabel. 1991. Effects of methanol vapors on testosterone production and testis morphology in rats. Toxicol. Ind. Health 7:261-275.

Machiele, P. 1990. A health and safety assessment of methanol as an alternative fuel. In: Kohl, W. L., ed. Methanol as an Alternative Fuel Choice: An assessment. Washington, D.C.: The Johns Hopkins Foreign Policy Institute.

McNally, W.D. 1937. Toxicology. Industrial Medicine, Chicago, Illinois. p. 615.

Nelson, B.K., Brightwell, S.W., MacKenzie, D.R., Kahn, A., Burg, J.R., Weigel, W.W., and Goad, P.T. 1985. Teratological assessment of methanol and ethanol at high inhalation levels in rats. Fund. Appl. Toxicol. 5: 727-736.

NIOSH. 1976. Criteria for a recommended standard-occupational exposure to methyl alcohol. Washington, DC: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health. DHEW Pub. No. 76-148.

OSHA. 1989. Preamble to OSHA final rule revising workplace air contaminant limits. A BNA special supplement. Washington, DC: U.S. Department of Labor, Occupational Safety and Health Administration. Federal Register 54: 2332-2929.

Rogers, J.M., Chernoff, N., and Mole, M.L. 1991. Developmental toxicity of inhaled methanol in mice. The Toxicologist, Vol. 11, page 344.

Rogers, J.M., M.L. Mole, N. Chernoff, B.D. Barbee, C.I. Turner, T.R. Logsdon, and R.J. Kavlock. 1992. The developmental toxicity of inhaled methanol in the CD-1 mouse, with application of quantitative dose-response modeling for estimation of benchmark doses. Teratology, in press.

Scott, E., M.K. Helz, and C.P. McCord. 1933. Am. J. Clin. Pathol. 3:311.

14.0 NONROAD MOBILE SOURCES

A brief discussion of toxic emissions from nonroad sources was provided in an EPA document, <u>Nonroad Engine and Vehicle Emission Study</u> (NEVES), EPA report number 21A-2001, produced by the Office of Mobile Sources in Ann Arbor, Michigan in November, 1991. For a complete analysis of the emission inventories and methodology used in this study, this document should be consulted. While Section 202(l) of the Act only addresses toxic air pollutants associated with motor vehicles and motor vehicle fuels, EPA included nonroad engines and vehicles in this study for purposes of completeness and to assess the relative impact of onroad and nonroad sources to total mobile source emissions.

The term "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. EPA considered more than 80 different types of equipment in the NEVES. Locomotive and aircraft are not included in the NEVES because the Clean Air Act amendments provide for them separately.

Nonroad engines are not currently regulated for emissions, and very few nonroad engines currently use emission control technology. However, EPA plans to propose regulations setting standards for certain categories of nonroad equipment. Such regulations may include emission standards for diesel engines at or above 50 horsepower and small gasoline engines. 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 major uncontrolled mobile source category.

Because nonroad sources are among the few remaining uncontrolled sources of pollution, their emissions appear large in comparison to the emissions from sources that are already subject to substantial emission control requirements. For example, the Clean Air Act requires extreme ozone nonattainment areas to employ Reasonable Available Control Technology (RACT) on all stationary sources with VOC or NO_x emissions above 10 tons per year (tpy). Annual operation of only 10 crawler tractors or 24 agricultural tractors will produce 10 tpy of NO_x. Typical annual operation with only 74-142 boats with outboard motors or 730-1630 chain saws will emit 10 tpy of VOC. In contrast, it takes 700 new, current-technology passenger cars driving an average of 13,000 miles each year (a total of more than 9 million miles) to produce 10 tpy of VOC.

Nonroad engines were studied for their contribution to CO and the pollutants that contribute to ozone formation, namely volatile organic compounds (VOC) and oxides of nitrogen (NO_x). However, as mentioned above, the NEVES also briefly addressed

pollutants discussed in this report, including particulate matter (PM), benzene, aldehydes, 1,3-butadiene, and gasoline vapor.

Nonroad sources contribute substantially to summertime VOC and NO_x emissions and winter CO emissions. The median contribution of total nonroad emissions to VOC and NO_x inventories in summer, and CO inventories in winter, ranges from 7.4-12.6% VOC, 14.5-17.3% NO_x, and 5.2-9.4% winter CO, depending on the area. Under the most conservative assumptions, using new engine emission factors and choosing the lowest emission estimates from the combined emission inventories, the minimum contribution by pollutant for all cities studied in the NEVES (19 ozone and 16 CO nonattainment areas) were as follows: 2.9% VOC, 7.6% NO_x, and 2.2% CO.

The individual nonroad categories contributing most heavily to the results vary by pollutant and season. For instance, lawn and garden equipment and recreational marine equipment are major contributors to summertime VOC emissions, accounting for a median ranging from 2.4% to 4.7% and 2.2% to 4.0% of the total VOC inventory, respectively, in tons per summer day, depending on the area. Light commercial equipment and industrial equipment account for a median ranging from 2.0% to 3.7% and 1.1% to 1.5% of the total CO tons per winter day inventory, respectively. By far the largest contributors to nonroad NO_x emissions is construction equipment. Construction equipment accounts for a median of 8.4% to 9.7% of the total NO_x inventory. Agricultural, industrial, airport service, and commercial marine engines are also important contributor of NO_x in some areas. Particulate matter (PM) from nonroad sources is estimated to contribute a median of 1.8% of the total PM inventory. The two equipment categories that are the major contributors are construction equipment and commercial marine engines.

The limited availability of toxic emission data for nonroad sources makes it difficult to quantify precisely the contribution from these sources. Section 5.3.4 indicates that approximately 30% of mobile source benzene emissions, or 25% of total benzene emissions, is attributable to nonroad sources. This is almost identical to the estimate of 25.37% in the NEVES. The NEVES also provides an estimate of 13.05% of total formaldehyde from nonroad sources, and an estimate of 5.55% of total particulate matter from nonroad sources. The NEVES report does not provide an estimate of the 1,3-butadiene contribution from nonroad sources, but Section 7.3.4 indicates that 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 report nor the NEVES provides an estimate of the nonroad contribution to total acetaldehyde emissions.

15.0 INITIAL COST CONSIDERATIONS

15.1 Costs of Various Regulatory Programs

EPA has not done an independent evaluation of cost considerations associated with controlling toxic emissions from motor vehicles. 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 cost information in this section was taken from various regulatory impact analyses and related documents. The following regulatory programs were examined: Tier 1 standards, California standards, reformulated gasoline program, Inspection/Maintenance programs, winter oxygenated fuels CO program, diesel particulate standards, and the diesel fuel sulfur regulation. Cost information estimates for all the gasoline related programs are summarized in Table 15-1. Where available, costs are expressed per ton of VOC reduction. It should be noted that cost-effectiveness varies if all the cost is assigned to VOC, spread out over the sum of VOC and NO_x, assigned to NO_x, etc. The costs for the diesel related standards are not included in Table 15-1 since they are associated with reduction of diesel particulate rather than VOC emissions.

15.1.1 Tier 1 Standards

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 lower end of the range, the estimated year 2005 reductions in VOC for light-duty vehicles and trucks is 160,000 tons, at a cost of $590 million, or a cost-per-ton of about $3700 (EPA, 1991b; Pechan and Associates, 1990). This figure does not incorporate discounting of either the national cost or national emissions reduction. The 2005 model represents full implementation of the NMHC phase-in and reasonable turnover of older vehicles.

For the upper end of the range, EPA calculated a cost per ton of $6018 by adjusting the lower estimate to incorporate a 10 percent discount rate (EPA, 1991a,b). The discount factor is determined by $1/(1 + r)^{(t-0.5)}$, where r equals the appropriate discount rate (in this case 10% or 0.10) and t equals the age of the vehicle in years. The use of (t-0.5) as the exponent of the denominator has the effect of assigning the benefits for a given year at the mid-year point, rather than at the end. This process assumes a survival rate at 20 years of age of 33%, with all costs being discounted by 10% to adjust the benefits received in each year of the vehicle's life to 1991 dollars.

Table 15-1. Cost/Effectiveness of Various Regulatory Programs.

Program	Cost	References
Tier 1 Standards	$3700 or $6018/ton VOC	Regulatory Impact Analysis: Tier 1 Light-Duty Tailpipe Standards and Useful Life Requirements, January, 1991
Reformulated Gasoline Program	$1500 to $3700/ton VOC	Memo from Christine M. Brunner to John Chamberlin: Cost-Effectiveness of Phase I Reformulated Gasoline, March 1993
I/M Programs	$461 and $4518/ton VOC accounting for CO and NO_x (approximately 33 to 41% of the VOC reduction is attributable to exhaust emissions)	I/M Costs, Benefits, and Impacts Analysis (draft), February 1992
Winter Oxygenated Fuels Program	$685 to $880 million per year	Assessment of the Long-Run Costs of the Oxygenated Fuel Provisions, Sobotka and Co., Inc. for EPA, June 26, 1991

15.1.2 California Standards

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. A range of cost
estimates has been submitted to EPA 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. Many of these
estimates are associated with the cost of electrically heated
catalyst systems, which may be used to meet LEV standards. At
the low end of the range of cost estimates, the California Air
Resources Board estimated an added cost, in 1990 dollars, of $70
for gasoline-powered TLEVs, and $170 for gasoline-powered LEVs
and ULEVs (Albu et al., 1992). However, a consulting firm
estimates that electrically heated catalyst systems will add, on
average, a $1010 incremental retail price increase in 1991
dollars to the cost of a vehicle (Automotive Consulting Group,
1991). In contrast, an electrically heated catalyst manufacturer
has estimated the cost of a system would range from $200 to $330,
depending on engine size, in the first years of production (W. R.
Grace and Co., 1992). In addition, CARB claims that improved
fuel controls and conventional catalysts will be sufficient for
most vehicles to achieve the LEV standards, without the need for
electrically heated catalysts (CARB, 1992).

15.1.3 Reformulated Gasoline Program

EPA's cost-effectiveness estimates for a Phase I
reformulated gasoline is $1,500 to $3,700/ton VOC (EPA, 1993).
This range includes the effects of RVP class and type of I/M
program. Since it was evaluated as a VOC control strategy, only
the cost of summertime RVP and oxygenate use is included in this
estimate because they alone contribute to VOC reductions. The
costs associated with toxics reductions, namely, the cost of
benzene control and wintertime oxygenate use, were not included.

15.1.4 Inspection/Maintenance (I/M) Programs

The cost of an I/M program is determined by summing the
estimated inspection fee costs, the estimated repair costs, and
the negative cost of estimated fuel economy benefits (gallons of
fuel saved × dollars per gallon). The emission benefits of an
I/M program are determined by subtracting the estimated emissions
with the program from the emissions with no I/M program (EPA,
1992a). By using the Cost Effectiveness Model (CEM) and Mobile
4.1, a variety of runs are conducted to interpolate the final ton
per year value.

Since the I/M program yields CO benefits as well as VOC
benefits, and some areas need reductions in both, the determined
costs have been split among pollutants. High-option I/M can also
obtain significant NO_x benefits, so the cost is also split to
account for this. The actual 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. These costs represent
both ends of the I/M spectrum, the biennial high option and the
basic I/M program, respectively.

15.1.5 Winter Oxygenated Fuels Program

Most of the information below is contained in a document
prepared by Sobotka and Company, Inc. for EPA (Sobotka and
Company, 1991).

The Clean Air Act Amendments of 1990 require, starting in
1992, that gasoline sold during the winter in CO nonattainment
areas must contain at least 2.7 percent oxygen by weight. The
time period during which gasoline must contain oxygenates depends
on local conditions but must be at least four months (a shorter
time period is possible). The period runs from November through
February for most of the cities affected by the regulation.
However, the period may last year-long for the New York
metropolitan area.

The long-run social costs of the oxygenate program probably
would be in the range of about $685 to $880 million per year,
depending on the extent of spillover and on the price of crude
oil, or about 4.2 to 4.9 cents per gallon of oxygenated fuel.
This includes the cost of the ethanol subsidy ($140 million), the
cost of the mileage loss associated with oxygenated gasoline
($160 million), the cost of a summer oxygenate program for the
New York Consolidated Metropolitan Statistical Area (CMSA) ($20
to $40 million), the cost of transporting to the Gulf Coast
Alaskan crude oil that is displaced from the West Coast ($90
million), and other costs incurred by refiners to produce
oxygenated gasoline ($270 to $450 million).

Costs for the program on the West Coast are projected to be
about double those for the rest of the country on a per gallon
basis, mainly because the percentage of West Coast gasoline that
would be oxygenated is high, about 90 percent. This limits the
ability of refiners to offset the cost of MTBE and ethanol by
shifting high value blendstocks to conventional gasoline.

Prices of oxygenated gasoline at the pump are likely to
increase by about 4.2 to 5 cents per gallon on the West Coast and
by about 2.4 to 3.4 cents per gallon elsewhere. The effects of
oxygenates on mileage effectively would increase gasoline costs
to the consumer by about another cent a gallon. Cost estimates
expressed as cost per ton VOC or CO reduced were not estimated
for this program.

15.1.6 Diesel Particulate Standards

Various particulate standards for heavy-duty diesel engines
are in place for the 1988 through 1990 model years, the 1991
through 1993 model years, and for 1994 and later model years.
Cost estimates are presented in Table 15-2 for 1988-90, 1991-93,
and 1994-96 for the heavy-duty diesel particulate standards (EPA,
1985). All figures are in 1984 dollars, and a 10 percent
discount rate is assumed. The ranges given for the three year
aggregate costs are due to the range of projected fuel economy
penalties.

15.1.7 Diesel Fuel Sulfur Regulation

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
particulate matter reduced and were estimated using a calendar-
year approach discounted over a 33-year period (1994-2025). Cost
effectiveness of sulfur control, in dollars per ton of
particulate reduced, are shown in Table 15-3 for three engine
wear benefit scenarios, as well as results assuming no engine
wear benefits exist (EPA, 1990b). This rule was finalized on May
7, 1992 (EPA, 1992b).

The cost effectiveness of sulfur control was developed by
taking into account the wear credit. The wear credit is the
result of lower engine wear with low sulfur fuel. The potential
effects of reduced engine wear could result in lower engine oil
cost and less frequent oil change intervals, or longer engine and
vehicle life, or longer engine life with fewer total rebuilds
(EPA, 1990a).
Benefits were estimated for three different scenarios. The
maximum wear credits scenario includes both an extension in
engine and vehicle life. This credit is three to six times the
refining cost for sulfur controls. The minimum wear credits
result from an increase in oil change intervals and a slight
decrease in oil cost per quart (lower total additive content due
to low sulfur fuel). This credit is applicable to 1991 and later
vehicles. In 2025, this credit is between 31 percent and 74
percent of the refinery costs. In all cases, the credits for
medium and heavy-duty diesel vehicles together are at least 65
percent of the total credit.
15.2 Qualitative Discussion of Toxics Benefits

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

Table 15-2.   Cost Associated With Achieving the Heavy-Duty Diesel Particulate Standards.

Time Period	Standard To Be Achieved	Three Year Aggregate Cost
1988-1990	0.60 g/BHP-hr	$44 million (discounted to 1988)
1991-1993	0.25 g/BHP-hr	$746 to $868 million (discounted to 1991)
1994-1996	0.10 g/BHP-hr	$338 to $394 million (discounted to 1994)

Table 15-3.   33-Year Urban Cost-Effectiveness Analysis of Sulfur Control.

Wear Credit Scenario	Cost-Effectiveness ($/ton)
Maximum Wear Credit	-$68,148 to -$19,253
Minimum Wear Credit	-$3906 to $4304
No Wear Credit included	$2826 to $6773

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 you are using gasoline, 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.

15.2.1 Tier 1 Standards

The Tier 1 standards were developed to reduce exhaust emissions and will be phased in beginning with the 1994 model year. This regulation will reduce exhaust VOC and thus should reduce benzene, 1,3-butadiene, formaldehyde, and acetaldehyde proportionally. These standards should also reduce the atmospheric transformation of hydrocarbons into secondary aldehydes due to the reduction of VOCs. There is no measure included in this regulation to control evaporative emissions.

15.2.2 California Standards

The California standards, as they relate to LEVs and ULEVs using gasoline, should also reduce exhaust emissions as discussed above. They should also affect atmospheric aldehyde formation and not influence evaporative emissions. This program adopts these increasingly stringent vehicle certification standards beginning in 1994.

15.2.3 Reformulated Gasoline Program

During Phase 1 of the program, which runs from January 1, 1995 through 1999, federal reformulated fuel must contain at least 2.0% oxygen, and must not result in a NO_x increase. This program is mandated in the nine worst ozone areas, with other areas able to opt in to the program. During the high ozone season, reduction of both ozone forming VOCs and air toxics must be at least 15%, relative to emission levels of the 1990 model year vehicles with baseline gasoline. The approach for achieving the Phase 1 reductions is to reduce the RVP, add an oxygenated component, and limit the benzene and aromatics content of the gasoline. This results in both exhaust and evaporative emissions reductions.

During Phase 2 of the program, which begins in 2000, VOC and NO_x reductions must be at least 25%, or 20% if the 25% reduction is judged to be unfeasible. A complex model is being developed by EPA to predict VOC and toxics benefits as a function of a number of fuel parameters. At this point in time, it is difficult to predict the future composition of Phase 2 gasoline; however, since air toxics reductions are implicitly required, both Phase 1 and 2 reformulated gasolines should clearly result in air toxics benefits.

15.2.4 Inspection/Maintenance (I/M) Programs

In addition to tighter standards on new vehicles and their fuels, the Clean Air Act Amendments of 1990 required the implementation of I/M programs in areas that have been determined to be in non-attainment for ozone or carbon monoxide. Depending on the severity of the problem, moderate non-attainment areas (or marginal areas with an I/M program) will have to implement a basic I/M program. Enhanced I/M programs will be implemented in serious, severe, and extreme non-attainment areas.

The basic I/M program includes an annual, centralized idle test and a visual inspection for the catalyst and fuel inlet restrictor. For the basic program, EPA estimates that there will be an 11% reduction in total VOCs for light-duty gas vehicles (LDGV) when compared to a non-I/M scenario. Basic I/M assumes only exhaust control; thus, the 11% decrease in VOCs is totally attributable to exhaust VOC. Exhaust VOC is approximately 33% of the total VOCs emitted by the vehicle.

The biennial high-option includes an IM240 exhaust test and purge testing of the evaporative control system of 1986 and later vehicles. The high-option I/M program then addresses both exhaust and evaporative emissions and results in a 34% reduction in total VOCs for LDGV when compared to a non-I/M scenario. The increase in the VOC reduction for LDGV is mainly accounted for by the enhanced testing of the evaporative control system (44% of VOC reduction) when compared to a non-I/M scenario. The reduction in exhaust emissions improves slightly, increasing by about 9% over the basic I/M program. The exhaust VOC would account for approximately 41% of the total VOC for LDGV due to the increased control of evaporative emissions. Air toxics would decrease in proportion to the exhaust VOC reductions and benzene would be reduced further due to the tighter evaporative controls.

15.2.5 Winter Oxygenated Gasoline Program

The winter oxygenated gasoline program should have very similar benefits to that of the Phase 1 reformulated gasoline program due to the similarity of the fuels. This program is scheduled to go into effect in November 1992.

15.2.6 Diesel Particulate Standards and Fuel Sulfur Regulation

The overall effects of the diesel particulate standards and the diesel fuel sulfur regulation have been combined to determine the benefits of controlling diesel emissions. The analysis of these effects is based on the cancer risks determined in Section 9.7 and represent a composite of light and heavy duty diesel engines.

It was determined that 106 cancer cases would be due to diesel particulate exposure in 1990. This number decreases by 40% in 1995, 57% in 2000, and 72% in 2010.

15.3 References for Section 15

Albu, Steve, Liwen Kao, and Tom Cackette. 1992. The Effectiveness of the California Low-Emission Vehicle Program. Prepared for the Air and Waste Management Association. AWMA Paper 92-168.01.

Automotive Consulting Group. 1991. The Electrically Heated Catalyst: A "Systems" Cost Analysis. Automotive Consulting Group, Ann Arbor, MI, September 10, 1991.

CARB. 1990. Proposed Regulations for Low-Emission Vehicles and Clean Fuels, Technical Support Document. August 13, 1990.

CARB. 1992. Supplemental Submittal of the California Air Resources Board to EPA Docket No. A-91-71. Submittal IV-G-49, August 19, 1992.

EPA. 1985. Federal Register, 40 CFR Parts 86 and 600, March 15, 1985, pages 10606-10708.

EPA. 1990a. Regulatory Impact Analysis: Control of Sulfur and Aromatics Contents of On-Highway Diesel Fuel. Office of Air and Radiation, Office of Mobile Sources, Ann Arbor, MI. June, 1990.

EPA. 1990b. Federal Register, Vol. 55, 40 CFR Parts 80 and 86, August 21, 1990, pages 34120-34151.

EPA. 1991a. Analysis of NMHC Cost-Effectiveness. Memo from James McCargar to Bob Maxwell. January 31, 1991.

EPA. 1991b. Regulatory Impact Analysis: Tier 1 Light-Duty Tailpipe Standards and Useful Life Requirements. Office of Air and Radiation, Office of Mobile Sources, Ann Arbor, MI. January, 1991.

EPA. 1992a. I/M Costs, Benefits, and Impacts, Draft. Office of Air and Radiation, Office of Mobile Sources, Ann Arbor, MI. February, 1992.

EPA. 1992b. Federal Register, Vol. 57, No. 89, 40 CFR Parts 80 and 86, May 7, 1992, pages 19535-19537.

EPA. 1993. Cost-Effectiveness of Phase I Reformulated Gasoline. Memo from Christine M. Brunner to John Chamberlin. March 18, 1993.

Pechan and Associates, Inc. 1990. Ozone Nonattainment Analysis A Comparison of Bills. January 26, 1990.

Sobotka and Company, Inc. 1991. Assessment of the Long-Run Costs of the Oxygenated Fuel Provisions, Draft. June 26, 1991.

W. R. Grace and Co. 1992. Letter from Alan D. Fiers, Vice President and Director of Government Relations to Charles Freed, EPA. November 30, 1992. Submittal IV-G-73 to EPA Docket No. A-91-71.

16.0 MOTOR VEHICLE TOXICS IN TITLE III AND METALLIC POLLUTANTS

The list of 189 compounds in Title III of the Clean Air Act Amendments of 1990 were reviewed to identify those compounds that are either known or, based on their structure, have the potential to be emitted from motor vehicles. The metals chosen are all potential fuel additives. The various health-based criteria that have been developed for these compounds are presented in Table 16-1. This information has been largely excerpted from the contractor report, <u>Motor Vehicle Air Toxics Health Information</u>, Clement International Corporation, September, 1991 and updated where appropriate. A section devoted to dioxins is included, since EPA has received public comments specifically related to dioxins from motor vehicles. In addition, a section on methyl tertiary butyl ether (MTBE) is included due to recent public health issues associated with gasoline containing 15% MTBE. Finally, a brief section on n-nitrosodimethylamine (NDMA) is included, since it has been measured in vehicle interior emissions and in diesel crankcase emissions. NDMA is classified by EPA as a probable human carcinogen and is present in the Title III list.

16.1 Dioxins

Over 75 different chlorinated dioxin isomers have been identified. One of the 22 isomers with four chlorinated atoms is 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). This dioxin compound and other PCDDs have high molecular weights and in the atmosphere are expected to preferentially adsorb to airborne particulate matter, particularly the more chlorinated species. Removal mechanisms include dry and wet deposition and photodegradation. Photodegradation appears to be the most significant natural degradation mechanism for PCDDs. 2,3,7,8-TCDD is the dioxin compound of most interest since it is thought to be the most toxic of the chlorinated dioxins and is most often associated with exposure and potential health risks to humans based on available data.

There has been recent interest expressed to the EPA Office of Mobile Sources (see specific comments by Konheim and Ketcham and Zephyr Consulting in Appendix I) that gasoline and diesel powered vehicles may be responsible for more dioxin emissions than the combined emissions from waste incineration sources. Several European studies, Rappe, et al., (1988), Ballschmitter, et al., (1986), and Marklund, et al., (1987) appear to indicate that much of the background ambient dioxin levels are attributable to the use of leaded gasoline and the use of halogenated additives. Marklund, et al., (1987) could not find PCDDs or PCDFs in exhaust samples using unleaded gasoline fueled vehicles equipped with catalytic converters. A Norwegian tunnel study (Larssen et al., 1990) appears to implicate diesel engines as a major source of dioxin.

Several commentors reiterated these points with particular emphasis on dioxin emissions from diesels.

In the U.S., a preliminary study by the California Air Resources Board (CARB, 1987), described an exploratory dioxin sampling program that was conducted on exhaust from seven vehicles. The major limitations CARB cites in the study itself are the small sample size, the use of low resolution (rather than high resolution) mass spectrometry, and the presence of interferences from other organic compounds in the samples which prohibit the use of the data for estimating emissions. CARB states that this report was not intended to support general conclusions about dioxins in motor vehicle exhaust. CARB also has stated that, due to the report's preliminary nature, it should not be cited or quoted (CARB, 1992). More definitive dioxin measurements from motor vehicles would be helpful to better quantify emissions and reduce the uncertainties.

CARB also commissioned a study to assess the ambient concentrations of polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in the South Coast Air Basin (Hunt et al., 1990). The objective of this research effort was to determine baseline ambient concentrations of PCDDs/PCDFs, particularly in areas of high population density. In general, the conclusions state that the evidence is strongly suggestive that combustion sources are the major contributing factor to atmospheric burdens of PCDDs/PCDFs; however, confirmation of the vehicular contribution cannot be provided by examination of the data currently available from these samples.

It was further found that the majority of the atmospheric burdens of PCDDs/PCDFs in the South Coast Air Basin are represented by non 2,3,7,8-substituted species, which are not of toxicological significance as defined by the California Department of Health Services. TCDDs, and in particular 2,3,7,8-TCDD, are virtually non-existent in the South Coast Air Basin. 2,3,7,8-TCDD was confirmed in only two samples, both of which were collected during the Spring 1989 session. These levels were measured as 8.6 fg/m^3 at West Long Beach (monitor near a petroleum refinery) and 34 fg/m^3 at the Cal Trans site (monitor near a highway intersection). The measurement fg/m^3 is 1×10^{-15} (one quadrillionth) gram per cubic meter. Photochemical degradation of this isomer may account in part for its virtual absence in the atmosphere in Southern California. It was also stated that the average PCDDs/PCDFs concentrations for all sessions (excluding the high measurements from the December 1987 campaign) represent values typically found in other U.S. urban and suburban locations.

It should also be noted that the EPA's Office of Research and Development is presently carrying out a scientific reassessment of dioxins and related compounds. A workshop draft report is presently undergoing agency and public review. Also, EPA OMS plans a reevaluation of mobile source data.

16.2 MTBE

The Clean Air Act, as amended in 1990, requires the use of oxygenated gasoline in the winter months in 39 areas of the country that exceed national health standards for carbon monoxide (CO). In these areas, gasoline was changed by adding oxygenates such as ethanol or methyl tertiary butyl ether (known as MTBE), and reducing certain other organic compounds.

Recently, a public health issue has arisen as a result of public complaints in three cities using oxyfuels with approximately 15% MTBE. Although the program in other cities has run relatively smoothly, citizens in Fairbanks and Anchorage, Alaska, and Missoula, Montana have complained of health symptoms (headaches, coughs, eye irritation, nausea, and dizziness) which they believe are associated with exposures to gasoline blended with MTBE. While current data suggest these acute symptoms are generally mild and of short duration, EPA is currently conducting a cooperative research program to determine whether there is, in fact, an increase in such symptoms associated with exposures to MTBE-blended gasoline. This research is being supported and/or conducted by EPA, the State of Alaska, the Centers for Disease Control and Prevention of the Department of Health and Human Services, and industry. In the interim, EPA recently completed a paper which discusses the public health issues associated with MTBE-oxygenated gasolines (EPA, 1993). This paper will be revised this summer based on the findings of ongoing research.

16.3 N-Nitrosodimethylamine

N-Nitrosodimethylamine (NDMA) is classified by EPA as a B2, probable human carcinogen. This classification is based on the induction of tumors at multiple sites in both rodents and nonrodent mammals exposed by various routes. The unit risk estimate from inhalation exposure is 1.4×10^{-2} per $\mu g/m^3$. No RfD or RfC is available (EPA, 1992).

Nitrosamines have been measured at low levels in vehicle interior emissions and in diesel crankcase emissions. In Smith and Baines (1982), fifty-eight vehicle interiors were sampled for nitrosamines, including NDMA. Of the fifty-eight vehicles sampled in the program, forty-nine contained NDMA at concentrations ranging from 0.024 to 0.388 $\mu g/m^3$. Total nitrosamine levels ranged from 0.01 to 0.63 $\mu g/m^3$. The study estimated that the daily intake of nitrosamines from vehicle interiors for a commuter in a vehicle 3 hours/day is less than that from a can of beer or from a strip of bacon.

An artifact-free method for the analysis of nitrosamines in diesel engine crankcase emissions was developed (Goff et al., 1980a). Nitrosamine emissions from diesel engine crankcases were measured from three heavy-duty engines and one light-duty engine (Goff et al., 1980b). NDMA was present in all engine tests. The maximum concentration was 28 $\mu g/m^3$. The study

concludes that nitrosamine levels in both heavy-duty and
light-duty diesel crankcase emissions result from nitrosation of
amine-type compounds in the lubricating oil vapors by oxides of
nitrogen in the crankcase emissions.

No work has been done since these studies.

Table 16-1. Health-Based Criteria for CAAA Title III Motor Vehicle Air Toxics and Metals.

Chemical Name	Reference Concentration (RfC)(mg/m^3)	Reference Dose (RfD) (mg/kg/day)	OSHA Final Rule Limits STEL (ppm) [mg/m^3]	ACGIH Threshold Limit Values TLV-TWA (ppm) [mg/m^3]	STEL (ppm) [mg/m^3]
Acetonitrile	Under review	6×10^{-3}	60 [105]	40 – S[1] [67]	60 [101]
Acrylic Acid	3×10^{-4}	8×10^{-2}	NA[2]	10 – S [30]	NA
Acrolein	2×10^{-5}	NA	0.3 [0.8]	0.1 [0.23]	0.3 [0.69]
Carbon Disulfide	NA	0.1	12 [36]	10 – S [31]	NA
Catechol	NA	NA	NA	5 [23]	NA
Chlorine	NA	NA	1 [3]	0.5 [1.5]	1 [3]
Cresols	NA	5×10^{-2}	NA	5 – S [22]	NA
Carbonyl Sulfide	NA	NA	NA	NA	NA
Dimethyl Sulfate	NA	NA	NA	0.1 – S [0.5]	NA

---

[1]"S," or "skin designation," refers to the prevention of employee skin absorption through use of gloves, coverall, goggles, or other appropriate personal protective equipment, engineering controls or work practices.

[2]Not Available

Chemical Name	Reference Concentration (RfC)(mg/m^3)	Reference Dose (RfD) (mg/kg/day)	OSHA Final Rule Limits STEL (ppm) [mg/m^3]	ACGIH Threshold Limit Values TLV-TWA (ppm) [mg/m^3]	STEL (ppm) [mg/m^3]
1,4-Dioxane	NA	NA	NA	25 - S [90]	NA
Diethyl Sulfate	NA	NA	NA	NA	NA
Dibenzofurans	NA	NA	NA	NA	NA
Ethylene Dibromide	NA	NA	NA	NA	NA
Ethyl Benzene	1	0.1	125 [545]	100 [435]	125 [545]
Ethylene Dichloride	NA	NA	2 [8]	10 [40]	NA
Hexane	2×10^{-1}	NA	NA	50 [180]	NA
Hexane Isomers	NA	NA	1,000 [3,600]	500 [1,800]	1,000 [3,600]
Lead	NA	NA	NA	[0.15]^3	NA
Methanol	NA	0.5	250 [328]	200 - S [260]	250 [328]
Methyl Ethyl Ketone	1	5×10^{-2}	300 [885]	200 [590]	300 [885]
Methyl t-Butyl Ether	5×10^{-1} ^{4}	NA	NA	NA	NA

---

^{3}For inorganic dusts and fumes

^{4}The RfC for MTBE is in a state of flux and is likely to change.

Chemical Name	Reference Concentration (RfC)(mg/m^3)	Reference Dose (RfD) (mg/kg/day)	OSHA Final Rule Limits	ACGIH Threshold Limit Values	
			STEL (ppm) [mg/m^3]	TLV-TWA (ppm) [mg/m^3]	STEL (ppm) [mg/m^3]
Naphthalene	NA	Withdrawn	15 [79]	10 [52]	15 [79]
Phenol	NA[5]	0.6	NA	5 - S [19]	NA
PAHs	NA	NA	NA	NA	NA
Propionaldehyde	NA[6]	NA	NA	NA	NA
Styrene	1	0.2	100 [426]	50 - S [213]	100 [426]
Toluene	4×10^{-1}	0.2	150 [565]	100 [377]	150 [565]
2,2,4-Trimethylpentane	NA	NA	NA	NA	NA
Xylenes	Under review	2	150 [651]	100 [434]	150 [651]
Copper	NA	1.3 mg/l[7]	NA	0.38[8] [1]	NA
Copper fumes	NA	NA	NA	0.08 [0.2]	NA

[5]Health effects data have been reviewed by EPA and determined to be inadequate for derivation of an RfC.

[6]EPA has determined that the database is insufficient to develop an RfC for propionaldehyde.

[7]No oral RfD exists for copper. This drinking water standard was derived by EPA for chronic and subchronic exposure.

[8]Copper dusts and mists.

16-7

Chemical Name	Reference Concentration (RfC)(mg/m^3)	Reference Dose (RfD) (mg/kg/day)	OSHA Final Rule Limits	ACGIH Threshold Limit Values	
			STEL (ppm) [mg/m^3]	TLV-TWA (ppm) [mg/m^3]	STEL (ppm) [mg/m^3]
Iron	NA	NA	NA	0.77[9] [5]	NA
Cerium	NA	NA	NA	NA	NA
Manganese	4×10^{-4}	1×10^{-1}	NA	2.23[10] [5]	NA
Manganese fumes	NA	NA	1.34 [3]	0.45 [1]	1.34 [3]
Selenium	NA	5×10^{-3}	NA	0.06 [0.2]	NA
Platinum	NA	NA	NA	0.13[11] [1]	NA
Platinum soluble salts	NA	NA	NA	0.0003 [0.002]	NA

[9]Iron oxides.

[10]Manganese dust and compounds.

[11]Platinum metals.

16-8

16.2 References for Chapter 16

Ballschmitter, K. et al. 1986. Automobile exhausts versus municipal waste incineration as sources of the polychlorinated dibenzodioxins (PCDD) and furans (PCDF) found in the environment. Chemosphere 15:901.

CARB. 1987. Evaluation Test for PCDD and PCDF Emissions from Motor Vehicles. Draft Report No. C-87-029, October, 1987.

CARB. 1992. Letter from William V. Loscutoff, Chief, CARB Monitoring and Laboratory Division to David Cleverly, U.S. EPA. November 25, 1992.

EPA. 1993. MTBE-Oxygenated Gasoline and Public Health Issues. Office of Research and Development. February, 1993.

EPA. 1992. Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

Goff, E.U., J.R. Coombs, D.H. Fine, and T.M. Baines. 1980a. Determination of n-nitrosamines from diesel engine crankcase emissions. Anal. Chem. 52:1833-1836.

Goff, E.U., J.R. Coombs, D.H. Fine, and T.M. Baines. 1980b. Nitrosamine Emissions from Diesel Engine Crankcases. SAE Technical Paper 801374.

Hunt, G., B. Maisel, and M. Hoyt. 1990. Ambient Concentrations of PCDDs/PCDFs in the South Coast Air Basin. Final Report prepared for the California Air Resources Board Document No. 1200-005-700. January 1990.

Larssen, S., E.M. Brevik, and M. Oehme. 1990. Emission factors of PCDD and PCDF for road vehicles obtained by a tunnel experiment. Dioxin 90, Vol. I.:453-456.

Marklund, S., C. Rappe, M. Tyskling, and K.E. Egebäck. 1987. Identification of polychlorinated dibenzofurans and dioxins in exhausts from cars run on leaded gasoline. Chemosphere, 16:29-36.

Rappe, C. et al. 1988. Identification and quantification of PCDDs and PCDFs in urban air. Chemosphere, 17:1.

Smith L.R. and T.M. Baines. 1982. Nitrosamines in Vehicle Interiors. SAE Technical Paper 820785.

APPENDIX A

EPA Workgroup Members

EPA Work Group Members

Mr. Dwight R. Atkinson
Office of Policy Analysis
Office of Policy, Planning, and Evaluation

Dr. Joseph J. Bufalini
Gas Kinetics and Photochemistry Research Branch
Atmospheric Research and Exposure Assessment Laboratory
Office of Modeling, Monitoring Systems, and Quality Assurance
Office of Research and Development

Mr. James W. Caldwell
Fuels Section
Field Operations and Support Division
Office of Mobile Sources
Office of Air and Radiation

Dr. Ila L. Cote
Health Effects Research Laboratory
Office of Health Research
Office of Research and Development

Mr. Stanley B. Durkee
Office of Technology Transfer and Regulatory Support
Office of Research and Development

Mr. Robert B. Faoro
Data Analysis Section
Monitoring and Reports Branch
Office of Air Quality Planning and Standards
Office of Air and Radiation

Mr. Bob Fegley
Office of Technology Transfer and Regulatory Support
Office of Research and Development

Mr. John T. Hannon
Office of General Counsel

Dr. Oscar Hernandez
Risk Analysis Branch
Existing Chemical Assessment Division
Office of Toxic Substances
Office of Pesticides and Toxic Substances

Dr. Alan H. Huber
Human Exposure Modeling Branch
Atmospheric Research and Exposure Assessment Laboratory
Office of Modeling, Monitoring Systems, and Quality Assurance
Office of Research and Development

Dr. Gary L. Kimmel
Human Health Assessment Group
Office of Health and Environmental Assessment
Office of Research and Development

Dr. Kenneth T. Knapp
Mobile Source Emissions Research Branch
Atmospheric Research and Exposure Assessment Laboratory
Office of Modeling, Monitoring Systems, and Quality Assurance
Office of Research and Development

Mr. Thomas F. Lahre
Pollutant Assessment Branch
Office of Air Quality Planning and Standards
Office of Air and Radiation

Mr. Mike Lidgard
Region 10

Dr. Chris Lindhjem
Regulatory Development and Support Division
Office of Mobile Sources
Office of Air and Radiation

Mr. Thomas R. McCurdy
Ambient Standards Branch
Office of Air and Radiation

Dr. William E. Pepelko
Human Health Assessment Group
Office of Health and Environmental Assessment
Office of Research and Development

Mr. Steven A. Shedd
Chemicals and Petroleum Branch
Office of Air Quality Planning and Standards
Office of Air and Radiation

Dr. Chon R. Shoaf
Hazardous Pollutant Assessment Branch
Environmental Criteria and Assessment Office
Office of Health and Environmental Assessment
Office of Research and Development

Data Sources Used in Calculation of Mass Fractions

1) Auto/Oil Air Quality Improvement Research Program. (Speciation data from this program obtained on disk.)

   Technical Bulletin No. 1: Initial Mass Exhaust Emissions Results from Reformulated Gasolines. December, 1990.

   Technical Bulletin No. 4: Mass Exhaust Emissions Results From Reformulated Gasolines in Older Vehicles. May, 1991.

   Technical Bulletin No. 5: Exhaust Emissions of Toxic Air Pollutants Using Reformulated Gasolines. June, 1991.

   Technical Bulletin No. 6: Emissions Results of Oxygenated Gasolines and Changes in RVP. September, 1991.

   Purpose of Study: These technical bulletins assess the exhaust and evaporative emissions and air quality effects of reformulated gasoline blends (15% MTBE and 0% MTBE) and 10% ethanol blends relative to industry average gasoline. We analyzed data for baseline gasoline, 8 15% MTBE blends and 4 10% ethanol blends.

   Vehicle Information: Speciation data was available for 10 current model LDGVs and LDGTs (1989 model year) with three-way and three-way plus oxidation catalysts, PFI, TBI, and carburetor fuel systems, low mileage (10,000 to 29,000 miles). Speciation data was available for 7 older model LDGVs and LDGTs (1983 - 1985 model year) with three-way, oxidation, and three-way plus oxidation catalysts, TBI and carburetor fuel systems, high mileage (42,000 - 79,000 miles). Speciated emissions were measured on current and older vehicles for baseline fuel and MTBE blends, but for 10% ethanol blends only speciation from current vehicles was measured.

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

   Purpose of Study: Comparison of exhaust and evaporative emissions and reactivity from vehicles running on unleaded regular gasoline and EC-1, an MTBE reformulated blend (5.5% MTBE).

   Vehicle Information: This study analyzed data from 5 1989 model year LDGVs with three-way catalysts, PFI, low-mileage.

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

Purpose of Study: Compare exhaust and evaporative emissions
and reactivity from vehicles running on unleaded premium
gasoline, pilot EC-Premium reformulated gasoline (9.0%
MTBE), and production EC-Premium reformulated gasoline
(12.5% MTBE).

Vehicle Information: 20 1980 to 1989 LDGVs and LDGTs with
three-way, oxidation and three-way plus oxidation catalysts,
PFI, TBI, and carburetor fuel systems, odometers ranging
from 5,000 to 140,000 miles.

4)   CARB memo from K. D. Drachand to Terry McGuire and Peter
     Venturini, "Butadiene Emission Factors," July 17, 1991.

     Purpose of Study: Determine 1,3-butadiene emissions for
     various vehicle categories and emission technologies, for
     vehicles running on gasoline.

     Vehicle Information: 78 LDGVs/LDGTs with three-way,
     oxidation, and no catalysts, PFI, TBI, and carburetor fuel
     systems, 2 LDDVs and 1 HDDV, mileage ranging from 10,000 to
     200,000 miles.

5)   Colorado Department of Health. 1987. Unpublished data from
     a motor vehicle emissions toxics study of regulated and non-
     regulated pollutants. Aurora Emission Technical Center.

     Purpose of Study: Measurement of exhaust TOG, total
     aldehydes, formaldehyde, acetaldehyde and benzene from
     vehicles running on unleaded gasoline, 10% ethanol and 11%
     MTBE. 1,3-butadiene measurements were not taken.

     Vehicle Information: Emissions were measured for 19 LDGVs.
     We analyzed data for 17 LDGVs running on gasoline and 10%
     ethanol. Of these, 6 vehicles had three-way catalysts, one
     had a three-way plus oxidation catalyst, one had an
     oxidation catalyst and 4 had no catalyst. 2 had PFI, 4 had
     TBI and the other 11 were carbureted. Odometer readings for
     these vehicles were unavailable.

6)   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.

     Purpose of Study: Compare exhaust and evaporative emissions
     and reactivity from vehicles running on industry average
     base fuels and four reformulated gasoline test blends (15%
     MTBE). We analyzed data for test blends 1 and 2, which Arco
     stated were the most commercially feasible.

Vehicle Information: 10 1990 LDGVs and LDGTs with three-way and three-way plus oxidation catalysts, PFI and TBI, low-mileage.

6)   Sigsby, John E., Silvestre Tejeda, William Ray, John M. Lang, and John W. Duncan. 1987. Volatile Organic Compound Emissions from 46 In-Use Passenger Cars. *Environ. Sci. Technol.* 21: 466-475.

Purpose of Study: Determine mass, detailed hydrocarbon, and aldehyde emissions from in-use vehicles running on gasoline. No 1,3-butadiene measurements were taken in this study.

Vehicle Information: 46 LDGVs with three-way, oxidation, three-way plus oxidation and no catalysts, PFI, TBI and carburetor fuel systems.

7)   Smith, Lawrence R. 1981. Characterization of Exhaust Emissions from High Mileage Catalyst-Equipped Automobiles. U.S. Environmental Protection Agency, Ann Arbor, Michigan. (EPA-460/3-81-024)

Purpose of Study: Regulated and unregulated exhaust emissions, particularly aldehydes, were evaluated for high mileage LDGVs. Benzene, formaldehyde, and acetaldehyde were measured, but not 1,3-butadiene.

Vehicle Information: 10 LDGVs, with odometer readings ranging from 37,000 to 68,000 miles, were included in this study. 3 had three-way catalysts, while the rest had oxidation catalysts. We analyzed only the vehicles with oxidation catalysts. All were carbureted.

8)   Springer, Karl J. 1979. Characterization of Sulfates, Odor, Smoke, POM and Particulates from Light and Heavy-Duty Engines -- Part IX. U.S. Environmental Protection Agency, Ann Arbor, Michigan. (EPA-460/3-79-007)

Purpose of Study: Regulated and unregulated exhaust emissions were compared for LDGVs, HDGVs, LDDVs and HDDVs, running on unleaded gasoline and no. 2 diesel fuel. Benzene, formaldehyde, and acetaldehyde were measured, but not 1,3-butadiene.

Vehicle Information: This study contained regulated and unregulated exhaust emissions data for 2 LDGVs with oxidation catalysts, one with fuel injection and one carbureted, 2LDDVs, 2 HDDVs, and 1 HDGV with no catalyst. Odometer readings for these vehicles were not available. We used data collected from all these vehicles in our analyses.

4

9)   Springer, Karl J. 1977. Investigation of Diesel-Powered
      Vehicle Emissions VII. U.S. Environmental Agency, Ann
      Arbor, Michigan. (EPA-460/3-76-034)

      Purpose of Study: Regulated exhaust emissions were measured
      for five diesel engine configurations, and both regulated
      and unregulated exhaust emissions were measured for 5 LDDVs.
      Benzene, formaldehyde, and acetaldehyde were measured for
      the LDDVs, but not 1,3-butadiene.

      Vehicle Information: We analyzed data from the 5 LDDVs.
      All were low mileage vehicles, with odometer readings
      ranging from about 2000 to 10,000 miles.

10)   Stump, Fred D., Kenneth T. Knapp and William D. Ray. 1990.
      Seasonal impact of blending oxygenated organics with
      gasoline on motor vehicle tailpipe and evaporative
      emissions. *J. Air Waste Manage. Assoc.* 40: 872-880.

      Stump, Fred D., Kenneth T. Knapp, William D. Ray, Charles
      Burton and Richard Snow. 1990. The Seasonal Impact of
      Blending Oxygenated Organics with Gasoline on Motor Vehicle
      Tailpipe and Evaporative Emissions -- Part II. SAE 902129.

      Purpose of Study: Speciated exhaust and evaporative
      emission rates were measured for vehicles running on MTBE
      (16.2% and 5.5%) and ethanol (8.1%) oxygenated fuel blends
      at several ambient temperatures.

      Vehicle Information: Emissions were measured for two LDGVs
      with TBI and three-way catalysts. Mileage on both vehicles
      was about 30,000 miles. We only analyzed data for emissions
      using 8.1% EtOH.

11)   Stump, Fred, Silvestre Tejeda, William Ray, David Dropkin,
      Frank Black, Richard Snow, William Crews, Paula Siudak, C.
      O. Davis and Phillip 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, Fred, Silvestre Tejada, William Ray, David Dropkin,
      Frank Black, Richard Snow, William Crews, Paula Siudak, C.
      O. Davis, Linnie Baker and Ned 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.

Purpose of Study: Speciated exhaust emissions data for
vehicles running on summer and winter grade gasoline were
measured at ambient temperatures ranging from about 70°C to
20°C.

Vehicle Information: 20 LDGVs were tested. 13 had three-
way catalysts, 6 had three-way plus oxidation catalysts, and
one had an oxidation catalyst. 8 had PFI, 4 had TBI, and 8
were carbureted. Mileage ranged from about 5,000 to
100,000. We omly included 6 vehicles with three-way plus
oxidation catalysts and one with an oxidation catalyst in
our analysis.

12)   Stump, Fred D., Kenneth T. Knapp, William D. Ray, Richard
       Snow and Charles Burton. The composition of Motor Vehicle
       Organic Emissions Under Elevated Temperature Summer Driving
       Conditions (75 to 105°F). Unpublished.

       Purpose of Study: Speciated exhaust and evaporative
       emissions data for vehicles running on summer grade gasoline
       were measured at ambient temperatures of 75°F and 105°F.

       Vehicle Information: The database used for this study
       consisted of 15 vehicles. 14 had three-way catalysts and
       one had a three-way plus oxidation catalyst. We only used
       data from the vehicle with a three-way plus oxidation
       catalyst, which was carbureted and had 39970 miles on the
       odometer.

13)   Warner-Selph, Mary Ann, and Craig A. Harvey. 1990.
       Assessment of Unregulated Emissions from Gasoline Oxygenated
       Blends. SAE 902131.

       Purpose of Study: Compare exhaust and evaporative emission
       data from tests using baseline gasoline, 10% ethanol, 16.4%
       MTBE and 19.1% ETBE to determine what statistically
       significant differences existed for a number of unregulated
       emissions.

       Vehicle Information: 5 LDGVs were tested. 3 vehicles had
       three-way catalysts, one an oxidation catalyst, and one no
       catalyst. One had PFI, one TBI, and three were carbureted.
       Mileage ranged from about 5,000 to 80,000 miles.

14)   Warner-Selph, Mary Ann, and Joseph DeVita. 1989.
       Measurements of Toxic Exhaust Emissions from Gasoline-
       Powered Light Duty Vehicles. SAE 892075.

       Purpose of Study: Exhaust emission rates of toxics under
       California's air toxics program were measured for vehicles
       running on gasoline.

Vehicle Information: Toxics emissions were measured for two LDGVs, both with fuel injection. One had a three-way plus oxidation catalyst, while the other had a three-way catalyst. Mileage for both vehicles was between 20,000 and 30,000 miles.

15)   Urban, Charles. 1980. Regulated and Unregulated Exhaust Emissions from Malfunctioning Non-Catalyst and Oxidation Catalyst Gasoline Automobiles. U.S. Environmental Protection Agency, Ann Arbor, Michigan. (EPA-460/3-80-003)

Urban, Charles. 1980. Regulated and Unregulated Exhaust Emissions from Malfunctioning Three-Way Catalyst Gasoline Automobiles. U.S. Environmental Protection Agency, Ann Arbor, Michigan. (EPA-460/3-80-004)

Urban, Charles. 1980. Regulated and Unregulated Exhaust Emissions from a Malfunctioning Three-Way Catalyst Gasoline Automobile. U.S. Environmental Protection Agency, Ann Arbor, Michigan. (EPA-460/3-8-005)

Urban, Charles. 1981. Unregulated Exhaust Emissions from Non-Catalyst Baseline Cars Under Malfunction Conditions. U.S. Environmental Protection Agency, Ann Arbor, Michigan. (EPA-460/3-81-020)

Purpose of Study: Regulated and unregulated exhaust emissions from LDGVs with various catalyst types running on unleaded gasoline were measured under properly functioning and malfunctioning conditions. Benzene, formaldehyde, and acetaldehyde were measured, but not 1,3-butadiene.

Vehicle Information: Emissions from 13 LDGVs running on unleaded gasoline were analyzed in these reports. 2 had three-way catalysts, 2 had three-way plus oxidation catalysts, 4 had oxidation catalysts and five had no catalysts. The remaining vehicle had a 1978 California emission control package. All these vehicles except one were carbureted. We analyzed data from the 2 three-way plus ox-cat, 4 ox-cat and 5 non-cat vehicles. Odometer readings for the vehicles were not given in the reports.

LDGV (3-WAY+OX-CAT -- FTP Data)

| | Exhaust mg/mile | | | | | | | Fraction of TOG | | | | | |
	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene	TOG, g/mile	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene
0% MTBE													
Urban, 1980b, 1980c (EPA-460/3-80-004, 005)													
41 1978 Pinto CARB	47.400	3.200	1.270	0.000	N.A.	3.060	0.11	0.4235	0.0286	0.0113	0.0000	N.A.	0.0273
1979 Marquis CARB	73.310	7.450	3.880	0.180	N.A.	7.440	0.21	0.3431	0.0349	0.0182	0.0008	N.A.	0.0348
Average	60.355	5.325	2.575	0.090		5.250	0.16	0.3833	0.0317	0.0148	0.0004		0.0311
Sigsby et al., 1987 (46 car study)													
3 1981 Chev Citation (Carb)	93.430	9.400	1.750	0.100	N.A.	11.230	0.30	0.3166	0.0319	0.0059	0.0003	N.A.	0.0381
8 1982 Mazda RX-7 (Carb)	242.490	35.280	18.100	6.520	N.A.	7.375	0.85	0.2837	0.0413	0.0212	0.0076	N.A.	0.0086
21 1982 Olds Delta 88 (Carb)	127.190	25.130	5.750	2.170	N.A.	11.224	0.52	0.2451	0.0484	0.0111	0.0042	N.A.	0.0216
28 1981 Merc Lynx (Carb)	160.480	22.400	6.570	2.880	N.A.	6.880	0.65	0.2464	0.0344	0.0101	0.0044	N.A.	0.0106
29 1981 VW Jetta (FI)	95.470	13.320	7.680	3.020	N.A.	10.080	0.54	0.1770	0.0247	0.0142	0.0056	N.A.	0.0187
30 1982 Chev Chevette (Carb)	98.140	6.790	6.570	2.430	N.A.	3.903	0.23	0.4194	0.0290	0.0281	0.0104	N.A.	0.0167
36 1981 Chev Citation (Carb)	102.340	20.800	9.750	3.100	N.A.	8.996	0.38	0.2718	0.0552	0.0259	0.0082	N.A.	0.0239
41 1981 Chev Citation (Carb)	85.290	15.460	1.350	0.860	N.A.	3.480	0.19	0.4412	0.0800	0.0070	0.0044	N.A.	0.0180
Average	125.604	18.573	7.190	2.635		7.896	0.46	0.3002	0.0431	0.0154	0.0057		0.0195
Stump et al., 1989, 1990 (MSERB Low Temp. Study All Carb)													
1986 Ford Mustang	126.900	29.000	24.900	7.030	0.200	21.800	1.13	0.1124	0.0257	0.0220	0.0062	0.0002	0.0193
1987 Nissan Sentra	46.310	2.570	6.250	0.450	0.050	4.810	0.16	0.2845	0.0158	0.0384	0.0028	0.0003	0.0295
1986 Dodge Omni	165.300	25.100	9.380	*	0.190	14.240	1.14	0.1451	0.0220	0.0082	N.A.	0.0002	0.0125
1987 Toyota Corolla	64.400	8.000	1.210	0.840	0.100	14.400	0.30	0.2182	0.0271	0.0041	0.0028	0.0003	0.0488
1986 Olds Cutlass Sup	158.100	31.900	1.500	0.830	0.200	4.400	0.36	0.4439	0.0896	0.0042	0.0023	0.0006	0.0124
1987 Chev Monte Carlo	132.200	38.700	5.800	2.300	0.500	20.800	0.55	0.2406	0.0704	0.0106	0.0042	0.0009	0.0379
Average	115.535	22.545	8.173	1.908	0.207	13.408	0.61	0.2408	0.0418	0.0146	0.0031	0.0004	0.0267
Stump et al., unpublished (MSERB High Temp. Study)													
1986 Chev Caprice (Carb)	103.880	27.430	23.520	15.280	4.280	22.530	1.36	0.0762	0.0201	0.0173	0.0112	0.0031	0.0165
							0.00						
Warner-Selph and DeVita, 1989 (CARB Toxics Study)							0.00						
1987 Ford Taurus (PFI)	N.A.	N.A.	2.100	1.080	0.770	15.000	0.32	N.A.	N.A.	0.0067	0.0034	0.0024	0.0476
Arco 91-03 (Unleaded Premium)													
1985 Ford Bronco (PFI)	157.200	30.100	8.800	5.000	0.700	27.800	0.66	0.2395	0.0459	0.0134	0.0076	0.0011	0.0424
1983 Ford Thunderbird (TBI)	397.200	54.460	26.970	9.250	8.040	105.180	2.68	0.1481	0.0203	0.0101	0.0035	0.0030	0.0392
1985 Chevrolet Camaro (Carb)	111.160	29.480	4.370	1.760	0.550	12.400	0.37	0.2993	0.0794	0.0118	0.0047	0.0015	0.0334
Average	221.853	38.013	13.380	5.337	3.097	48.460	1.24	0.2290	0.0485	0.0117	0.0053	0.0018	0.0383
Auto/Oil Study													
1989 Ford Mustang (SFI)	61.593	14.301	2.625	0.903	0.609	5.985	0.23	0.2632	0.0611	0.0112	0.0039	0.0026	0.0256
1985 Chevrolet Impala (Carb)	69.502	17.174	4.743	0.899	0.992	11.873	0.32	0.2203	0.0544	0.0150	0.0029	0.0031	0.0376
1984 Pontiac Grand Prix (Carb)	111.499	27.047	2.537	0.946	0.599	12.642	0.46	0.2435	0.0591	0.0055	0.0021	0.0013	0.0276
1985 Ford Tempo (TBI)	60.078	20.064	4.408	2.014	1.368	18.582	0.43	0.1406	0.0469	0.0103	0.0047	0.0032	0.0435
Average	75.668	19.647	3.578	1.191	0.892	12.271	0.36	0.2169	0.0554	0.0105	0.0034	0.0026	0.0336
AVERAGE**	115.634	20.582	7.671	2.794	0.766	15.444	0.58	0.2497	0.0418	0.0137	0.0043	0.0010	0.0277
9.0% MTBE													
Arco 91-03													
1985 Ford Bronco (PFI)	122.200	31.400	10.800	4.900	1.700	14.200	0.62	0.1977	0.0508	0.0175	0.0079	0.0027	0.0230
1983 Ford Thunderbird (TBI)	328.180	57.930	20.720	6.330	7.560	49.180	1.66	0.1982	0.0350	0.0125	0.0038	0.0046	0.0297
1985 Chevrolet Camaro (Carb)	115.350	32.530	7.120	2.770	0.750	12.570	0.45	0.2574	0.0726	0.0159	0.0062	0.0017	0.0280
1985 Chevrolet Camaro (Carb)	103.590	36.150	5.170	1.420	0.830	11.110	0.41	0.2514	0.0877	0.0125	0.0034	0.0020	0.0270
AVERAGE**	167.330	39.503	10.953	3.855	2.710	21.765	0.78	0.2262	0.0615	0.0146	0.0053	0.0028	0.0269
12.5% MTBE													
Arco 91-03													
1985 Chevrolet Camaro (Carb)	111.070	38.580	4.200	1.160	0.620	7.630	0.40	0.2789	0.0969	0.0105	0.0029	0.0016	0.0192

15.0% MTBE

Auto/Oil Study

1989 Ford Mustang (SFI)	59.641	12.996	3.629	1.064	0.513	6.023	0.21	0.2874	0.0626	0.0175	0.0051	0.0025	0.0290
1985 Chevrolet Impala (Carb)	68.520	16.950	3.330	0.750	1.020	12.390	0.31	0.2201	0.0545	0.0107	0.0024	0.0033	0.0398
1984 Pontiac Grand Prix (Carb)	108.224	25.042	3.686	1.064	0.494	12.274	0.42	0.2608	0.0603	0.0089	0.0026	0.0012	0.0296
1985 Ford Tempo (TBI)	57.645	18.305	4.900	1.295	1.295	17.290	0.40	0.1425	0.0452	0.0121	0.0032	0.0032	0.0427
AVERAGE**	73.508	18.323	3.886	1.043	0.831	11.994	0.33	0.2277	0.0557	0.0123	0.0033	0.0025	0.0353

* Acetaldehyde level (129.68 mg/mile) suspicious and not included.
**Data used in subsequent emission fractions calculations

01/28/1988

LDGV (3-WAY -- FTP Data)

	Exhaust mg/mile							Fraction of TOG					
	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene	TOG, g/mile	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene
0% MTBE													
Arco 91-02 (Unleaded Reg) All FI													
21 1989 Toyota Camry	26.320	4.780	3.520	1.060	1.220	12.240	0.20	0.1333	0.0242	0.0178	0.0054	0.0062	0.0620
22 1989 Pont. Grand Prix	44.120	8.460	7.400	3.420	1.570	17.550	0.26	0.1709	0.0328	0.0287	0.0132	0.0061	0.0680
23 1989 Ford Taurus	39.880	4.200	5.230	2.080	1.360	13.080	0.28	0.1432	0.0151	0.0188	0.0075	0.0049	0.0470
24 1989 Dodge Dynasty	50.560	9.670	5.960	3.240	3.640	22.810	0.53	0.0951	0.0182	0.0112	0.0061	0.0068	0.0429
25 1989 Nissan Maxima	27.380	5.110	3.870	1.500	2.210	11.340	0.27	0.1020	0.0190	0.0144	0.0056	0.0082	0.0423
Average	37.652	6.444	5.196	2.260	2.000	15.404	0.31	0.1289	0.0219	0.0182	0.0076	0.0064	0.0524
Arco 91-03 (Unleaded Premium)													
35 1988 Pontiac Grand Am (PFI)	8.100	2.600	3.200	2.500	0.400	8.900	0.15	0.0526	0.0169	0.0208	0.0162	0.0026	0.0578
37 1981 Olsmobile Delta 88 (Carb)	251.630	29.280	22.730	9.980	11.540	84.710	1.71	0.1473	0.0171	0.0133	0.0058	0.0068	0.0496
39 1984 Honda Accord (Carb)	103.350	8.310	12.741	4.503	1.840	35.570	0.88	0.1173	0.0094	0.0145	0.0051	0.0021	0.0404
40 1987 Nissan 300ZX (PFI)	62.730	14.000	8.290	1.900	2.400	18.160	0.48	0.1318	0.0294	0.0174	0.0040	0.0050	0.0382
41 1988 Toyota 4WD Pickup (PFI)	61.270	7.130	2.850	0.730	1.050	24.310	0.48	0.1269	0.0148	0.0059	0.0015	0.0022	0.0503
42 1988 Toyota Celica (PFI)	40.250	5.680	1.200	0.800	0.600	12.800	0.32	0.1254	0.0177	0.0037	0.0025	0.0019	0.0399
44 1986 Mercedes 190E (PFI)	101.220	25.990	4.040	1.700	3.130	58.230	0.98	0.1036	0.0266	0.0041	0.0017	0.0032	0.0596
45 1989 Lincoln Continental (PFI)	67.700	11.570	3.480	0.900	1.120	32.250	0.55	0.1231	0.0210	0.0063	0.0016	0.0020	0.0587
46 1990 Plymouth Acclaim (PFI)	62.880	13.220	1.920	1.240	2.510	37.210	0.75	0.0839	0.0176	0.0026	0.0017	0.0034	0.0497
47 1984 Toyota Celica (PFI)	89.600	10.500	4.700	1.300	1.800	64.600	0.98	0.0912	0.0107	0.0048	0.0013	0.0018	0.0658
48 1985 Ford Mustang (Carb)	99.500	22.250	11.150	4.300	2.150	28.950	0.75	0.1328	0.0297	0.0149	0.0057	0.0029	0.0386
49 1987 Oldsmobile Regency (PFI)	76.800	15.300	1.100	1.500	1.400	56.000	0.86	0.0892	0.0178	0.0013	0.0017	0.0016	0.0651
56 1982 Nissan Maxima (PFI)	132.900	28.500	6.300	3.900	3.500	83.300	1.17	0.1136	0.0244	0.0054	0.0033	0.0030	0.0712
57 1986 Buick Park Avenue (PFI)	44.000	4.400	4.200	1.400	1.100	13.500	0.29	0.1525	0.0152	0.0146	0.0049	0.0038	0.0468
58 1989 Toyota Tercel (Carb)	29.700	3.200	1.000	0.700	0.600	12.100	0.22	0.1333	0.0144	0.0045	0.0031	0.0027	0.0543
59 1982 Buick Regal (Carb)	87.300	16.900	8.400	3.900	2.000	79.200	0.98	0.0893	0.0173	0.0086	0.0040	0.0020	0.0811
Average	82.433	13.677	6.081	2.578	2.321	40.612	0.72	0.1134	0.0188	0.0089	0.0040	0.0029	0.0542
Arco 91-06 (Ind. Avg. Baseline)													
350 1990 Ford Taurus (PFI)	55.520	6.740	1.100	1.030	2.510	24.200	0.43	0.1306	0.0158	0.0026	0.0024	0.0059	0.0569
351 1990 Toyota Camry (PFI)	24.190	4.330	0.510	0.470	1.750	9.270	0.21	0.1138	0.0204	0.0024	0.0022	0.0082	0.0436
352 1990 Plymouth Sundance (TBI)	31.790	4.920	0.540	0.350	1.340	17.130	0.21	0.1495	0.0231	0.0025	0.0016	0.0063	0.0806
353 1990 Honda Accord (PFI)	46.220	6.250	0.870	0.890	2.640	15.290	0.27	0.1691	0.0229	0.0032	0.0033	0.0097	0.0559
354 1990 Nissan Stanza (PFI)	48.370	9.690	1.320	0.840	3.330	26.790	0.36	0.1327	0.0266	0.0036	0.0023	0.0091	0.0735
355 1990 Pontiac Grand Am (PFI)	39.190	8.190	1.610	0.860	3.350	19.480	0.33	0.1173	0.0245	0.0048	0.0026	0.0100	0.0583
356 1990 Ford Crown Victoria (PFI)	54.220	7.950	1.260	0.700	0.920	13.550	0.35	0.1530	0.0224	0.0036	0.0020	0.0026	0.0382
357 1990 Plymouth Voyager (PFI)	61.500	10.830	3.280	1.880	3.320	39.030	0.62	0.0996	0.0175	0.0053	0.0030	0.0054	0.0632
358 1990 Nissan Pickup (PFI)	27.100	6.350	2.280	1.360	3.170	19.200	0.35	0.0765	0.0179	0.0064	0.0038	0.0089	0.0542
359 1990 Buick LeSabre (PFI)	40.260	6.510	1.010	0.570	1.460	15.590	0.28	0.1420	0.0230	0.0036	0.0020	0.0051	0.0550
Average	42.836	7.176	1.378	0.895	2.379	19.953	0.34	0.1284	0.0214	0.0038	0.0025	0.0071	0.0579
Auto/Oil Study*													
1989 Plymouth Sundance (TBI)	24.692	4.294	1.697	1.365	0.461	9.040	0.12	0.2074	0.0361	0.0143	0.0115	0.0039	0.0759
1989 Dodge Shadow (PFI)	26.489	2.853	1.810	1.390	0.639	7.359	0.13	0.2009	0.0216	0.0137	0.0105	0.0048	0.0558
1989 Ford Taurus (PFI)	28.179	2.931	1.033	0.528	0.602	9.189	0.17	0.1705	0.0177	0.0063	0.0032	0.0036	0.0556
1989 Ford Aerostar (PFI)	60.646	9.694	0.605	0.720	0.656	18.875	0.30	0.1989	0.0318	0.0020	0.0024	0.0022	0.0619
1989 Toyota Camry (PFI)	17.102	3.176	0.778	0.829	0.696	7.974	0.15	0.1153	0.0214	0.0052	0.0056	0.0047	0.0538
1989 Honda Accord (Carb)	39.561	2.854	0.912	1.249	1.214	6.693	0.18	0.2214	0.0160	0.0051	0.0070	0.0068	0.0375
1989 Chevrolet Suburban (TBI)	51.843	12.142	4.240	2.462	1.913	29.348	0.46	0.1136	0.0266	0.0093	0.0054	0.0042	0.0643
1989 Pontiac Grand Am (PFI)	22.918	3.437	1.962	1.133	1.268	6.963	0.17	0.1377	0.0207	0.0118	0.0068	0.0076	0.0418
1989 Oldsmobile Delta 88 (SFI)	22.169	1.651	1.146	0.762	0.528	7.965	0.12	0.1879	0.0140	0.0097	0.0065	0.0045	0.0675
Average	32.622	4.781	1.576	1.160	0.886	11.490	0.20	0.1726	0.0229	0.0086	0.0065	0.0047	0.0571
AVERAGE (All 38 vehicles)**	55.729	9.146	3.781	1.799	1.973	25.744	0.46	0.1324	0.0207	0.0087	0.0047	0.0048	0.0556
CARB Butadiene Study (55 vehicles)												0.0060	
											Average	0.0055	

5.5% MTBE (EC-1)

Arco 91-02 All FI

21 1989 Toyota Camry	26.550	5.830	1.520	1.000	0.960	5.880	0.20	0.1335	0.0293	0.0076	0.0050	0.0048	0.0296
22 1989 Pont. Grand Prix	57.820	14.050	7.880	3.400	1.550	10.470	0.26	0.2223	0.0540	0.0303	0.0131	0.0060	0.0403
23 1989 Ford Taurus	43.780	5.360	3.780	1.950	1.380	9.880	0.28	0.1561	0.0191	0.0135	0.0070	0.0049	0.0352
24 1989 Dodge Dynasty	60.730	13.240	6.290	2.960	2.960	3.530	12.380	0.54	0.1134	0.0247	0.0117	0.0055	0.0066	0.0231
25 1989 Nissan Maxima	32.210	6.440	6.230	2.930	2.250	4.790	0.27	0.1192	0.0238	0.0231	0.0108	0.0083	0.0177
AVERAGE**	44.218	8.984	5.140	2.448	1.934	8.680	0.31	0.1489	0.0302	0.0172	0.0083	0.0061	0.0292

9.0% MTBE

Arco 91-03

35 1988 Pontiac Grand Am (PFI)	26.600	4.100	1.500	1.400	0.900	6.500	0.14	0.1855	0.0286	0.0105	0.0098	0.0063	0.0453
35 1988 Pontiac Grand Am (PFI)	21.850	5.750	4.550	1.800	1.250	6.950	0.15	0.1471	0.0387	0.0306	0.0121	0.0084	0.0468
37 1981 Olsmobile Delta 88 (Carb)	216.750	31.700	24.210	9.280	11.840	44.220	1.34	0.1613	0.0236	0.0180	0.0069	0.0088	0.0329
37 1981 Olsmobile Delta 88 (Carb)	303.870	37.070	32.860	10.850	17.680	64.290	1.98	0.1533	0.0187	0.0166	0.0055	0.0089	0.0324
39 1984 Honda Accord (Carb)	90.720	9.190	1.410	1.080	2.010	14.010	0.49	0.1845	0.0187	0.0029	0.0022	0.0041	0.0285
39 1984 Honda Accord (Carb)	83.530	9.410	2.190	1.130	2.260	13.490	0.49	0.1717	0.0193	0.0045	0.0023	0.0046	0.0277
40 1987 Nissan 300ZX (PFI)	66.450	17.240	8.500	2.190	3.100	13.790	0.40	0.1642	0.0426	0.0210	0.0054	0.0077	0.0341
40 1987 Nissan 300ZX (PFI)	67.560	12.920	3.160	1.030	4.030	13.640	0.42	0.1608	0.0308	0.0075	0.0025	0.0096	0.0325
41 1988 Toyota 4WD Pickup (PFI)	68.590	8.570	1.530	0.890	1.960	20.420	0.47	0.1471	0.0184	0.0033	0.0019	0.0042	0.0438
41 1988 Toyota 4WD Pickup (PFI)	52.070	8.070	2.380	0.640	1.710	12.060	0.35	0.1473	0.0228	0.0067	0.0018	0.0048	0.0341
42 1988 Toyota Celica (PFI)	45.720	7.410	2.600	0.620	1.130	10.470	0.29	0.1566	0.0254	0.0089	0.0021	0.0039	0.0359
42 1988 Toyota Celica (PFI)	46.660	7.690	1.070	0.580	1.060	9.620	0.31	0.1503	0.0248	0.0034	0.0019	0.0034	0.0310
44 1986 Mercedes 190E (PFI)	95.980	25.360	3.340	2.070	4.710	38.170	0.87	0.1102	0.0291	0.0038	0.0024	0.0054	0.0438
44 1986 Mercedes 190E (PFI)	86.390	24.460	2.920	1.440	4.340	31.570	0.79	0.1095	0.0310	0.0037	0.0018	0.0055	0.0400
45 1989 Lincoln Continental (PFI)	72.610	13.520	1.670	0.970	1.780	22.060	0.50	0.1461	0.0272	0.0034	0.0020	0.0036	0.0444
45 1989 Lincoln Continental (PFI)	57.060	11.090	0.860	0.610	1.660	15.330	0.35	0.1614	0.0314	0.0024	0.0017	0.0047	0.0434
46 1990 Plymouth Acclaim (PFI)	64.470	13.820	2.450	1.370	3.860	25.280	0.67	0.0961	0.0206	0.0037	0.0020	0.0058	0.0377
46 1990 Plymouth Acclaim (PFI)	53.880	12.990	2.670	1.010	3.570	18.440	0.49	0.1107	0.0267	0.0055	0.0021	0.0073	0.0379
47 1984 Toyota Celica (PFI)	103.800	13.000	4.000	2.200	2.100	51.600	0.81	0.1275	0.0160	0.0049	0.0027	0.0026	0.0634
47 1984 Toyota Celica (PFI)	108.650	15.150	3.800	2.050	1.600	42.600	0.80	0.1351	0.0188	0.0047	0.0025	0.0020	0.0530
48 1985 Ford Mustang (Carb)	96.350	24.500	17.450	6.700	2.700	17.000	0.65	0.1479	0.0376	0.0268	0.0103	0.0041	0.0261
48 1985 Ford Mustang (Carb)	74.300	25.250	13.950	5.200	1.950	24.750	0.72	0.1036	0.0352	0.0195	0.0073	0.0027	0.0345
49 1987 Oldsmobile Regency (PFI)	63.800	11.250	1.850	1.250	1.750	34.700	0.62	0.1024	0.0181	0.0030	0.0020	0.0028	0.0557
56 1982 Nissan Maxima (PFI)	55.000	7.000	9.200	4.100	1.800	60.200	1.01	0.0545	0.0069	0.0091	0.0041	0.0018	0.0597
57 1986 Buick Park Avenue (PFI)	32.900	2.900	2.600	0.900	1.000	8.600	0.26	0.1259	0.0111	0.0100	0.0034	0.0038	0.0329
57 1986 Buick Park Avenue (PFI)	46.550	6.850	4.400	1.450	1.200	8.750	0.25	0.1855	0.0273	0.0175	0.0058	0.0048	0.0349
58 1989 Toyota Tercel (Carb)	32.900	2.900	2.600	0.900	1.000	8.600	0.20	0.1647	0.0145	0.0130	0.0045	0.0050	0.0431
58 1989 Toyota Tercel (Carb)	32.550	3.950	1.100	0.800	0.850	9.650	0.18	0.1816	0.0220	0.0061	0.0045	0.0047	0.0538
59 1982 Buick Regal (Carb)	89.900	20.600	10.900	2.900	3.100	63.600	0.82	0.1097	0.0251	0.0133	0.0035	0.0038	0.0776
AVERAGE**	77.843	13.576	5.921	2.324	3.031	24.495	0.58	0.1415	0.0245	0.0098	0.0040	0.0050	0.0416

12.5% MTBE

Arco 91-03

35 1988 Pontiac Grand Am (PFI)	38.350	5.800	4.750	1.850	1.100	7.150	0.15	0.2570	0.0389	0.0318	0.0124	0.0074	0.0479
37 1981 Olsmobile Delta 88 (Carb)	314.750	38.790	27.280	8.660	14.980	53.670	2.01	0.1568	0.0193	0.0136	0.0043	0.0075	0.0267
39 1984 Honda Accord (Carb)	89.680	10.710	3.410	1.280	2.140	11.290	0.48	0.1874	0.0224	0.0071	0.0027	0.0045	0.0236
40 1987 Nissan 300ZX (PFI)	68.710	12.900	3.520	1.020	3.960	9.680	0.38	0.1829	0.0343	0.0094	0.0027	0.0105	0.0258
41 1988 Toyota 4WD Pickup (PFI)	56.350	8.210	1.580	0.680	1.850	8.980	0.33	0.1685	0.0245	0.0047	0.0020	0.0055	0.0268
42 1988 Toyota Celica (PFI)	42.830	7.260	1.810	0.620	1.010	8.430	0.30	0.1435	0.0243	0.0061	0.0021	0.0034	0.0282
44 1986 Mercedes 190E (PFI)	91.630	25.590	3.480	1.450	3.710	23.390	0.71	0.1296	0.0362	0.0049	0.0021	0.0052	0.0331
45 1989 Lincoln Continental (PFI)	68.120	10.770	1.070	0.670	1.570	12.230	0.38	0.1789	0.0283	0.0028	0.0018	0.0041	0.0321
46 1990 Plymouth Acclaim (PFI)	48.100	13.030	2.270	1.030	3.300	13.310	0.43	0.1113	0.0301	0.0053	0.0024	0.0076	0.0308
47 1984 Toyota Celica (PFI)	94.600	12.950	3.200	1.850	2.900	24.600	0.64	0.1483	0.0203	0.0050	0.0029	0.0045	0.0386
48 1985 Ford Mustang (Carb)	77.200	27.750	14.600	5.550	2.050	14.950	0.68	0.1137	0.0409	0.0215	0.0082	0.0030	0.0220
57 1986 Buick Park Avenue (PFI)	59.550	6.000	4.150	1.500	1.400	8.350	0.23	0.2572	0.0259	0.0179	0.0065	0.0060	0.0361
58 1989 Toyota Tercel (Carb)	30.350	3.300	1.450	0.850	0.900	6.200	0.15	0.1966	0.0214	0.0094	0.0055	0.0058	0.0402

AVERAGE**	83.094	14.082	5.582	2.078	3.144	15.556	0.53	0.1717	0.0282	0.0107	0.0043	0.0058	0.0317

15.0% MTBE

Arco 91-06 (Test Blend 1)

350 1990 Ford Taurus (PFI)	59.730	6.930	1.550	0.840	1.810	10.820	0.31	0.1928	0.0224	0.0050	0.0027	0.0058	0.0349
351 1990 Toyota Camry (PFI)	23.290	2.950	0.510	0.380	1.240	5.510	0.17	0.1410	0.0179	0.0031	0.0023	0.0075	0.0334
352 1990 Plymouth Sundance (TBI)	26.090	3.240	1.040	0.160	0.520	4.840	0.13	0.1944	0.0241	0.0077	0.0012	0.0039	0.0361
353 1990 Honda Accord (PFI)	49.430	6.330	1.660	0.490	1.900	7.300	0.20	0.2520	0.0323	0.0085	0.0025	0.0097	0.0372
354 1990 Nissan Stanza (PFI)	52.280	5.930	1.440	0.620	1.870	10.640	0.25	0.2110	0.0239	0.0058	0.0025	0.0075	0.0429
355 1990 Pontiac Grand Am (PFI)	36.120	6.250	1.720	0.770	2.420	8.670	0.24	0.1521	0.0263	0.0072	0.0032	0.0102	0.0365
356 1990 Ford Crown Victoria (PFI)	54.030	7.740	1.120	0.590	0.770	9.040	0.23	0.2379	0.0341	0.0049	0.0026	0.0034	0.0398
357 1990 Plymouth Voyager (PFI)	51.000	7.170	1.740	2.410	2.040	8.330	0.35	0.1453	0.0204	0.0050	0.0069	0.0058	0.0237
358 1990 Nissan Pickup (PFI)	24.770	3.720	3.330	1.080	2.150	10.640	0.28	0.0889	0.0133	0.0119	0.0039	0.0077	0.0382
359 1990 Buick LeSabre (PFI)	28.660	3.240	1.390	0.460	1.130	8.740	0.19	0.1542	0.0174	0.0075	0.0025	0.0061	0.0470
Average	40.540	5.350	1.550	0.780	1.585	8.453	0.23	0.1770	0.0232	0.0067	0.0030	0.0068	0.0370

Arco 91-06 (Test Blend 2)

350 1990 Ford Taurus (PFI)	54.010	4.420	1.660	0.800	1.780	11.970	0.30	0.1804	0.0148	0.0055	0.0027	0.0059	0.0400
351 1990 Toyota Camry (PFI)	24.910	2.910	0.630	0.440	1.020	10.170	0.18	0.1419	0.0166	0.0036	0.0025	0.0058	0.0579
352 1990 Plymouth Sundance (TBI)	30.850	3.570	0.900	0.370	0.580	5.970	0.13	0.2298	0.0266	0.0067	0.0028	0.0043	0.0445
353 1990 Honda Accord (PFI)	52.300	5.020	1.400	0.470	1.450	7.800	0.20	0.2666	0.0256	0.0071	0.0024	0.0074	0.0398
354 1990 Nissan Stanza (PFI)	59.300	8.360	1.450	1.450	2.170	11.030	0.30	0.1981	0.0279	0.0048	0.0048	0.0072	0.0368
355 1990 Pontiac Grand Am (PFI)	39.740	6.230	1.690	0.530	2.510	9.850	0.25	0.1604	0.0251	0.0068	0.0021	0.0101	0.0398
356 1990 Ford Crown Victoria (PFI)	67.940	5.930	0.980	0.310	0.780	7.280	0.25	0.2742	0.0239	0.0040	0.0013	0.0031	0.0294
357 1990 Plymouth Voyager (PFI)	52.900	8.410	2.670	1.030	2.390	14.020	0.38	0.1385	0.0220	0.0070	0.0027	0.0063	0.0367
358 1990 Nissan Pickup (PFI)	25.120	3.780	3.050	1.020	2.460	11.650	0.27	0.0936	0.0141	0.0114	0.0038	0.0092	0.0434
359 1990 Buick LeSabre (PFI)	24.820	3.710	1.170	0.490	0.800	12.400	0.15	0.1603	0.0240	0.0076	0.0032	0.0052	0.0801
Average	43.189	5.234	1.560	0.691	1.594	10.214	0.24	0.1844	0.0221	0.0065	0.0028	0.0065	0.0448

Auto/Oil Study

1989 Plymouth Sundance (TBI)	20.860	2.870	0.800	0.440	0.450	7.880	0.12	0.1684	0.0232	0.0065	0.0036	0.0036	0.0636
1989 Dodge Shadow (PFI)	25.900	2.618	0.924	0.630	0.980	8.918	0.18	0.1476	0.0149	0.0053	0.0036	0.0056	0.0508
1989 Ford Taurus (PFI)	28.713	3.468	1.190	0.731	0.782	10.438	0.21	0.1391	0.0168	0.0058	0.0035	0.0038	0.0505
1989 Ford Aerostar (PFI)	55.356	8.456	1.344	0.980	1.204	19.040	0.33	0.1676	0.0256	0.0041	0.0030	0.0036	0.0576
1989 Toyota Camry (PFI)	17.199	3.159	1.677	0.455	0.637	7.930	0.18	0.0980	0.0180	0.0096	0.0026	0.0036	0.0452
1989 Honda Accord (Carb)	39.480	2.296	1.610	0.798	0.840	5.292	0.17	0.2390	0.0139	0.0097	0.0048	0.0051	0.0320
1989 Chevrolet Suburban (TBI)	56.870	11.891	5.687	2.820	1.833	34.357	0.52	0.1102	0.0230	0.0110	0.0055	0.0036	0.0666
1989 Pontiac Grand Am (PFI)	21.045	3.045	1.995	1.080	1.425	7.620	0.18	0.1199	0.0173	0.0114	0.0062	0.0081	0.0434
1989 Oldsmobile Delta 88 (SFI)	21.024	2.496	1.908	0.660	0.792	8.052	0.14	0.1455	0.0173	0.0132	0.0046	0.0055	0.0557
Average	31.827	4.478	1.904	0.955	0.994	12.170	0.22	0.1483	0.0189	0.0085	0.0041	0.0047	0.0517
AVERAGE (All vehicles)**	38.750	5.039	1.663	0.804	1.405	10.214	0.23	0.1706	0.0215	0.0072	0.0033	0.0060	0.0443

*Only data for fuel A considered.
**Data used in subsequent emission fractions calculations

09/15/1987

LDGV (Carb -- FTP Data)

	Diurnal Evap (mg/test)			Fraction of THC		Hot Soak Evap (mg/test)			Fraction of THC	
	MTBE*	Benzene	THC (g/test)	MTBE	Benzene	MTBE*	Benzene	THC (g/test)	MTBE	Benzene
0% MTBE										
Arco 91-03 (Unleaded Premium)										
37 1981 Olsmobile Delta 88 (TWC)	25.070	94.370	12.427	0.0020	0.0076	0.000	167.210	5.970	0.0000	0.0280
39 1984 Honda Accord (TWC)	23.450	19.580	0.516	0.0455	0.0380	20.680	48.910	0.817	0.0253	0.0599
48 1985 Ford Mustang (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
58 1989 Toyota Tercel (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
59 1982 Buick Regal (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
43 1985 Chevrolet Camaro (TWC + OX)	201.750	47.710	3.133	0.0644	0.0152	436.560	121.150	10.321	0.0423	0.0117
Average	41.712	26.943	2.679	0.0186	0.0101	76.207	56.212	2.851	0.0113	0.0166
Auto/Oil Study										
1989 Honda Accord (Carb)**	0.000	13.320	1.470	0.0000	0.0091	0.000	34.850	1.560	0.0000	0.0223
1985 Plymouth Reliant (TWC)	1.232	10.010	0.770	0.0016	0.0130	7.224	33.927	1.290	0.0056	0.0263
1985 Honda Accord (TWC)	1.188	8.856	0.270	0.0044	0.0328	1.975	22.436	0.790	0.0025	0.0284
1985 Chevrolet Impala (TWC + OX)	1.610	11.109	1.610	0.0010	0.0069	4.263	25.491	0.870	0.0049	0.0293
1984 Pontiac Grand Prix (TWC + OX)	6.540	8.938	1.090	0.0060	0.0082	5.688	42.660	1.580	0.0036	0.0270
1984 Chevrolet Suburban (OX)	1.440	14.220	1.800	0.0008	0.0079	5.922	25.756	0.940	0.0063	0.0274
1983 Ford F-150 (OX)	4.275	21.150	0.750	0.0057	0.0282	10.416	39.556	1.240	0.0084	0.0319
Average	2.326	12.515	1.109	0.0028	0.0152	5.070	32.097	1.181	0.0045	0.0275
Stump et al., unpublished (MSERB High Temp. Study)										
1986 Chev Caprice (TWC + OX)	0.000	5.700	0.756	0.0000	0.0075	0.000	19.100	1.772	0.0000	0.0108
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)										
1977 Mercury Marquis (OX)	41.000	27.670	5.300	0.0077	0.0052	27.170	87.670	14.000	0.0019	0.0063
1974 Chevy Impala (Non-cat)	0.330	31.330	3.330	0.0001	0.0094	3.000	47.000	2.620	0.0011	0.0179
Average	20.665	29.500	4.315	0.0039	0.0073	15.085	67.335	8.310	0.0015	0.0121
AVERAGE (All vehicles)***	20.526	20.043	2.117	0.0093	0.0120	34.860	45.391	2.814	0.0068	0.0203
9.0% MTBE										
Arco 91-03										
37 1981 Olsmobile Delta 88 (TWC)	529.930	50.480	7.109	0.0745	0.0071	577.410	59.490	3.358	0.1720	0.0177
37 1981 Olsmobile Delta 88 (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
39 1984 Honda Accord (TWC)	48.190	13.190	0.450	0.1072	0.0293	79.920	27.880	0.749	0.1068	0.0372
39 1984 Honda Accord (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
48 1985 Ford Mustang (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
48 1985 Ford Mustang (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
58 1989 Toyota Tercel (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
58 1989 Toyota Tercel (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
59 1982 Buick Regal (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
43 1985 Chevrolet Camaro (TWC + OX)	226.500	44.950	1.987	0.1140	0.0226	573.120	112.450	6.779	0.0845	0.0166
Average***	80.462	10.862	0.955	0.0296	0.0059	123.045	19.982	1.089	0.0363	0.0072

15.0% MTBE

Auto/Oil Study

1989 Honda Accord (TWC)	122.130	13.570	1.150	0.1062	0.0118	288.288	32.592	1.680	0.1716	0.0194
1985 Plymouth Reliant (TWC)	115.368	11.088	0.880	0.1311	0.0126	408.360	56.334	2.460	0.1660	0.0229
1985 Honda Accord (TWC)	30.591	8.910	0.330	0.0927	0.0270	169.983	24.442	1.010	0.1683	0.0242
1985 Chevrolet Impala (TWC + OX)	120.897	9.918	1.710	0.0707	0.0058	143.152	26.588	0.920	0.1556	0.0289
1984 Pontiac Grand Prix (TWC + OX)	116.035	11.155	1.150	0.1009	0.0097	132.388	25.208	0.920	0.1439	0.0274
1984 Chevrolet Suburban (OX)	242.109	15.876	1.890	0.1281	0.0084	116.964	28.188	1.080	0.1083	0.0261
1983 Ford F-150 (OX)	106.400	19.040	0.800	0.1330	0.0238	178.976	37.128	1.360	0.1316	0.0273
Average***	121.933	12.794	1.130	0.1090	0.0142	205.444	32.926	1.347	0.1493	0.0252

16.4% MTBE

Warner-Selph and Smith, 1991 (EPA-460/3-91-02)

1977 Mercury Marquis (OX)	228.000	18.000	3.600	0.0633	0.0050	1762.670	136.330	15.800	0.1116	0.0086
1974 Chevy Impala (Non-cat)	240.000	22.330	2.400	0.1000	0.0093	537.330	49.000	3.770	0.1425	0.0130
Average***	234.000	20.165	3.000	0.0817	0.0072	1150.000	92.665	9.785	0.1270	0.0108

*Evaporative MTBE emissions in fuels with 0% MTBE is likely due to carryover from tests done on MTBE blends.
**Only data for fuel A considered.
***Data used in subsequent emission fractions calculations.

01/28/1988

| | DIURNAL | | | | | HOT SOAK | | | | |
| | Evap (mg/test) | | THC (g/test) | Fraction of THC | | Evap (mg/test) | | THC (g/test) | Fraction of THC | |
	Ethanol	Benzene		Ethanol	Benzene	Ethanol	Benzene		Ethanol	Benzene
LDGV -- CARB										
E0										
Auto/Oil (Fuel A/Industry Avg.)										
1989 Accord Sedan (TWC)	N.A.	9.31	0.95		0.0098	0.00	25.66	0.91	0.0000	0.0282
E10										
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)										
1983 Buick Regal (TWC)	65.00	12.00	1.29	0.0504	0.0093	234.67	39.67	1.55	0.1514	0.0256
1977 Mercury Marquis (Oxy)	246.67	37.33	5.80	0.0425	0.0064	1230.00	86.33	20.70	0.0594	0.0042
1974 Chevy Impala (Noncat)	106.00	29.00	2.44	0.0434	0.0119	173.67	44.00	3.61	0.0481	0.0122
Average	139.22	26.11	3.18	0.0455	0.0092	546.11	56.67	8.62	0.0863	0.0140
Auto/Oil (Fuel X/Industry Avg. + 10% EtOH, 10 RVP)										
1989 Accord Sedan (TWC)	143.22	11.11	1.10	0.1302	0.0101	303.03	40.53	2.10	0.1443	0.0193
Auto/Oil (Fuel W/Industry Avg. + 10% EtOH, 9 RVP)										
1989 Accord Sedan (TWC)	150.18	12.58	1.04	0.1444	0.0121	281.39	39.95	1.93	0.1458	0.0207
Auto/Oil (Fuel U/low ole., low T90, low arom., 10% EtOH, 10 RVP)										
1989 Accord Sedan (TWC)	24.10	11.64	1.03	0.0234	0.0113	294.12	38.02	1.97	0.1493	0.0193
Auto/Oil (Fuel T/low ole., low T90, low arom., 10% EtOH, 9 RVP)										
1989 Accord Sedan (TWC)	0.00	11.40	0.82	0.0000	0.0139	108.16	40.80	1.60	0.0676	0.0255
Average*	105.02	17.87	1.93	0.0621	0.0107	375.01	47.04	4.78	0.1094	0.0181
LDGV -- TBI										
E0										
Auto/Oil (Fuel A/Industry Avg.)										
1989 Plymouth Sundance (TWC)	N.A.	5.68	0.66		0.0086	N.A.	5.72	0.10		0.0572
1989 Dodge Shadow (TWC)	N.A.	7.52	0.66		0.0114	N.A.	7.75	0.17		0.0456
1989 Chevy Suburban (TWC)	N.A.	9.88	0.40		0.0247	N.A.	16.34	0.36		0.0454
Average*		7.69	0.57		0.0149		9.94	0.21		0.0494
8.1% EtOH										
Stump et al., 1990 (MSERB Oxyfuels Study)										
CO665 1988 Chevy Corsica (TWC)	2.70	3.40	0.50	0.0054	0.0068	0.00	6.70	0.21	0.0000	0.0319
CO322 1987 Chevy Corsica (TWC)	10.30	4.70	0.46	0.0224	0.0102	12.40	4.70	0.17	0.0729	0.0276
Average*	6.50	4.05	0.48	0.0139	0.0085	6.20	5.70	0.19	0.0365	0.0298

| | Evap (mg/test) | | THC (g/test) | Fraction of THC | | Evap (mg/test) | | THC (g/test) | Fraction of THC | |
	Ethanol	Benzene		Ethanol	Benzene	Ethanol	Benzene		Ethanol	Benzene
E10										
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)										
1990 Chevy Lumina (TWC)	20.00	3.67	0.38	0.0526	0.0097	20.33	10.67	0.29	0.0701	0.0368
Auto/Oil (Fuel X/Industry Avg. + 10% EtOH, 10 RVP)										
1989 Plymouth Sundance (TWC)	70.53	10.86	0.77	0.0916	0.0141	70.62	11.46	0.30	0.2354	0.0382
1989 Dodge Shadow (TWC)	13.44	2.10	0.35	0.0384	0.0060	38.26	5.48	0.20	0.1913	0.0274
1989 Chevy Suburban (TWC)	59.89	15.28	0.59	0.1015	0.0259	68.81	16.02	0.45	0.1529	0.0356
Average	47.95	9.41	0.57	0.0772	0.0153	59.23	10.99	0.32	0.1932	0.0337
Auto/Oil (Fuel W/Industry Avg. + 10% EtOH, 9 RVP)										
1989 Plymouth Sundance (TWC)	49.08	10.99	0.41	0.1197	0.0268	75.42	11.33	0.32	0.2357	0.0354
1989 Dodge Shadow (TWC)	7.38	2.65	0.18	0.0410	0.0147	9.41	5.52	0.14	0.0672	0.0394
1989 Chevy Suburban (TWC)	44.62	16.68	0.67	0.0666	0.0249	60.14	15.55	0.48	0.1253	0.0324
Average	33.69	10.11	0.42	0.0758	0.0221	48.33	10.80	0.31	0.1427	0.0357
Auto/Oil (Fuel U/low ole., low T90, low arom., 10% EtOH, 10 RVP)										
1989 Plymouth Sundance (TWC)	37.03	5.30	0.22	0.1683	0.0241	44.77	9.26	0.26	0.1722	0.0356
1989 Dodge Shadow (TWC)	8.88	1.63	0.25	0.0355	0.0065	45.25	6.93	0.18	0.2514	0.0385
1989 Chevy Suburban (TWC)	31.35	10.78	0.55	0.0570	0.0196	72.48	22.75	0.62	0.1169	0.0367
Average	25.75	5.90	0.34	0.0869	0.0167	54.17	12.98	0.35	0.1802	0.0369
Auto/Oil (Fuel T/low ole., low T90, low arom., 10% EtOH, 9 RVP)										
1989 Plymouth Sundance (TWC)	24.89	8.57	0.51	0.0488	0.0168	37.70	10.58	0.24	0.1571	0.0441
1989 Dodge Shadow (TWC)	10.04	1.65	0.15	0.0669	0.0110	25.09	5.40	0.13	0.1930	0.0415
1989 Chevy Suburban (TWC)	23.30	8.47	0.24	0.0971	0.0353	32.19	17.92	0.41	0.0785	0.0437
Average	19.41	6.23	0.30	0.0709	0.0210	31.66	11.30	0.26	0.1429	0.0431
Average (All E10)*	30.80	7.59	0.41	0.0758	0.0181	46.19	11.45	0.31	0.1575	0.0373
LDGV -- PFI										
E0										
Auto/Oil (Fuel A/Industry Avg.)										
1989 Ford Mustang (TWC)	N.A.	3.92	0.53		0.0074	N.A.	3.46	0.05		0.0692
1989 Ford Taurus (TWC)	N.A.	5.93	0.15		0.0395	N.A.	10.01	0.19		0.0527
1989 Ford Aerostar (TWC)	N.A.	14.04	0.35		0.0401	N.A.	26.11	0.68		0.0384
1989 Camry Sedan (TWC)	N.A.	3.12	0.24		0.0130	N.A.	12.83	0.27		0.0475
1989 Pontiac Grand Am (TWC)	N.A.	7.28	0.28		0.0260	N.A.	17.36	0.33		0.0526
1989 Oldsmobile Delta 88 (TWC)	N.A.	8.82	0.45		0.0196	N.A.	11.48	0.28		0.0410
Average*		7.18	0.33		0.0243		13.54	0.30		0.0502

| | Evap (mg/test) | | THC (g/test) | Fraction of THC | | Evap (mg/test) | | THC (g/test) | Fraction of THC | |
	Ethanol	Benzene		Ethanol	Benzene	Ethanol	Benzene		Ethanol	Benzene

E10

Warner-Selph and Smith, 1991 (EPA-460/3-91-02)

1990 Ford Probe (TWC)	19.50	5.50	0.12	0.1625	0.0458	92.50	12.50	0.30	0.3083	0.0417

Auto/Oil (Fuel X/Industry Avg. + 10% EtOH, 10 RVP)

1989 Ford Mustang (TWC)	13.71	3.90	0.30	0.0457	0.0130	19.02	5.40	0.11	0.1729	0.0491
1989 Ford Taurus (TWC)	59.55	6.34	0.16	0.3722	0.0396	211.15	17.38	0.55	0.3839	0.0316
1989 Ford Aerostar (TWC)	60.53	20.24	0.57	0.1062	0.0355	171.91	37.51	1.14	0.1508	0.0329
1989 Camry Sedan (TWC)	37.03	3.80	0.25	0.1481	0.0152	57.39	10.47	0.29	0.1979	0.0361
1989 Pontiac Grand Am (TWC)	41.18	9.14	0.36	0.1144	0.0254	68.17	16.81	0.39	0.1748	0.0431
1989 Oldsmobile Delta 88 (TWC)	18.76	6.56	0.29	0.0647	0.0226	22.30	9.89	0.32	0.0697	0.0309
Average	38.46	8.33	0.32	0.1419	0.0252	91.66	16.24	0.47	0.1917	0.0373

Auto/Oil (Fuel W/Industry Avg. + 10% EtOH, 9 RVP)

1989 Ford Mustang (TWC)	0.00	2.63	0.09	0.0000	0.0292	43.18	7.51	0.17	0.2540	0.0442
1989 Ford Taurus (TWC)	15.56	5.41	0.13	0.1197	0.0416	179.82	15.70	0.47	0.3826	0.0334
1989 Ford Aerostar (TWC)	45.50	17.91	0.45	0.1011	0.0398	129.64	41.82	1.02	0.1271	0.0410
1989 Camry Sedan (TWC)	17.45	4.64	0.13	0.1342	0.0357	54.50	0.00	0.35	0.1557	0.0000
1989 Pontiac Grand Am (TWC)	17.54	7.71	0.21	0.0835	0.0367	38.01	16.28	0.31	0.1226	0.0525
1989 Oldsmobile Delta 88 (TWC)	7.99	6.42	0.22	0.0363	0.0292	27.16	11.15	0.27	0.1006	0.0413
Average	17.34	7.45	0.21	0.0791	0.0354	78.72	15.41	0.43	0.1904	0.0354

Auto/Oil (Fuel U/low ole., low T90, low arom., 10% EtOH, 10 RVP)

1989 Ford Mustang (TWC)	16.64	3.58	0.20	0.0832	0.0179	42.91	6.52	0.14	0.3065	0.0466
1989 Ford Taurus (TWC)	66.28	7.29	0.21	0.3156	0.0347	199.75	18.59	0.56	0.3567	0.0332
1989 Ford Aerostar (TWC)	54.00	17.49	0.49	0.1102	0.0357	57.92	17.74	0.96	0.0603	0.0185
1989 Camry Sedan (TWC)	33.46	6.70	0.24	0.1394	0.0279	62.55	13.20	0.29	0.2157	0.0455
1989 Pontiac Grand Am (TWC)	32.58	15.37	0.32	0.1018	0.0480	62.37	20.35	0.47	0.1327	0.0433
1989 Oldsmobile Delta 88 (TWC)	18.49	6.89	0.26	0.0711	0.0265	40.90	13.79	0.32	0.1278	0.0431
Average	36.91	9.55	0.29	0.1369	0.0318	77.73	15.03	0.46	0.2000	0.0384

Auto/Oil (Fuel T/low ole., low T90, low arom., 10% EtOH, 9 RVP)

1989 Ford Mustang (TWC)	8.99	2.16	0.17	0.0529	0.0127	20.64	5.15	0.12	0.1720	0.0429
1989 Ford Taurus (TWC)	41.02	6.55	0.18	0.2279	0.0364	148.56	18.56	0.51	0.2913	0.0364
1989 Ford Aerostar (TWC)	49.06	14.13	0.37	0.1326	0.0382	141.70	39.85	1.08	0.1312	0.0369
1989 Camry Sedan (TWC)	13.82	4.40	0.15	0.0921	0.0293	41.30	9.44	0.28	0.1475	0.0337
1989 Pontiac Grand Am (TWC)	34.48	11.41	0.35	0.0985	0.0326	58.96	20.99	0.41	0.1438	0.0512
1989 Oldsmobile Delta 88 (TWC)	18.65	5.89	0.18	0.1036	0.0327	22.53	12.50	0.29	0.0777	0.0431
Average	27.67	7.42	0.23	0.1179	0.0303	72.28	17.75	0.45	0.1606	0.0407
Average (All E10)*	29.67	8.08	0.26	0.1207	0.0313	80.59	15.96	0.44	0.1906	0.0381

MTBE Blends -- Auto/Oil

Evap (mg/test)		THC (g/test)	Fraction of THC		Evap (mg/test)		THC (g/test)	Fraction of THC	
MTBE	Benzene		MTBE	Benzene	MTBE	Benzene		MTBE	Benzene

LDGV -- CARB

Auto/Oil (Fuel N2/low ole., low T90, low arom., 15% MTBE, 9 RVP)

1989 Accord Sedan (TWC)	62.43	7.70	0.55	0.1135	0.0140	299.92	26.90	1.63	0.1840	0.0165

Auto/Oil (Fuel MM/low ole., low T90, low arom., 15% MTBE, 8 RVP)

1989 Accord Sedan (TWC)	99.85	7.40	0.86	0.1161	0.0086	277.38	30.98	1.75	0.1585	0.0177
Average (All 15% MTBE)*	81.14	7.55	0.71	0.1151	0.0107	288.65	28.94	1.69	0.1708	0.0171

LDGV -- TBI

Auto/Oil (Fuel N2/low ole., low T90, low arom., 15% MTBE, 9 RVP)

1989 Plymouth Sundance (TWC)	0.00	3.20	0.25	0.0000	0.0128	0.00	6.66	0.18	0.0000	0.0370
1989 Dodge Shadow (TWC)	0.00	1.32	0.14	0.0000	0.0094	0.00	3.95	0.07	0.0000	0.0564
1989 Chevy Suburban (TWC)	0.00	12.65	0.55	0.0000	0.0230	46.54	14.68	0.41	0.1135	0.0358
Average	0.00	5.72	0.31	0.0000	0.0151	15.51	8.43	0.22	0.0378	0.0431

Auto/Oil (Fuel MM/low ole., low T90, low arom., 15% MTBE, 8 RVP)

1989 Plymouth Sundance (TWC)	5.12	5.18	0.33	0.0155	0.0157	22.84	8.07	0.23	0.0993	0.0351
1989 Dodge Shadow (TWC)	0.00	1.33	0.13	0.0000	0.0102	0.00	5.76	0.12	0.0000	0.0480
1989 Chevy Suburban (TWC)	12.65	8.81	0.37	0.0342	0.0238	59.51	18.18	0.57	0.1044	0.0319
Average	5.92	5.10	0.28	0.0166	0.0166	27.45	10.67	0.31	0.0679	0.0383
Average (All 15% MTBE)*	2.96	5.41	0.30	0.0083	0.0158	21.48	9.55	0.26	0.0529	0.0407

LDGV -- PFI

Auto/Oil (Fuel N2/low ole., low T90, low arom., 15% MTBE, 9 RVP)

1989 Ford Mustang (TWC)	0.00	0.75	0.08	0.0000	0.0094	2.72	2.67	0.07	0.0388	0.0382
1989 Ford Taurus (TWC)	0.00	1.96	0.06	0.0000	0.0327	6.89	5.57	0.15	0.0459	0.0371
1989 Ford Aerostar (TWC)	0.00	14.58	0.45	0.0000	0.0324	48.36	31.25	0.93	0.0520	0.0336
1989 Camry Sedan (TWC)	0.00	4.31	0.11	0.0000	0.0392	20.77	6.65	0.19	0.1093	0.0350
1989 Pontiac Grand Am (TWC)	34.08	6.18	0.22	0.1549	0.0281	62.66	13.64	0.38	0.1649	0.0359
1989 Oldsmobile Delta 88 (TWC)	10.73	4.56	0.13	0.0825	0.0351	21.23	5.95	0.17	0.1249	0.0350
Average	7.47	5.39	0.18	0.0396	0.0295	27.10	10.95	0.32	0.0893	0.0358

Auto/Oil (Fuel MM/low ole., low T90, low arom., 15% MTBE, 8 RVP)

1989 Ford Mustang (TWC)	31.23	2.40	0.16	0.1952	0.0150	0.00	4.70	0.07	0.0000	0.0672
1989 Ford Taurus (TWC)	8.10	3.60	0.10	0.0810	0.0360	18.60	6.43	0.17	0.1094	0.0378
1989 Ford Aerostar (TWC)	14.52	13.24	0.40	0.0363	0.0331	34.58	28.12	0.91	0.0380	0.0309
1989 Camry Sedan (TWC)	0.00	6.16	0.19	0.0000	0.0324	0.00	11.17	0.29	0.0000	0.0385
1989 Pontiac Grand Am (TWC)	9.70	4.79	0.14	0.0693	0.0342	0.00	11.09	0.22	0.0000	0.0504
1989 Oldsmobile Delta 88 (TWC)	0.00	5.80	0.23	0.0000	0.0252	31.69	15.23	0.46	0.0689	0.0331
Average	10.59	6.00	0.20	0.0636	0.0293	14.15	12.79	0.35	0.0361	0.0430
Average (All 15% MTBE)*	9.03	5.69	0.19	0.0516	0.0294	20.62	11.87	0.33	0.0627	0.0394

*Data used in subsequent emission fractions calculations

01/28/1987

LDGV (FTP Data) EtOH/Misc Blends

	Methane	Ethane	Formaldehyde	Exhaust (mg/mi) Acetaldehyde	1,3-Butadiene	Benzene	Methanol	Ethanol	MTBE	ETBE	TOG, g/mile
TWC											
M85											
AB234 Study											
1987 Ford Crown Victoria (FI)	44.000	1.169	22.366	1.395	N.A.	2.118	156.217	0.000	N.A.	N.A.	
MSERB FFV Study											
1988 Chevrolet Corsica (TBI)	19.760	0.610	27.470	0.470	0.330	2.640	290.000	0.000	N.A.	N.A.	
AVERAGE	31.880	0.890	24.918	0.933	0.165	2.379	223.109	0.000			
M100											
AB234 Study											
1987 Ford Crown Victoria (FI)	21.000	0.105	34.987	0.434	N.A.	0.520	504.287	0.000	N.A.	N.A.	
MSERB FFV Study											
1988 Chevrolet Corsica (TBI)	9.170	0.000	636.150	4.290	0.000	0.210	820.000	0.000	N.A.	N.A.	
AVERAGE	15.085	0.053	335.569	2.362	0.000	0.365	662.144	0.000			
E95											
AB234 Study											
1987 Ford Crown Victoria (FI)	195.000	8.222	12.264	60.971	N.A.	3.097	0.000	742.710	N.A.	N.A.	
E85											
AB234 Study											
1987 Ford Crown Victoria (TBI)	167.000	10.742	10.638	52.514	N.A.	3.424	0.000	783.371	N.A.	N.A.	
E0											
Auto/Oil (Fuel A/Industry Avg.)											
1989 Plymouth Sundance (TBI)	21.912	3.590	0.443	0.492	0.646	6.571	N.A.	N.A.	0.000	0.239	0.14
1989 Dodge Shadow (TBI)	26.424	3.262	0.927	0.366	1.024	8.911	N.A.	N.A.	0.000	0.179	0.17
1989 Ford Mustang (PFI)	66.792	13.003	1.244	0.942	0.690	7.414	N.A.	N.A.	1.564	0.289	0.33
1989 Ford Taurus (PFI)	25.379	3.203	0.577	1.322	0.912	8.900	N.A.	N.A.	0.000	0.354	0.19
1989 Ford Aerostar (PFI)	52.372	8.280	0.688	0.172	1.404	14.841	N.A.	N.A.	2.550	0.372	0.31
1989 Camry Sedan (PFI)	16.024	3.374	0.564	2.612	0.448	5.708	N.A.	N.A.	0.000	0.215	0.15
1989 Accord Sedan (Carb)	35.396	2.672	2.557	2.502	0.822	4.917	N.A.	N.A.	0.000	0.152	0.14
1989 Chevy Suburban (TBI)	59.359	13.842	3.661	3.087	2.194	32.781	N.A.	N.A.	0.310	1.038	0.52
1989 Pontiac Grand Am (PFI)	16.154	2.706	1.060	3.013	1.353	6.501	N.A.	N.A.	0.000	0.251	0.16
1989 Oldsmobile Delta 88 (PFI)	18.131	2.549	0.763	0.540	1.025	5.647	N.A.	N.A.	0.000	0.188	0.14
Average*	33.794	5.648	1.248	1.505	1.052	10.219			0.442	0.328	0.23
E10											
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)											
1990 Ford Probe (PFI)	N.A.	N.A.	3.800	2.200	0.800	8.000	N.A.	8.500	N.A.	N.A.	0.21
1990 Chevy Lumina (TBI)	N.A.	N.A.	4.100	2.600	0.800	6.100	N.A.	0.000	N.A.	N.A.	0.15
1983 Buick Regal (Carb)	N.A.	N.A.	5.900	7.000	1.100	21.200	N.A.	18.100	N.A.	N.A.	0.81
Average			4.600	3.933	0.900	11.767		8.867			0.39
Auto/Oil (Fuel X/Industry Avg. +10% EtOH, 10 RVP)											
1989 Plymouth Sundance (TBI)	23.904	3.921	0.832	1.352	0.586	6.330	N.A.	3.217	0.000	0.239	0.13
1989 Dodge Shadow (TBI)	26.078	2.853	9.163	1.718	1.000	7.320	N.A.	3.135	0.000	0.167	0.17

1989 Ford Mustang (PFI)	64.576	12.983	1.782	1.879	0.444	4.204	N.A.	3.240	0.000	0.000	0.28
1989 Ford Taurus (PFI)	27.260	3.451	0.901	1.916	0.761	7.627	N.A.	4.734	0.000	0.308	0.20
1989 Ford Aerostar (PFI)	53.185	7.810	0.845	2.583	1.355	13.589	N.A.	7.371	0.000	0.368	0.26
1989 Camry Sedan (PFI)	16.767	4.080	0.202	0.941	0.523	5.978	N.A.	2.507	0.000	0.248	0.16
1989 Accord Sedan (Carb)	38.525	4.107	2.979	1.688	0.713	3.720	N.A.	2.327	0.000	0.190	0.14
1989 Chevy Suburban (TBI)	63.135	14.147	4.889	8.614	3.282	28.014	N.A.	18.459	0.000	0.871	0.50
1989 Pontiac Grand Am (PFI)	17.943	2.792	2.028	2.303	1.071	5.428	N.A.	5.555	0.000	0.105	0.19
1989 Oldsmobile Delta 88 (PFI)	22.547	3.144	1.206	1.279	0.733	5.973	N.A.	3.369	0.000	0.195	0.13
Average	35.392	5.929	2.483	2.427	1.047	8.818		5.391	0.000	0.269	0.22
Auto/Oil (Fuel W/Industry Avg. +10% EtOH, 9 RVP)											
1989 Plymouth Sundance (TBI)	26.382	4.649	0.762	1.517	0.511	8.771	N.A.	3.414	0.000	0.242	0.14
1989 Dodge Shadow (TBI)	23.528	3.751	1.273	0.692	0.886	6.948	N.A.	3.377	0.000	0.166	0.15
1989 Ford Mustang (PFI)	65.124	12.429	2.389	4.384	0.636	5.436	N.A.	3.200	0.000	0.263	0.25
1989 Ford Taurus (PFI)	24.782	2.956	1.052	1.792	0.769	6.631	N.A.	3.902	0.000	0.266	0.19
1989 Ford Aerostar (PFI)	51.101	7.850	6.568	3.195	1.465	12.326	N.A.	7.237	0.000	0.347	0.25
1989 Camry Sedan (PFI)	15.547	3.400	0.708	0.657	0.531	5.498	N.A.	2.275	0.000	0.215	0.15
1989 Accord Sedan (Carb)	38.152	2.397	0.877	1.658	0.753	4.283	N.A.	2.337	0.000	0.247	0.14
1989 Chevy Suburban (TBI)	61.457	12.229	5.008	7.226	2.062	25.370	N.A.	19.274	0.052	0.921	0.49
1989 Pontiac Grand Am (PFI)	18.358	3.425	1.783	2.341	1.230	6.306	N.A.	5.966	0.000	0.290	0.19
1989 Oldsmobile Delta 88 (PFI)	14.572	2.002	1.057	2.225	0.714	4.097	N.A.	3.170	0.000	0.000	0.09
Average	33.900	5.509	2.148	2.569	0.956	8.567		5.415	0.005	0.296	0.20
Auto/Oil (Fuel U/Low ole., low T90, low arom., 10% EtOH, 10 RVP)											
1989 Plymouth Sundance (TBI)	34.250	6.285	0.896	1.862	0.440	7.259	N.A.	5.030	0.000	0.000	0.15
1989 Dodge Shadow (TBI)	31.175	3.235	1.384	1.125	0.779	6.591	N.A.	5.432	0.000	0.156	0.18
1989 Ford Mustang (PFI)	73.645	15.817	2.162	1.322	0.381	4.171	N.A.	3.509	0.000	0.000	0.27
1989 Ford Taurus (PFI)	29.994	3.051	0.871	1.272	0.531	4.683	N.A.	4.113	0.000	0.000	0.18
1989 Ford Aerostar (PFI)	77.244	10.113	0.934	2.514	0.755	14.822	N.A.	8.639	0.000	0.258	0.28
1989 Camry Sedan (PFI)	17.356	3.191	0.086	0.649	0.374	3.958	N.A.	3.788	0.000	0.000	0.13
1989 Accord Sedan (Carb)	45.912	3.348	1.359	0.946	0.522	3.033	N.A.	2.632	0.000	0.000	0.15
1989 Chevy Suburban (TBI)	70.974	14.711	0.168	1.628	0.505	24.032	N.A.	18.642	0.000	0.000	0.55
1989 Pontiac Grand Am (PFI)	18.595	2.756	1.965	2.013	0.978	4.084	N.A.	4.867	0.000	1.965	0.13
1989 Oldsmobile Delta 88 (PFI)	20.169	2.771	1.463	3.079	0.988	5.671	N.A.	4.388	0.000	0.000	0.13
Average	41.931	6.528	1.129	1.641	0.625	7.830		6.104	0.000	0.238	0.21
Auto/Oil (Fuel T/Low ole., low T90, low arom., 10% EtOH, 9 RVP)											
1989 Plymouth Sundance (TBI)	36.568	6.460	0.790	1.565	0.600	9.924	N.A.	4.639	0.000	0.000	0.15
1989 Dodge Shadow (TBI)	27.838	2.735	0.492	1.378	0.767	5.983	N.A.	4.173	0.000	0.000	0.17
1989 Ford Mustang (PFI)	72.887	14.916	1.475	2.392	0.453	3.967	N.A.	3.790	0.000	0.000	0.28
1989 Ford Taurus (PFI)	33.009	4.228	1.368	1.962	0.607	6.665	N.A.	4.306	0.000	0.000	0.17
1989 Ford Aerostar (PFI)	66.609	9.812	1.025	2.048	0.751	11.219	N.A.	6.920	0.000	0.329	0.25
1989 Camry Sedan (PFI)	16.995	2.105	0.288	0.649	0.278	4.092	N.A.	3.445	0.000	0.195	0.13
1989 Accord Sedan (Carb)	41.088	3.795	1.006	1.071	0.635	4.040	N.A.	2.458	0.000	0.000	0.15
1989 Chevy Suburban (TBI)	69.105	13.865	7.208	2.531	2.256	24.099	N.A.	18.982	0.000	0.000	0.54
1989 Pontiac Grand Am (PFI)	17.515	2.823	2.141	1.907	0.991	4.043	N.A.	6.939	0.000	0.163	0.14
1989 Oldsmobile Delta 88 (PFI)	19.332	2.450	0.983	0.966	0.610	3.839	N.A.	3.749	0.000	0.000	0.11
Average	40.095	6.319	1.678	1.647	0.795	7.787		5.940	0.000	0.069	0.21
AVERAGE*	35.190	5.648	2.050	2.201	0.859	8.496		5.933	0.001	0.203	0.22

8.1% EtOH

Stump et al., 1990 (MSERB Oxyfuels Study)											
CO665 1988 Chevy Corsica TBI	45.200	11.630	2.870	2.910	0.570	15.320	0.000	17.070	N.A.	N.A.	0.34
CO322 1987 Chevy Corsica TBI	36.160	14.830	4.530	3.500	0.600	13.980	0.000	24.850	N.A.	N.A.	0.36
AVERAGE*	40.680	13.230	3.700	3.205	0.585	14.650	0.000	20.960			0.35

19.1% ETBE

1990 SwRI Report Data

1990 Ford Probe (PFI)	N.A.	N.A.	3.400	1.800	0.700	9.500	N.A.	0.000
1990 Chevy Lumina (TBI)	N.A.	N.A.	4.300	2.600	0.800	8.200	N.A.	0.000
1983 Buick Regal (Carb)	N.A.	N.A.	5.300	5.100	0.800	17.500	N.A.	0.000
AVERAGE			4.333	3.167	0.767	11.733		0.000

OXY

E10

Warner-Selph and Smith, 1991 (EPA-460/3-91-02)

1977 Mercury Marquis (Carb)	N.A.	N.A.	44.400	22.400	1.700	13.200	N.A.	3.000	N.A.	N.A.	1.11

NOCAT

E10

Warner-Selph and Smith, 1991 (EPA-460/3-91-02)

1974 Chevy Impala (Carb)	N.A.	N.A.	80.300	26.600	68.000	332.000	N.A.	44.800	N.A.	N.A.	8.31

TWC

MTBE Blends

Auto/Oil (Fuel N2/low ole., low T90, low arom., 15% MTBE, 9 RVP)

1989 Plymouth Sundance (TBI)	24.888	3.755	0.511	0.350	0.579	5.721	N.A.	N.A.	4.953	0.000	0.14
1989 Dodge Shadow (TBI)	58.939	2.746	0.387	0.276	0.682	6.045	N.A.	N.A.	6.838	0.147	0.18
1989 Ford Mustang (PFI)	68.630	13.240	0.613	1.612	0.477	4.769	N.A.	N.A.	2.544	0.000	0.25
1989 Ford Taurus (PFI)	24.485	2.542	0.428	2.181	0.589	6.048	N.A.	N.A.	4.322	0.000	0.14
1989 Ford Aerostar (PFI)	63.054	9.012	0.599	0.928	0.838	14.072	N.A.	N.A.	5.928	0.269	0.35
1989 Camry Sedan (PFI)	16.332	2.540	0.146	0.292	0.315	4.159	N.A.	N.A.	4.620	0.146	0.12
1989 Accord Sedan (Carb)	39.613	3.038	0.538	0.863	0.713	3.700	N.A.	N.A.	0.000	0.000	0.14
1989 Chevy Suburban (TBI)	63.693	13.846	2.822	2.978	2.142	24.453	N.A.	N.A.	31.873	0.470	0.54
1989 Pontiac Grand Am (PFI)	17.628	2.464	0.775	1.638	1.105	4.394	N.A.	N.A.	5.321	0.000	0.13
1989 Oldsmobile Delta 88 (PFI)	20.773	2.854	0.408	1.223	0.540	4.827	N.A.	N.A.	2.843	0.000	0.13
Average*	39.803	5.604	0.723	1.234	0.798	7.819			6.924	0.103	0.21

Auto/Oil (Fuel MM/low ole., low T90, low arom., 15% MTBE, 8 RVP)

1989 Plymouth Sundance (TBI)	24.812	4.942	0.354	1.479	0.455	5.953	N.A.	N.A.	4.222	0.000	0.14
1989 Dodge Shadow (TBI)	31.411	4.618	0.000	0.000	0.731	5.909	N.A.	N.A.	3.903	0.171	0.17
1989 Ford Mustang (PFI)	66.130	12.544	0.612	3.734	0.435	3.378	N.A.	N.A.	0.000	0.000	0.22
1989 Ford Taurus (PFI)	25.365	2.963	1.723	1.125	0.627	5.465	N.A.	N.A.	6.121	0.154	0.15
1989 Ford Aerostar (PFI)	60.077	7.848	0.642	0.782	0.894	12.289	N.A.	N.A.	5.558	0.279	0.33
1989 Camry Sedan (PFI)	16.843	2.778	0.096	0.299	0.310	4.035	N.A.	N.A.	8.171	0.085	0.13
1989 Accord Sedan (Carb)	42.966	3.291	0.633	1.159	0.726	4.295	N.A.	N.A.	2.905	0.000	0.17
1989 Chevy Suburban (TBI)	58.530	12.093	3.073	5.501	2.379	21.955	N.A.	N.A.	23.392	0.545	0.52
1989 Pontiac Grand Am (PFI)	15.590	2.501	0.750	1.322	1.072	4.169	N.A.	N.A.	4.562	0.167	0.12
1989 Oldsmobile Delta 88 (PFI)	19.671	2.748	0.371	0.796	0.573	4.435	N.A.	N.A.	4.361	0.000	0.12
Average*	36.140	5.633	0.826	1.620	0.820	7.188			6.319	0.140	0.21

*Data used in subsequent emission fractions calculations

01/28/1987

Methane	Ethane	Fraction of TOG Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene	Methanol	Ethanol	MTBE	ETBE	
										TWC
										M85
										AB234 Study
										1987 Ford Crown Victoria (FI)
										MSERB FFV Study
										1988 Chevrolet Corsica (TBI)
										AVERAGE
										M100
										AB234 Study
										1987 Ford Crown Victoria (FI)
										MSERB FFV Study
										1988 Chevrolet Corsica (TBI)
										AVERAGE
										E95
										AB234 Study
										1987 Ford Crown Victoria (FI)
										E85
										AB234 Study
										1987 Ford Crown Victoria (TBI)
										E0
										Auto/Oil (Fuel A/Industry Avg.)
0.1528	0.0250	0.0031	0.0034	0.0045	0.0458	N.A.	N.A.	0.0000	0.0017	1989 Plymouth Sundance (TBI)
0.1525	0.0188	0.0054	0.0021	0.0059	0.0514	N.A.	N.A.	0.0000	0.0010	1989 Dodge Shadow (TBI)
0.2031	0.0395	0.0038	0.0029	0.0021	0.0225	N.A.	N.A.	0.0048	0.0009	1989 Ford Mustang (PFI)
0.1319	0.0166	0.0030	0.0069	0.0047	0.0463	N.A.	N.A.	0.0000	0.0018	1989 Ford Taurus (PFI)
0.1669	0.0264	0.0022	0.0005	0.0045	0.0473	N.A.	N.A.	0.0081	0.0012	1989 Ford Aerostar (PFI)
0.1060	0.0223	0.0037	0.0173	0.0030	0.0378	N.A.	N.A.	0.0000	0.0014	1989 Camry Sedan (PFI)
0.2599	0.0196	0.0188	0.0184	0.0060	0.0361	N.A.	N.A.	0.0000	0.0011	1989 Accord Sedan (Carb)
0.1145	0.0267	0.0071	0.0060	0.0042	0.0633	N.A.	N.A.	0.0006	0.0020	1989 Chevy Suburban (TBI)
0.0997	0.0167	0.0065	0.0186	0.0084	0.0401	N.A.	N.A.	0.0000	0.0016	1989 Pontiac Grand Am (PFI)
0.1271	0.0179	0.0053	0.0038	0.0072	0.0396	N.A.	N.A.	0.0000	0.0013	1989 Oldsmobile Delta 88 (PFI)
0.1515	0.0230	0.0059	0.0080	0.0050	0.0430			0.0013	0.0014	Average*
										E10
										Warner-Selph and Smith, 1991 (EPA-460/3-91-02)
N.A.	N.A.	0.0183	0.0106	0.0039	0.0386	N.A.	0.0410	N.A.	N.A.	1990 Ford Probe (PFI)
N.A.	N.A.	0.0283	0.0179	0.0055	0.0421	N.A.	0.0000	N.A.	N.A.	1990 Chevy Lumina (TBI)
N.A.	N.A.	0.0073	0.0087	0.0014	0.0262	N.A.	0.0224	N.A.	N.A.	1983 Buick Regal (Carb)
		0.0180	0.0124	0.0036	0.0356		0.0211			Average
										Auto/Oil (Fuel X/Industry Avg. +10% EtOH, 10 RVP)
0.1828	0.0300	0.0064	0.0103	0.0045	0.0484	N.A.	0.0246	0.0000	0.0018	1989 Plymouth Sundance (TBI)
0.1511	0.0165	0.0531	0.0100	0.0058	0.0424	N.A.	0.0182	0.0000	0.0010	1989 Dodge Shadow (TBI)

0.2267	0.0456	0.0063	0.0066	0.0016	0.0148	N.A.	0.0114	0.0000	0.0000	1989 Ford Mustang (PFI)
0.1372	0.0174	0.0045	0.0096	0.0038	0.0384	N.A.	0.0238	0.0000	0.0016	1989 Ford Taurus (PFI)
0.2066	0.0303	0.0033	0.0100	0.0053	0.0528	N.A.	0.0286	0.0000	0.0014	1989 Ford Aerostar (PFI)
0.1050	0.0256	0.0013	0.0059	0.0033	0.0374	N.A.	0.0157	0.0000	0.0016	1989 Camry Sedan (PFI)
0.2790	0.0297	0.0216	0.0122	0.0052	0.0269	N.A.	0.0169	0.0000	0.0014	1989 Accord Sedan (Carb)
0.1256	0.0281	0.0097	0.0171	0.0065	0.0557	N.A.	0.0367	0.0000	0.0017	1989 Chevy Suburban (TBI)
0.0943	0.0147	0.0107	0.0121	0.0056	0.0285	N.A.	0.0292	0.0000	0.0006	1989 Pontiac Grand Am (PFI)
0.1706	0.0238	0.0091	0.0097	0.0055	0.0452	N.A.	0.0255	0.0000	0.0015	1989 Oldsmobile Delta 88 (PFI)
0.1679	0.0262	0.0126	0.0104	0.0047	0.0391		0.0231	0.0000	0.0012	Average
										Auto/Oil (Fuel W/Industry Avg. +10% EtOH, 9 RVP)
0.1867	0.0329	0.0054	0.0107	0.0036	0.0621	N.A.	0.0242	0.0000	0.0017	1989 Plymouth Sundance (TBI)
0.1622	0.0259	0.0088	0.0048	0.0061	0.0479	N.A.	0.0233	0.0000	0.0011	1989 Dodge Shadow (TBI)
0.2619	0.0500	0.0096	0.0176	0.0026	0.0219	N.A.	0.0129	0.0000	0.0011	1989 Ford Mustang (PFI)
0.1313	0.0157	0.0056	0.0095	0.0041	0.0351	N.A.	0.0207	0.0000	0.0014	1989 Ford Taurus (PFI)
0.2065	0.0317	0.0265	0.0129	0.0059	0.0498	N.A.	0.0292	0.0000	0.0014	1989 Ford Aerostar (PFI)
0.1072	0.0234	0.0049	0.0045	0.0037	0.0379	N.A.	0.0157	0.0000	0.0015	1989 Camry Sedan (PFI)
0.2732	0.0172	0.0063	0.0119	0.0054	0.0307	N.A.	0.0167	0.0000	0.0018	1989 Accord Sedan (Carb)
0.1249	0.0249	0.0102	0.0147	0.0042	0.0516	N.A.	0.0392	0.0001	0.0019	1989 Chevy Suburban (TBI)
0.0965	0.0180	0.0094	0.0123	0.0065	0.0331	N.A.	0.0314	0.0000	0.0015	1989 Pontiac Grand Am (PFI)
0.1563	0.0215	0.0113	0.0239	0.0077	0.0439	N.A.	0.0340	0.0000	0.0000	1989 Oldsmobile Delta 88 (PFI)
0.1707	0.0261	0.0098	0.0123	0.0050	0.0414		0.0247	0.0000	0.0013	Average
										Auto/Oil (Fuel U/Low ole., low T90, low arom., 10% EtOH, 10 RVP)
0.2317	0.0425	0.0061	0.0126	0.0030	0.0491	N.A.	0.0340	0.0000	0.0000	1989 Plymouth Sundance (TBI)
0.1770	0.0184	0.0079	0.0064	0.0044	0.0374	N.A.	0.0308	0.0000	0.0009	1989 Dodge Shadow (TBI)
0.2734	0.0587	0.0080	0.0049	0.0014	0.0155	N.A.	0.0130	0.0000	0.0000	1989 Ford Mustang (PFI)
0.1661	0.0169	0.0048	0.0070	0.0029	0.0259	N.A.	0.0228	0.0000	0.0000	1989 Ford Taurus (PFI)
0.2758	0.0361	0.0033	0.0090	0.0027	0.0529	N.A.	0.0309	0.0000	0.0009	1989 Ford Aerostar (PFI)
0.1323	0.0243	0.0007	0.0049	0.0028	0.0302	N.A.	0.0289	0.0000	0.0000	1989 Camry Sedan (PFI)
0.3166	0.0231	0.0094	0.0065	0.0036	0.0209	N.A.	0.0181	0.0000	0.0000	1989 Accord Sedan (Carb)
0.1293	0.0268	0.0003	0.0030	0.0009	0.0438	N.A.	0.0340	0.0000	0.0000	1989 Chevy Suburban (TBI)
0.1452	0.0215	0.0153	0.0157	0.0076	0.0319	N.A.	0.0380	0.0000	0.0153	1989 Pontiac Grand Am (PFI)
0.1498	0.0206	0.0109	0.0229	0.0073	0.0421	N.A.	0.0326	0.0000	0.0000	1989 Oldsmobile Delta 88 (PFI)
0.1997	0.0289	0.0067	0.0093	0.0037	0.0350		0.0283	0.0000	0.0017	Average
										Auto/Oil (Fuel T/Low ole., low T90, low arom., 10% EtOH, 9 RVP)
0.2424	0.0428	0.0052	0.0104	0.0040	0.0658	N.A.	0.0308	0.0000	0.0000	1989 Plymouth Sundance (TBI)
0.1603	0.0158	0.0028	0.0079	0.0044	0.0345	N.A.	0.0240	0.0000	0.0000	1989 Dodge Shadow (TBI)
0.2573	0.0527	0.0052	0.0084	0.0016	0.0140	N.A.	0.0134	0.0000	0.0000	1989 Ford Mustang (PFI)
0.1968	0.0252	0.0082	0.0117	0.0036	0.0397	N.A.	0.0257	0.0000	0.0000	1989 Ford Taurus (PFI)
0.2615	0.0385	0.0040	0.0080	0.0029	0.0440	N.A.	0.0272	0.0000	0.0013	1989 Ford Aerostar (PFI)
0.1278	0.0158	0.0022	0.0049	0.0021	0.0308	N.A.	0.0259	0.0000	0.0015	1989 Camry Sedan (PFI)
0.2787	0.0257	0.0068	0.0073	0.0043	0.0274	N.A.	0.0167	0.0000	0.0000	1989 Accord Sedan (Carb)
0.1283	0.0257	0.0134	0.0047	0.0042	0.0447	N.A.	0.0352	0.0000	0.0000	1989 Chevy Suburban (TBI)
0.1215	0.0196	0.0148	0.0132	0.0069	0.0280	N.A.	0.0481	0.0000	0.0011	1989 Pontiac Grand Am (PFI)
0.1764	0.0224	0.0090	0.0088	0.0056	0.0350	N.A.	0.0342	0.0000	0.0000	1989 Oldsmobile Delta 88 (PFI)
0.1951	0.0284	0.0072	0.0085	0.0040	0.0364		0.0281	0.0000	0.0004	Average
0.1705	0.0255	0.0097	0.0103	0.0043	0.0378		0.0257	0.0000	0.0011	AVERAGE*
										8.1% EtOH
										Stump et al., 1990 (MSERB Oxyfuels Study)
0.1322	0.0340	0.0084	0.0085	0.0017	0.0448	0.0000	0.0499	N.A.	N.A.	CO665 1988 Chevy Corsica TBI
0.0997	0.0409	0.0125	0.0097	0.0017	0.0386	0.0000	0.0685	N.A.	N.A.	CO322 1987 Chevy Corsica TBI
0.1160	0.0375	0.0104	0.0091	0.0017	0.0417	0.0000	0.0592			AVERAGE*
										19.1% ETBE

1990 SwRI Report Data
1990 Ford Probe (PFI)
1990 Chevy Lumina (TBI)
1983 Buick Regal (Carb)
AVERAGE

OXY
E10

Warner-Selph and Smith, 1991 (EPA-460/3-91-02)

N.A.	N.A.	0.0399	0.0201	0.0015	0.0119	N.A.	0.0027	N.A.	N.A.	1977 Mercury Marquis (Carb)

NOCAT
E10

Warner-Selph and Smith, 1991 (EPA-460/3-91-02)

N.A.	N.A.	0.0097	0.0032	0.0082	0.0400	N.A.	0.0054	N.A.	N.A.	1974 Chevy Impala (Carb)

TWC

MTBE Blends

Auto/Oil (Fuel N2/low ole., low T90, low arom., 15% MTBE, 9 RVP)

0.1722	0.0260	0.0035	0.0024	0.0040	0.0396	N.A.	N.A.	0.0343	0.0000	1989 Plymouth Sundance (TBI)
0.3358	0.0156	0.0022	0.0016	0.0039	0.0344	N.A.	N.A.	0.0390	0.0008	1989 Dodge Shadow (TBI)
0.2770	0.0534	0.0025	0.0065	0.0019	0.0192	N.A.	N.A.	0.0103	0.0000	1989 Ford Mustang (PFI)
0.1694	0.0176	0.0030	0.0151	0.0041	0.0418	N.A.	N.A.	0.0299	0.0000	1989 Ford Taurus (PFI)
0.1796	0.0257	0.0017	0.0026	0.0024	0.0401	N.A.	N.A.	0.0169	0.0008	1989 Ford Aerostar (PFI)
0.1318	0.0205	0.0012	0.0024	0.0025	0.0336	N.A.	N.A.	0.0373	0.0012	1989 Camry Sedan (PFI)
0.2741	0.0210	0.0037	0.0060	0.0049	0.0256	N.A.	N.A.	0.0000	0.0000	1989 Accord Sedan (Carb)
0.1186	0.0258	0.0053	0.0055	0.0040	0.0455	N.A.	N.A.	0.0594	0.0009	1989 Chevy Suburban (TBI)
0.1313	0.0184	0.0058	0.0122	0.0082	0.0327	N.A.	N.A.	0.0396	0.0000	1989 Pontiac Grand Am (PFI)
0.1548	0.0213	0.0030	0.0091	0.0040	0.0360	N.A.	N.A.	0.0212	0.0000	1989 Oldsmobile Delta 88 (PFI)
0.1945	0.0245	0.0032	0.0063	0.0040	0.0349			0.0288	0.0004	Average*

Auto/Oil (Fuel MM/low ole., low T90, low arom., 15% MTBE, 8 RVP)

0.1717	0.0342	0.0024	0.0102	0.0031	0.0412	N.A.	N.A.	0.0292	0.0000	1989 Plymouth Sundance (TBI)
0.1901	0.0280	0.0000	0.0000	0.0044	0.0358	N.A.	N.A.	0.0236	0.0010	1989 Dodge Shadow (TBI)
0.3050	0.0579	0.0028	0.0172	0.0020	0.0156	N.A.	N.A.	0.0000	0.0000	1989 Ford Mustang (PFI)
0.1638	0.0191	0.0111	0.0073	0.0041	0.0353	N.A.	N.A.	0.0395	0.0010	1989 Ford Taurus (PFI)
0.1818	0.0238	0.0019	0.0024	0.0027	0.0372	N.A.	N.A.	0.0168	0.0008	1989 Ford Aerostar (PFI)
0.1305	0.0215	0.0007	0.0023	0.0024	0.0313	N.A.	N.A.	0.0633	0.0007	1989 Camry Sedan (PFI)
0.2601	0.0199	0.0038	0.0070	0.0044	0.0260	N.A.	N.A.	0.0176	0.0000	1989 Accord Sedan (Carb)
0.1134	0.0234	0.0060	0.0107	0.0046	0.0425	N.A.	N.A.	0.0453	0.0011	1989 Chevy Suburban (TBI)
0.1258	0.0202	0.0061	0.0107	0.0087	0.0336	N.A.	N.A.	0.0368	0.0013	1989 Pontiac Grand Am (PFI)
0.1588	0.0222	0.0030	0.0064	0.0046	0.0358	N.A.	N.A.	0.0352	0.0000	1989 Oldsmobile Delta 88 (PFI)
0.1801	0.0270	0.0038	0.0074	0.0041	0.0334			0.0307	0.0006	Average*

LDDV, HDDV, HDGV (FTP and 13 Mode Data)

LDDV

	Exhaust mg/mile							Fraction of TOG					
	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene	TOG, g/mile	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene
Springer, 1977 (EPA-460/3-76-034)													
1975 Mercedes 220D	31.620	0.000	4.050	1.610	N.A.	9.767	0.19	0.1675	0.0000	0.0214	0.0085		0.0517
1975 Mercedes 240D	8.960	0.000	6.370	1.820	N.A.	0.000	0.30	0.0295	0.0000	0.0209	0.0060		0.0000
1975 Mercedes 300D	6.340	0.000	6.110	1.790	N.A.	4.039	0.17	0.0378	0.0000	0.0364	0.0107		0.0241
1974 Peugeot 204D	14.960	0.000	18.100	6.890	N.A.	0.000	1.16	0.0128	0.0000	0.0155	0.0059		0.0000
1974 Perkins 6-247	39.390	0.000	61.420	16.890	N.A.	15.382	0.76	0.0522	0.0000	0.0813	0.0224		0.0204
Average	20.254	0.000	19.210	5.800		5.837	0.52	0.0599	0.0000	0.0351	0.0107		0.0192
Springer, 1979 (EPA-460/3-79-007)													
1976 Cutlass	20.430	6.760	25.420	10.460	N.A.	18.664	0.80	0.0256	0.0085	0.0319	0.0131		0.0234
1977 Rabbit	10.780	1.450	25.740	8.050	N.A.	8.206	0.39	0.0278	0.0037	0.0663	0.0207		0.0211
Average	15.605	4.105	25.580	9.255		13.435	0.59	0.0267	0.0061	0.0491	0.0169		0.0223
AVERAGE*	19.092	1.026	20.803	6.664		7.737	0.43	0.0516	0.0015	0.0386	0.0123		0.0200
CARB Butadiene Study (2 vehicles)												0.0090	

HDDV (mg/kw-hr)

	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene	TOG, g/mile	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene
Springer, 1979 (EPA-460/3-79-007)													
Mack ETAY(B)673A	6.900	0.680	16.590	0.940	N.A.	4.480	0.66	0.0105	0.0010	0.0251	0.0014		0.0068
Cat 3208/EGR	40.350	3.500	49.900	21.850	N.A.	21.930	1.61	0.0250	0.0022	0.0309	0.0136		0.0136
AVERAGE*	23.625	2.090	33.245	11.395		13.205	1.14	0.0177	0.0016	0.0280	0.0075		0.0102
CARB Butadiene Study (1 vehicle)												0.0136	

HDGV (mg/kw-hr)

	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene	TOG, g/mile	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene
Springer, 1979 (EPA-460/3-79-007)													
Chev. 366 (23 mode)	836.600	41.810	105.360	21.270	N.A.	348.780	3.40	0.2461	0.0123	0.0310	0.0063		0.1026

*Data used in subsequent emission fractions calculations
      01/28/1988

LDGV (NON-CAT -- FTP Data)

	Exhaust mg/mile							Fraction of TOG					
	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene	TOG, g/mile	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene
0% MTBE													
Urban, 1980a, 1981 (EPA-460/3-80-003, 81-020, All Carb)													
71 1970 Olds Delta 88 (Carb)	133.580	22.850	51.280	7.180	N.A.	82.192	2.38	0.0562	0.0096	0.0216	0.0030	N.A.	0.0346
72 1970 Dodge Challenger (Carb)	158.840	24.330	14.980	5.550	N.A.	86.544	2.84	0.0559	0.0086	0.0053	0.0020	N.A.	0.0305
73 1970 Chev Monte Carlo (Carb)	135.930	29.380	107.430	10.840	N.A.	91.104	3.41	0.0399	0.0086	0.0315	0.0032	N.A.	0.0267
74 1970 Ford Fairlane (Carb)	123.670	37.480	34.260	2.280	N.A.	85.456	3.90	0.0317	0.0096	0.0088	0.0006	N.A.	0.0219
11 1977 AMC Pacer (Carb)	77.230	11.260	15.620	3.780	N.A.	64.000	1.21	0.0639	0.0093	0.0129	0.0031	N.A.	0.0530
Average	125.850	25.060	44.714	5.926		81.859	2.75	0.0495	0.0091	0.0160	0.0024		0.0333
Sigsby et al., 1987 (46 car study)													
6 1976 Toyota Celica	44.160	9.890	77.640	19.680	N.A.	26.163	0.88	0.0503	0.0113	0.0884	0.0224	N.A.	0.0298
11 1977 AMC Hornet	1066.670	60.810	121.840	20.630	N.A.	112.889	6.31	0.1690	0.0096	0.0193	0.0033	N.A.	0.0179
24 1977 Datsun F-10	71.300	13.280	84.800	20.690	N.A.	21.436	1.98	0.0359	0.0067	0.0428	0.0104	N.A.	0.0108
31 1979 Mazda RX-7	43.840	11.540	65.680	14.760	N.A.	20.826	1.29	0.0340	0.0089	0.0509	0.0114	N.A.	0.0161
35 1980 Olds Cutlass	201.170	40.130	9.740	12.750	N.A.	42.768	2.07	0.0974	0.0194	0.0047	0.0062	N.A.	0.0207
Average	285.428	27.130	71.940	17.702		44.816	2.51	0.0773	0.0112	0.0412	0.0107		0.0191
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)													
1974 Chevy Impala (Carb)	N.A.	N.A.	84.100	19.500	60.400	421.000	8.31	N.A.	N.A.	0.0101	0.0023	0.0073	0.0507
AVERAGE (1 vehicle)*	186.945	23.723	60.670	12.513	5.491	95.852	3.14	0.0577	0.0092	0.0269	0.0062	0.0007	0.0284
CARB Butadiene Study (16 vehicles)												0.0097	
											Average	0.0096	
16.4% MTBE													
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)													
1974 Chevy Impala (Carb)	N.A.	N.A.	100.000	21.600	58.200	240.000	6.30	N.A.	N.A.	0.0159	0.0034	0.0092	0.0381

*Data used in subsequent emission fractions calculations
                01/28/1988

LDGV (OX-CAT -- FTP Data)

			Exhaust (mg/mi)					Fraction of TOG					
	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene	TOG, g/mile	Methane	Ethane	Formaldehyde	Acetaldehyde	1,3-Butadiene	Benzene
0% MTBE													
Urban, 1980a (EPA-460/3-80-003, All Carb)													
12 1978 Chev. Malibu	69.830	16.570	1.770	0.500	N.A.	26.560	0.51	0.1373	0.0326	0.0035	0.0010	N.A.	0.0522
14 1978 Ford Granada	59.530	22.530	4.650	0.230	N.A.	12.800	0.44	0.1361	0.0515	0.0106	0.0005	N.A.	0.0293
15 1978 Ford Mustang II	72.410	12.870	2.540	1.010	N.A.	22.400	0.52	0.1396	0.0248	0.0049	0.0019	N.A.	0.0432
Average	67.257	17.323	2.987	0.580		20.587	0.49	0.1377	0.0363	0.0063	0.0012		0.0416
Springer, 1979 (EPA-460/3-79-007)													
1977 Oldsmobile Cutlass (Carb)	47.466	23.680	4.183	0.644	N.A.	9.010	0.34	0.1381	0.0689	0.0122	0.0019	N.A.	0.0262
1977 Volkswagen Rabbit (FI)	53.097	10.298	0.644	0.000	N.A.	14.642	0.23	0.2318	0.0450	0.0028	0.0000	N.A.	0.0639
Average	50.281	16.989	2.414	0.322		11.826	0.29	0.1850	0.0569	0.0075	0.0009		0.0451
Sigsby et al., 1987 (46 car study)													
1 1979 Chev. Chevette (Carb)	138.320	22.800	7.550	2.450	N.A.	53.960	1.25	0.1106	0.0182	0.0060	0.0020	N.A.	0.0431
2 1978 Ford LTD Wagon (Carb)	129.930	27.800	18.470	7.630	N.A.	17.767	0.74	0.1750	0.0374	0.0249	0.0103	N.A.	0.0239
4 1979 Ply Volare (Carb)	222.500	40.000	12.740	6.170	N.A.	80.250	2.12	0.1052	0.0189	0.0060	0.0029	N.A.	0.0379
5 1978 Ford Mustang (FI)	468.940	72.810	68.100	18.910	N.A.	144.622	4.78	0.0981	0.0152	0.0142	0.0040	N.A.	0.0303
7 1980 VW Scirrocco (FI)	71.810	11.220	4.010	2.940	N.A.	8.825	0.45	0.1605	0.0251	0.0090	0.0066	N.A.	0.0197
12 1976 Olds Starfire (Carb)	239.730	66.690	18.990	12.080	N.A.	6.511	2.86	0.0839	0.0233	0.0066	0.0042	N.A.	0.0023
13 1976 Olds Regency (Carb)	295.370	43.290	17.040	8.130	N.A.	58.499	2.22	0.1332	0.0195	0.0077	0.0037	N.A.	0.0264
14 1977 Buick Skyhawk (Carb)	180.970	29.300	16.300	7.240	N.A.	29.700	1.51	0.1202	0.0195	0.0108	0.0048	N.A.	0.0197
15 1975 Ply Valiant (Carb)	134.640	25.610	35.760	10.660	N.A.	30.652	1.55	0.0871	0.0166	0.0231	0.0069	N.A.	0.0198
17 1978 Pont Phoenix (Carb)	209.980	35.790	17.020	11.210	N.A.	46.452	1.95	0.1075	0.0183	0.0087	0.0057	N.A.	0.0238
18 1979 Toyota Corolla (Carb)	114.970	31.560	27.360	9.730	N.A.	29.447	1.61	0.0715	0.0196	0.0170	0.0061	N.A.	0.0183
19 1980 Buick Electra (Carb)	155.750	28.480	16.360	7.660	N.A.	12.060	0.78	0.1989	0.0364	0.0209	0.0098	N.A.	0.0154
20 1977 Chev Chevette (Carb)	162.260	27.850	9.330	5.510	N.A.	24.898	1.70	0.0955	0.0164	0.0055	0.0032	N.A.	0.0147
22 1978 Ply Volare (Carb)	204.770	35.210	18.570	5.570	N.A.	34.020	1.86	0.1100	0.0189	0.0100	0.0030	N.A.	0.0183
23 1978 Datsun 200SX (Carb)	83.300	23.500	129.950	25.980	N.A.	18.512	1.55	0.0539	0.0152	0.0841	0.0168	N.A.	0.0120
25 1979 Ford Fairmont (Carb)	268.200	33.190	19.540	8.630	N.A.	25.565	2.26	0.1188	0.0147	0.0087	0.0038	N.A.	0.0113
26 1980 Mazda GLC (Carb)	178.230	21.170	48.750	14.290	N.A.	23.562	1.92	0.0927	0.0110	0.0254	0.0074	N.A.	0.0123
27 1981 Chev Chevette (Carb)	138.170	22.120	3.890	2.930	N.A.	20.121	0.83	0.1657	0.0265	0.0047	0.0035	N.A.	0.0241
32 1975 Olds Cutlass (Carb)	722.230	71.640	92.820	22.730	N.A.	87.355	5.36	0.1348	0.0134	0.0173	0.0042	N.A.	0.0163
37 1980 Chev Citation (Carb)	88.140	13.620	5.840	2.460	N.A.	6.705	0.37	0.2407	0.0372	0.0160	0.0067	N.A.	0.0183
39 1980 Ford Fairmont (Carb)	124.570	38.010	78.460	19.200	N.A.	39.104	1.78	0.0700	0.0214	0.0441	0.0108	N.A.	0.0220
43 1981 Crys Lebaron (Carb)	124.660	16.640	6.330	3.670	N.A.	10.363	0.41	0.3064	0.0409	0.0156	0.0090	N.A.	0.0255
44 1981 AMC Concord (Carb)	96.860	28.890	8.680	6.790	N.A.	27.305	1.38	0.0700	0.0209	0.0063	0.0049	N.A.	0.0197
46 1980 Crys Lebaron (Carb)	101.770	8.820	6.600	2.820	N.A.	10.363	0.38	0.2705	0.0234	0.0175	0.0075	N.A.	0.0275
Average	194.003	32.334	28.686	9.391		35.276	1.73	0.1325	0.0220	0.0171	0.0062		0.0209
Smith, 1981 (EPA-460/3-81-024, All Carb)													
1 1978 Buick Regal B	109.910	26.760	14.210	2.610	N.A.	86.528	1.74	0.0632	0.0154	0.0082	0.0015	N.A.	0.0498
4 1978 Ford Granada B	215.120	68.800	8.660	1.830	N.A.	59.664	1.77	0.1216	0.0389	0.0049	0.0010	N.A.	0.0337
A	195.700	68.060	11.860	2.700	N.A.	53.344	1.83	0.1069	0.0372	0.0065	0.0015	N.A.	0.0291
6 1978 Olds Cutlass B	87.220	15.590	1.820	3.440	N.A.	51.264	0.88	0.0986	0.0176	0.0021	0.0039	N.A.	0.0579
A	89.640	19.400	1.960	1.950	N.A.	43.712	0.78	0.1145	0.0248	0.0025	0.0025	N.A.	0.0558
7 1978 Chev Malibu B	143.640	19.550	10.230	2.140	N.A.	33.872	0.65	0.2207	0.0300	0.0157	0.0033	N.A.	0.0520
A	113.160	16.780	12.360	1.350	N.A.	33.520	0.67	0.1686	0.0250	0.0184	0.0020	N.A.	0.0499
8 1978 Chev Monte C B	34.350	10.510	8.540	0.000	N.A.	17.936	0.46	0.0751	0.0230	0.0187	0.0000	N.A.	0.0392
A	83.060	21.380	2.040	0.480	N.A.	48.464	0.85	0.0972	0.0250	0.0024	0.0006	N.A.	0.0567
9 1978 Ford Fiesta B	39.520	15.770	2.740	0.000	N.A.	12.512	0.65	0.0607	0.0242	0.0042	0.0000	N.A.	0.0192
A	52.940	16.520	2.160	0.000	N.A.	9.808	0.56	0.0946	0.0295	0.0039	0.0000	N.A.	0.0175
10 1978 Chy N YorkerB	472.180	54.580	27.370	4.540	N.A.	209.408	4.65	0.1016	0.0117	0.0059	0.0010	N.A.	0.0451
A	137.940	31.790	9.350	0.290	N.A.	67.248	1.36	0.1012	0.0233	0.0069	0.0002	N.A.	0.0493
Average	136.491	29.653	8.715	1.641		55.945	1.30	0.1096	0.0251	0.0077	0.0013		0.0427
Stump et al., 1989, 1990 (MSERB Low Temp. Study All Carb)													
1987 Chy Caravelle (FI)	82.500	7.600	2.460	1.320	0.800	16.080	0.41	0.2028	0.0187	0.0060	0.0032	0.0020	0.0395

Auto/Oil Study

1984 Chevrolet Suburban (Carb)	73.514	16.815	22.656	9.086	1.888	20.355	0.58	0.1268	0.0290	0.0391	0.0157	0.0033	0.0351
1983 Ford F-150 (Carb)	124.092	33.156	25.164	7.668	4.644	32.616	1.09	0.1140	0.0305	0.0231	0.0070	0.0043	0.0300
Average	98.803	24.986	23.910	8.377	3.266	26.486	0.83	0.1204	0.0297	0.0311	0.0114	0.0038	0.0325

Arco 91-03

1980 Chevrolet Monza (Carb)	130.800	14.200	8.900	0.600	4.300	42.100	1.42	0.0919	0.0100	0.0063	0.0004	0.0030	0.0296

Warner-Selph and Smith, 1991 (EPA-460/3-91-02)

1977 Mercury Marquis (Carb)	N.A.	N.A.	31.700	10.900	1.500	17.300	0.92	N.A.	N.A.	0.0346	0.0119	0.0016	0.0189

AVERAGE (5 vehicles)*	151.993	28.068	19.286	5.929	0.279	38.037	1.38	0.1260	0.0248	0.0139	0.0044	0.0003	0.0303
CARB Butadiene Study (7 vehicles)												0.0054	
											Average	0.0043	

9.0% MTBE

Arco 91-03

1980 Chevrolet Monza (Carb)	130.800	14.200	8.900	0.600	1.100	42.100	1.44	0.0908	0.0099	0.0062	0.0004	0.0008	0.0292

15.0% MTBE

Auto/Oil Study

1984 Chevrolet Suburban (Carb)	56.699	15.281	33.748	10.620	1.829	18.526	0.58	0.0976	0.0263	0.0581	0.0183	0.0031	0.0319
1983 Ford F-150 (Carb)	91.560	29.400	60.795	9.030	6.300	32.130	1.03	0.0892	0.0286	0.0592	0.0088	0.0061	0.0313
AVERAGE*	74.130	22.341	47.272	9.825	4.065	25.328	0.80	0.0934	0.0275	0.0587	0.0135	0.0046	0.0316

16.4% MTBE

Warner-Selph and Smith, 1991 (EPA-460/3-91-02)

1977 Mercury Marquis (Carb)	N.A.	N.A.	100.000	21.600	1.700	12.600	1.11	N.A.	N.A.	0.0900	0.0194	0.0015	0.0113

*Data used in subsequent emission fractions calculations
01/28/1988

LDGV (PFI -- FTP Data)

| | Diurnal Evap (mg/test) | | | Fraction of THC | | Hot Soak Evap (mg/test) | | | Fraction of THC | |
	MTBE*	Benzene	THC (g/test)	MTBE	Benzene	MTBE*	Benzene	THC (g/test)	MTBE	Benzene
0% MTBE										
Arco 91-02 (Unleaded Reg)**										
21 1989 Toyota Camry (TWC)	0.000	7.330	0.256	0.0000	0.0286	0.000	9.190	0.157	0.0000	0.0585
22 1989 Pont. Grand Prix (TWC)	0.000	1.200	0.578	0.0000	0.0021	0.000	9.680	0.359	0.0000	0.0270
23 1989 Ford Taurus (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
24 1989 Dodge Dynasty (TWC)	0.000	2.250	0.219	0.0000	0.0103	0.000	2.180	0.213	0.0000	0.0102
25 1989 Nissan Maxima (TWC)	0.000	9.720	0.469	0.0000	0.0207	0.000	41.130	1.203	0.0000	0.0342
Average	0.000	4.100	0.304	0.0000	0.0123	0.000	12.436	0.386	0.0000	0.0260
Arco 91-03 (Unleaded Premium)										
35 1988 Pontiac Grand Am (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
40 1987 Nissan 300ZX (TWC)	5.450	14.700	0.430	0.0127	0.0342	6.240	24.820	0.384	0.0163	0.0647
41 1988 Toyota 4WD Pickup (TWC)	4.210	5.050	0.146	0.0289	0.0346	2.180	9.040	0.129	0.0170	0.0703
42 1988 Toyota Celica (TWC)	1.160	6.600	0.264	0.0044	0.0250	0.860	8.140	0.109	0.0079	0.0746
44 1986 Mercedes 190E (TWC)	0.000	3.790	0.088	0.0000	0.0428	0.000	8.300	0.174	0.0000	0.0478
45 1989 Lincoln Continental (TWC)	0.000	4.840	0.157	0.0000	0.0309	0.000	5.840	0.137	0.0000	0.0426
46 1990 Plymouth Acclaim (TWC)	0.450	8.180	0.280	0.0016	0.0292	10.230	8.340	0.278	0.0368	0.0300
47 1984 Toyota Celica (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
49 1987 Oldsmobile Regency (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
56 1982 Nissan Maxima (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
57 1986 Buick Park Avenue (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
34 1985 Ford Bronco (TWC + OX)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
Average	0.939	3.597	0.114	0.0040	0.0164	1.626	5.373	0.101	0.0065	0.0275
Arco 91-06 (Ind. Avg. Baseline)**										
350 1990 Ford Taurus (TWC)	0.000	5.860	0.262	0.0000	0.0224	0.000	3.320	0.150	0.0000	0.0221
351 1990 Toyota Camry (TWC)	0.000	12.030	0.643	0.0000	0.0187	0.000	10.440	0.585	0.0000	0.0178
353 1990 Honda Accord (TWC)	0.000	1.220	0.120	0.0000	0.0102	4.270	3.030	0.193	0.0221	0.0157
354 1990 Nissan Stanza (TWC)	0.000	20.930	0.805	0.0000	0.0260	0.000	32.090	1.190	0.0000	0.0270
355 1990 Pontiac Grand Am (TWC)	0.000	3.650	0.150	0.0000	0.0243	0.000	11.230	0.285	0.0000	0.0394
356 1990 Ford Crown Victoria (TWC)	0.000	6.320	0.355	0.0000	0.0178	0.000	3.460	0.130	0.0000	0.0266
357 1990 Plymouth Voyager (TWC)	0.000	3.900	0.485	0.0000	0.0080	0.000	5.050	0.133	0.0000	0.0380
358 1990 Nissan Pickup (TWC)	0.000	9.150	0.358	0.0000	0.0256	0.000	10.040	0.293	0.0000	0.0343
359 1990 Buick LeSabre (TWC)	0.000	1.430	0.123	0.0000	0.0116	0.000	6.350	0.128	0.0000	0.0496
Average	0.000	7.166	0.367	0.0000	0.0183	0.474	9.446	0.343	0.0025	0.0301
Auto/Oil Study***										
1989 Dodge Shadow (TWC)	0.000	4.550	0.480	0.0000	0.0095	0.000	6.730	0.080	0.0000	0.0841
1989 Ford Taurus (TWC)	0.000	7.600	0.150	0.0000	0.0507	0.864	12.710	0.210	0.0041	0.0605
1989 Ford Aerostar (TWC)	3.370	19.280	0.460	0.0073	0.0419	16.180	42.700	1.090	0.0148	0.0392
1989 Toyota Camry (TWC)	0.000	10.380	0.660	0.0000	0.0157	0.000	11.050	0.290	0.0000	0.0381
1989 Pontiac Grand Am (TWC)	0.000	8.800	0.170	0.0000	0.0518	4.050	17.010	0.290	0.0140	0.0587
1989 Oldsmobile Delta 88 (TWC)	4.710	8.910	0.320	0.0147	0.0278	4.610	15.080	0.300	0.0154	0.0503
1989 Ford Mustang (TWC + OX)	1.975	4.750	0.250	0.0079	0.0190	0.000	6.251	0.070	0.0000	0.0893
Average	1.436	9.181	0.356	0.0043	0.0309	3.672	15.933	0.333	0.0069	0.0600
AVERAGE (All vehicles)****	0.646	5.831	0.263	0.0023	0.0194	1.500	9.794	0.259	0.0045	0.0349

5.5% MTBE (EC-1)

Arco 91-02 (Unleaded Reg)**

21 1989 Toyota Camry (TWC)	14.740	3.090	0.149	0.0989	0.0207	8.250	6.790	0.220	0.0375	0.0309
22 1989 Pont. Grand Prix (TWC)	0.000	6.090	0.470	0.0000	0.0130	2.740	4.530	0.277	0.0099	0.0164
23 1989 Ford Taurus (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
24 1989 Dodge Dynasty (TWC)	0.000	0.380	0.188	0.0000	0.0020	0.000	2.310	0.163	0.0000	0.0142
25 1989 Nissan Maxima (TWC)	23.090	10.420	0.597	0.0387	0.0175	6.640	28.210	1.276	0.0052	0.0221
Average****	7.566	3.996	0.281	0.0275	0.0106	3.526	8.368	0.387	0.0105	0.0167

9.0% MTBE

Arco 91-03

35 1988 Pontiac Grand Am (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
35 1988 Pontiac Grand Am (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
40 1987 Nissan 300ZX (TWC)	43.540	13.840	0.430	0.1012	0.0322	24.680	20.180	0.404	0.0611	0.0500
40 1987 Nissan 300ZX (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
41 1988 Toyota 4WD Pickup (TWC)	11.070	5.470	0.144	0.0768	0.0380	7.260	8.530	0.117	0.0621	0.0730
41 1988 Toyota 4WD Pickup (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
42 1988 Toyota Celica (TWC)	9.070	4.530	0.134	0.0676	0.0338	8.120	7.520	0.131	0.0620	0.0574
42 1988 Toyota Celica (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
44 1986 Mercedes 190E (TWC)	26.490	7.710	0.265	0.0999	0.0291	14.280	8.020	0.211	0.0676	0.0380
44 1986 Mercedes 190E (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
45 1989 Lincoln Continental (TWC)	19.870	5.070	0.211	0.0944	0.0241	4.260	5.780	0.134	0.0317	0.0430
45 1989 Lincoln Continental (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
46 1990 Plymouth Acclaim (TWC)	19.980	9.410	0.325	0.0615	0.0290	18.690	8.760	0.230	0.0814	0.0382
46 1990 Plymouth Acclaim (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
47 1984 Toyota Celica (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
47 1984 Toyota Celica (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
49 1987 Oldsmobile Regency (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
56 1982 Nissan Maxima (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
57 1986 Buick Park Avenue (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
57 1986 Buick Park Avenue (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
Average****	6.501	2.302	0.075	0.0251	0.0093	3.865	2.940	0.061	0.0183	0.0150

15.0% MTBE

Arco 91-06 (Test Blend 1)**

350 1990 Ford Taurus (TWC)	0.000	0.000	0.063	0.0000	0.0000	0.000	0.000	0.118	0.0000	0.0000
351 1990 Toyota Camry (TWC)	0.000	0.000	0.375	0.0000	0.0000	0.000	4.920	0.535	0.0000	0.0092
353 1990 Honda Accord (TWC)	51.450	6.270	0.470	0.1095	0.0133	42.940	6.620	0.615	0.0698	0.0108
354 1990 Nissan Stanza (TWC)	46.220	15.360	0.680	0.0680	0.0226	70.690	24.940	1.458	0.0485	0.0171
355 1990 Pontiac Grand Am (TWC)	4.100	2.790	0.105	0.0390	0.0266	10.410	11.330	0.260	0.0400	0.0436
356 1990 Ford Crown Victoria (TWC)	1.180	0.130	0.130	0.0091	0.0010	4.390	3.550	0.125	0.0351	0.0284
357 1990 Plymouth Voyager (TWC)	4.210	0.000	0.085	0.0495	0.0000	5.790	2.350	0.140	0.0414	0.0168
358 1990 Nissan Pickup (TWC)	7.810	5.650	0.190	0.0411	0.0297	13.380	9.940	0.320	0.0418	0.0311
359 1990 Buick LeSabre (TWC)	2.480	2.350	0.075	0.0331	0.0313	N.A.	N.A.	N.A.	N.A.	N.A.
Average	13.050	3.617	0.241	0.0388	0.0138	16.400	7.072	0.397	0.0307	0.0174

Arco 91-06 (Test Blend 2)**

350 1990 Ford Taurus (TWC)	0.000	0.680	0.090	0.0000	0.0076	0.000	4.040	0.135	0.0000	0.0299

351 1990 Toyota Camry (TWC)	0.000	7.290	0.325	0.0000	0.0224	0.000	11.860	0.575	0.0000	0.0206
353 1990 Honda Accord (TWC)	6.000	3.750	0.080	0.0750	0.0469	6.840	4.640	0.140	0.0489	0.0331
354 1990 Nissan Stanza (TWC)	11.450	8.720	0.437	0.0262	0.0200	4.900	21.710	0.912	0.0054	0.0238
355 1990 Pontiac Grand Am (TWC)	4.990	1.040	0.095	0.0525	0.0109	7.500	8.090	0.205	0.0366	0.0395
356 1990 Ford Crown Victoria (TWC)	2.570	2.100	0.185	0.0139	0.0114	3.640	0.000	0.100	0.0364	0.0000
357 1990 Plymouth Voyager (TWC)	6.900	4.550	0.100	0.0690	0.0455	3.790	1.310	0.140	0.0271	0.0094
358 1990 Nissan Pickup (TWC)	8.280	2.240	0.185	0.0448	0.0121	13.120	7.100	0.290	0.0452	0.0245
359 1990 Buick LeSabre (TWC)	2.130	2.620	0.060	0.0355	0.0437	5.080	6.180	0.145	0.0350	0.0426
Average	4.702	3.666	0.173	0.0352	0.0245	4.986	7.214	0.294	0.0261	0.0248
Auto/Oil Study										
1989 Dodge Shadow (TWC)	7.876	6.424	0.220	0.0358	0.0292	16.176	6.792	0.120	0.1348	0.0566
1989 Ford Taurus (TWC)	31.395	7.429	0.230	0.1365	0.0323	29.700	13.233	0.330	0.0900	0.0401
1989 Ford Aerostar (TWC)	30.996	16.031	0.410	0.0756	0.0391	5.151	3.596	1.010	0.0051	0.0036
1989 Toyota Camry (TWC)	40.428	6.516	0.360	0.1123	0.0181	39.970	12.775	0.350	0.1142	0.0365
1989 Pontiac Grand Am (TWC)	26.900	9.000	0.250	0.1076	0.0360	56.112	20.304	0.480	0.1169	0.0423
1989 Oldsmobile Delta 88 (TWC)	37.851	7.030	0.370	0.1023	0.0190	46.995	13.689	0.390	0.1205	0.0351
1989 Ford Mustang (TWC + OX)	28.593	7.128	0.270	0.1059	0.0264	8.001	5.895	0.090	0.0889	0.0655
Average	29.148	8.508	0.301	0.0966	0.0286	28.872	10.898	0.396	0.0958	0.0400
AVERAGE (All vehicles)****	14.552	5.004	0.234	0.0537	0.0218	15.783	8.195	0.359	0.0473	0.0264

*Evaporative MTBE emissions in fuels with 0% MTBE is likely due to carryover from tests done on MTBE blends.
**Evaporative emissions given in mass per mile. ARCO 91-02 and 91-06 converted mass per test data to mass per mile data using the MOBILE3 conversion.
***Only data for fuel A considered.
****Data used in subsequent emission fractions calculations.

09/09/1987

LDGV (TBI -- FTP Data)

	Diurnal Evap (mg/test)			Fraction of THC		Hot Soak Evap (mg/test)			Fraction of THC	
	MTBE*	Benzene	THC (g/test)	MTBE	Benzene	MTBE*	Benzene	THC (g/test)	MTBE	Benzene
0% MTBE										
Arco 91-03 (Unleaded Premium)										
1983 Ford Thunderbird (TWC + OX)	25.070	13.170	8.063	0.0031	0.0016	0.000	7.620	0.185	0.0000	0.0413
Arco 91-06 (Ind. Avg. Baseline)**										
352 1990 Plymouth Sundance (TWC)	0.000	3.840	0.583	0.0000	0.0066	0.000	4.760	0.382	0.0000	0.0125
Auto/Oil Study										
1989 Plymouth Sundance (TWC)***	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
1989 Chevrolet Suburban (TWC)***	7.030	6.790	0.230	0.0306	0.0295	10.080	12.710	0.300	0.0336	0.0424
1985 Ford Tempo (TWC + OX)	13.728	28.171	1.430	0.0096	0.0197	15.088	24.932	0.920	0.0164	0.0271
Average	6.919	11.654	0.553	0.0134	0.0164	8.389	12.547	0.407	0.0167	0.0232
AVERAGE (All vehicles)****	9.166	10.394	2.061	0.0087	0.0115	5.034	10.004	0.357	0.0100	0.0246
9.0% MTBE										
Arco 91-03										
1983 Ford Thunderbird (TWC + OX)****	82.090	8.760	2.972	0.0276	0.0029	25.570	4.760	0.205	0.1248	0.0232
15.0% MTBE										
Arco 91-06 (Test Blend 1)**										
352 1990 Plymouth Sundance (TWC)	12.870	0.000	0.205	0.0628	0.0000	12.490	2.240	0.190	0.0657	0.0118
Arco 91-06 (Test Blend 2)**										
352 1990 Plymouth Sundance (TWC)	1.110	8.110	0.245	0.0045	0.0331	10.880	6.650	0.205	0.0531	0.0324
Auto/Oil Study										
1989 Plymouth Sundance (TWC)	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
1989 Chevrolet Suburban (TWC)	33.205	6.467	0.290	0.1145	0.0223	40.672	11.315	0.310	0.1312	0.0365
1985 Ford Tempo (TWC + OX)	134.332	68.460	1.420	0.0946	0.0482	68.460	21.070	0.700	0.0978	0.0301
Average	55.846	24.976	0.570	0.0697	0.0235	36.377	10.795	0.337	0.0763	0.0222
AVERAGE (All vehicles)****	36.303	16.607	0.432	0.0553	0.0207	26.500	8.255	0.281	0.0696	0.0222

*Evaporative MTBE emissions in fuels with 0% MTBE is likely due to carryover from tests done on MTBE blends.
**Evaporative emissions given in mass per mile. ARCO 91-06 converted mass per test data to mass per mile data using the MOBILE3 conversion.
***Only data for fuel A considered.
****Data used in subsequent emission fractions calculations.

09/04/1987

EXHAUST Catalyst	Fuel	# of Studies	# of Vehicles	Formaldehyde (Percent TOG)	Acetaldehyde	Benzene	1,3-Butadiene	CARB Butadiene	Weighted 1,3-Butadiene CARB THC/TOG***	EPA THC/TOG****
3-WAY	Gasoline	4	38	0.87	0.47	5.56	0.48	0.60	0.55	0.57
3-WAY	5.5% MTBE	1	5	1.72	0.83	2.92	0.61			
3-WAY	9.0% MTBE	1	16*	0.98	0.40	4.16	0.50			
3-WAY	12.5% MTBE	1	11	1.07	0.43	3.17	0.58			
3-WAY	15% MTBE	2	19**	0.72	0.33	4.43	0.60			
3-WAY + OX	Gasoline	7	25	1.37	0.45	2.77	0.16			
3-WAY + OX	9.0% MTBE	1	4	1.46	0.53	2.69	0.28			
3-WAY + OX	12.5% MTBE	1	1	1.05	0.29	1.92	0.16			
3-WAY + OX	15.0% MTBE	1	4	1.23	0.33	3.53	0.25			
OX	Gasoline	8	41	1.39	0.44	3.03	0.28	0.54	0.43	0.44
OX	9.0% MTBE	1	1	0.62	0.04	2.92	0.08			
OX	15% MTBE	1	2	5.87	1.35	3.16	0.46			
OX	16.4% MTBE	1	1	9.00	1.94	1.13	0.15			
NONCAT	Gasoline	3	11	2.69	0.62	2.84	0.73	0.97	0.96	0.98
NONCAT	16.4% MTBE	1	1	1.59	0.34	3.81	0.92			
LDDV	Diesel	2	7	3.91	1.25	2.01	N.A.	0.90	0.90	1.03
HDDV	Diesel	1	2	2.80	0.75	1.02	N.A.	1.36	1.36	1.58
HDGV	Gasoline	1	1	3.10	0.63	10.26	N.A.			

*13 of these vehicles were tested twice in two separate experiments.
**10 vehicles were tested twice, using different 15% MTBE blends.
***CARB THC/TOG correction factors are described in CARB memo "Butadiene emission factors" from K. D. Drachand to Terry McGuire and Peter Venturini, July 17, 1991.
****EPA THC/TOG correction factors are described in EPA memo "Correction factors to convert THC to TOG" from Greg Janssen to Phil Lorang, September 3, 1991.

02/06/1988

Fuel System	Fuel	# of Studies	# of Vehicles	Diurnal MTBE*	Diurnal Benzene	Hot Soak MTBE*	Hot Soak Benzene
Carb.	Gasoline	4	13	1.16	1.50	0.85	2.54
Carb.	9.0% MTBE	1	3	9.86	1.97	12.11	2.38
Carb.	15.0% MTBE	1	7	10.90	1.42	14.93	2.52
Carb.	16.4% MTBE	1	2	8.17	0.72	12.70	1.08
TBI	Gasoline	3	5	1.08	1.44	1.25	3.08
TBI	9.0% MTBE	1	1	2.76	0.29	12.48	2.32
TBI	15.0% MTBE	3	4**	6.91	2.59	8.70	2.77
PFI	Gasoline	4	26	0.30	2.46	0.57	4.43
PFI	5.5% MTBE	1	4	3.44	1.33	1.31	2.09
PFI	9.0% MTBE	1	6	8.36	3.10	6.10	4.99
PFI	15.0% MTBE	3	16***	5.37	2.18	4.92	2.75

1987 Air Toxics Report (Carey, 1987)
   Benzene 1.11 to 1.53% of evaporative emissions for carbureted LDGVs and 0.35-0.46% for fuel injected vehicles.

*Evaporative emissions in fuels with 0% MTBE is likely due to carryover from tests done on MTBE blends.
**1 of these vehicles was tested twice, using different 15% MTBE blends.
***9 of these vehicles were tested twice, using different 15% MTBE blends.

   02/20/1988

LDGV (3-WAY -- FTP Data)

	Formaldehyde			Acetaldehyde			1,3-Butadiene			Benzene		
	0% MTBE TOG Fraction	15% MTBE TOG Fraction	Change	0% MTBE TOG Fraction	15% MTBE TOG Fraction	Change	0% MTBE TOG Fraction	15% MTBE TOG Fraction	Change	0% MTBE TOG Fraction	15% MTBE TOG Fraction	Change
Auto/Oil Study (pooled data)												
1989 Plymouth Sundance (TBI)	0.0048	0.0065	0.3542	0.0048	0.0036	-0.2500	0.0034	0.0036	0.0588	0.0637	0.0636	-0.0016
1989 Dodge Shadow (PFI)	0.0054	0.0053	-0.0185	0.0044	0.0036	-0.1818	0.0063	0.0056	-0.1111	0.0499	0.0508	0.0180
1989 Ford Taurus (PFI)	0.0047	0.0058	0.2340	0.0032	0.0035	0.0938	0.0039	0.0038	-0.0256	0.0482	0.0505	0.0477
1989 Ford Aerostar (PFI)	0.0038	0.0041	0.0789	0.0028	0.003	0.0714	0.0037	0.0036	-0.0270	0.0563	0.0576	0.0231
1989 Toyota Camry (PFI)	0.0026	0.0096	2.6923	0.0028	0.0026	-0.0714	0.0036	0.0036	0.0000	0.0477	0.0452	-0.0524
1989 Honda Accord (Carb)	0.0052	0.0097	0.8654	0.0045	0.0048	0.0667	0.0052	0.0051	-0.0192	0.0388	0.032	-0.1753
1989 Chevrolet Suburban (TBI)	0.0095	0.0110	0.1579	0.0054	0.0055	0.0185	0.0031	0.0036	0.1613	0.0653	0.0666	0.0199
1989 Pontiac Grand Am (PFI)	0.0105	0.0114	0.0857	0.0061	0.0062	0.0164	0.008	0.0081	0.0125	0.0419	0.0434	0.0358
1989 Oldsmobile Delta 88 (SFI)	0.0093	0.0132	0.4194	0.0047	0.0046	-0.0213	0.0056	0.0055	-0.0179	0.0513	0.0557	0.0858
1985 Plymouth Reliant (Carb)	0.0055			0.0020			0.0015			0.0258		
1985 Honda Accord (Carb)	0.0051			0.0028			0.005			0.0483		
AVERAGE			0.5410			-0.0286			0.0515			0.0001

	Formaldehyde					Acetaldehyde					1,3-Butadiene				
	0% MTBE TOG Fraction	15% MTBE B1* TOG Fraction	15% MTBE B2** TOG Fraction	Change B1	Change B2	0% MTBE TOG Fraction	15% MTBE B1* TOG Fraction	15% MTBE B2** TOG Fraction	Change B1	Change B2	0% MTBE TOG Fraction	15% MTBE B1* TOG Fraction	15% MTBE B2** TOG Fraction	Change B1	Change B2
Arco 91-06 (Ind. Avg. Baseline)															
350 1990 Ford Taurus (PFI)	0.0026	0.005	0.0055	0.9231	1.1154	0.0024	0.0027	0.0027	0.1250	0.1250	0.0059	0.0058	0.0059	-0.0169	0.0000
351 1990 Toyota Camry (PFI)	0.0024	0.0031	0.0036	0.2917	0.5000	0.0022	0.0023	0.0025	0.0455	0.1364	0.0082	0.0075	0.0058	-0.0854	-0.2927
352 1990 Plymouth Sundance (TBI)	0.0025	0.0077	0.0067	2.0800	1.6800	0.0016	0.0012	0.0028	-0.2500	0.7500	0.0063	0.0039	0.0043	-0.3810	-0.3175
353 1990 Honda Accord (PFI)	0.0032	0.0085	0.0071	1.6563	1.2188	0.0033	0.0025	0.0024	-0.2424	-0.2727	0.0097	0.0097	0.0074	0.0000	-0.2371
354 1990 Nissan Stanza (PFI)	0.0036	0.0058	0.0048	0.6111	0.3333	0.0023	0.0025	0.0048	0.0870	1.0870	0.0091	0.0075	0.0072	-0.1758	-0.2088
355 1990 Pontiac Grand Am (PFI)	0.0048	0.0072	0.0068	0.5000	0.4167	0.0026	0.0032	0.0021	0.2308	-0.1923	0.01	0.0102	0.0101	0.0200	0.0100
356 1990 Ford Crown Victoria (PFI)	0.0036	0.0049	0.004	0.3611	0.1111	0.002	0.0026	0.0013	0.3000	-0.3500	0.0026	0.0034	0.0031	0.3077	0.1923
357 1990 Plymouth Voyager (PFI)	0.0053	0.005	0.007	-0.0566	0.3208	0.003	0.0069	0.0027	1.3000	-0.1000	0.0054	0.0058	0.0063	0.0741	0.1667
358 1990 Nissan Pickup (PFI)	0.0064	0.0119	0.0114	0.8594	0.7813	0.0038	0.0039	0.0038	0.0263	0.0000	0.0089	0.0077	0.0092	-0.1348	0.0337
359 1990 Buick LeSabre (PFI)	0.0036	0.0075	0.0076	1.0833	1.1111	0.002	0.0025	0.0032	0.2500	0.6000	0.0051	0.0061	0.0052	0.1961	0.0196
AVERAGE	0.0038	0.0067	0.0065	0.8309	0.7588	0.0025	0.0030	0.0028	0.1872	0.1783	0.0071	0.0068	0.0065	-0.0196	-0.0634

	Benzene				
	0% MTBE TOG Fraction	15% MTBE B1* TOG Fraction	15% MTBE B2** TOG Fraction	Change B1	Change B2
Arco 91-06 (Ind. Avg. Baseline)					
350 1990 Ford Taurus (PFI)	0.0569	0.0349	0.04	-0.3866	-0.2970
351 1990 Toyota Camry (PFI)	0.0436	0.0334	0.0579	-0.2339	0.3280
352 1990 Plymouth Sundance (TBI)	0.0806	0.0361	0.0445	-0.5521	-0.4479
353 1990 Honda Accord (PFI)	0.0559	0.0372	0.0398	-0.3345	-0.2880
354 1990 Nissan Stanza (PFI)	0.0735	0.0429	0.0368	-0.4163	-0.4993
355 1990 Pontiac Grand Am (PFI)	0.0583	0.0365	0.0398	-0.3739	-0.3173
356 1990 Ford Crown Victoria (PFI)	0.0382	0.0398	0.0294	0.0419	-0.2304
357 1990 Plymouth Voyager (PFI)	0.0632	0.0237	0.0367	-0.6250	-0.4193
358 1990 Nissan Pickup (PFI)	0.0542	0.0382	0.0434	-0.2952	-0.1993
359 1990 Buick LeSabre (PFI)	0.055	0.047	0.0801	-0.1455	0.4564
AVERAGE	0.0579	0.0370	0.0448	-0.3321	-0.1914

*B1 = Arco reformulated gasoline EC-X, test blend no. 1
**B2 = Arco reformulated gasoline EC-X, test blend no. 2

Change is defined by solving the equation:
TOG frac @ 0%MTBE * (1 + (change/2.7) * Ox) = TOG frac @ 15% MTBE
  where Ox = 2.7

Overall averages (weighted average of the averages)
FORMALDEHYDE	0.6746
ACETALDEHYDE	0.0826
1,3-BUTADIENE	0.0025
BENZENE	-0.1377

02/23/1996

LDGV (3-WAY -- FTP Data)

	Formaldehyde			Acetaldehyde			1,3-Butadiene			Benzene		
	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)												
1990 Ford Probe (PFI)	0.0121	0.0183	0.5124	0.0029	0.0106	2.6552	0.0029	0.0039	0.3448	0.0512	0.0386	-0.2461
1990 Chevy Lumina (TBI)	0.0161	0.0283	0.7578	0.0073	0.0179	1.4521	0.0042	0.0055	0.3095	0.0556	0.0421	-0.2428
1983 Buick Regal (Carb)	0.0073	0.0073	0.0000	0.003	0.0087	1.9000	0.0017	0.0014	-0.1765	0.0346	0.0262	-0.2428
Average	0.0118	0.0180	0.4234	0.0044	0.0124	2.0024	0.0029	0.0036	0.1593	0.0471	0.0356	-0.2439
Auto/Oil (Fuel X/Industry Avg. +10% EtOH, 10 RVP)												
1989 Plymouth Sundance (TBI)	0.0031	0.0064	1.0645	0.0034	0.0103	2.0294	0.0045	0.0045	0.0000	0.0458	0.0484	0.0568
1989 Dodge Shadow (TBI)	0.0054	0.0531	8.8333	0.0021	0.01	3.7619	0.0059	0.0058	-0.0169	0.0514	0.0424	-0.1751
1989 Ford Mustang (PFI)	0.0038	0.0063	0.6579	0.0029	0.0066	1.2759	0.0021	0.0016	-0.2381	0.0225	0.0148	-0.3422
1989 Ford Taurus (PFI)	0.003	0.0045	0.5000	0.0069	0.0096	0.3913	0.0047	0.0038	-0.1915	0.0463	0.0384	-0.1706
1989 Ford Aerostar (PFI)	0.0022	0.0033	0.5000	0.0005	0.01	19.0000	0.0045	0.0053	0.1778	0.0473	0.0528	0.1163
1989 Camry Sedan (PFI)	0.0037	0.0013	-0.6486	0.0173	0.0059	-0.6590	0.003	0.0033	0.1000	0.0378	0.0374	-0.0106
1989 Accord Sedan (Carb)	0.0188	0.0216	0.1489	0.0184	0.0122	-0.3370	0.006	0.0052	-0.1333	0.0361	0.0269	-0.2548
1989 Chevy Suburban (TBI)	0.0071	0.0097	0.3662	0.006	0.0171	1.8500	0.0042	0.0065	0.5476	0.0633	0.0557	-0.1201
1989 Pontiac Grand Am (PFI)	0.0065	0.0107	0.6462	0.0186	0.0121	-0.3495	0.0084	0.0056	-0.3333	0.0401	0.0285	-0.2893
1989 Oldsmobile Delta 88 (PFI)	0.0053	0.0091	0.7170	0.0038	0.0097	1.5526	0.007	0.0055	-0.2143	0.0396	0.0452	0.1414
Average	0.0059	0.0126	1.2785	0.0080	0.0104	2.8516	0.0050	0.0047	-0.0302	0.0430	0.0391	-0.1048
Average (without Shadow)			0.3843									
Average (without Aerostar)						1.0573						
Auto/Oil (Fuel W/Industry Avg. +10% EtOH, 9 RVP)												
1989 Plymouth Sundance (TBI)	0.0031	0.0054	0.7419	0.0034	0.0107	2.1471	0.0045	0.0036	-0.2000	0.0458	0.0621	0.3559
1989 Dodge Shadow (TBI)	0.0054	0.0088	0.6296	0.0021	0.0048	1.2857	0.0059	0.0061	0.0339	0.0514	0.0479	-0.0681
1989 Ford Mustang (PFI)	0.0038	0.0096	1.5263	0.0029	0.0176	5.0690	0.0021	0.0026	0.2381	0.0225	0.0219	-0.0267
1989 Ford Taurus (PFI)	0.003	0.0056	0.8667	0.0069	0.0095	0.3768	0.0047	0.0041	-0.1277	0.0463	0.0351	-0.2419
1989 Ford Aerostar (PFI)	0.0022	0.0265	11.0455	0.0005	0.0129	24.8000	0.0045	0.0059	0.3111	0.0473	0.0498	0.0529
1989 Camry Sedan (PFI)	0.0037	0.0049	0.3243	0.0173	0.0045	-0.7399	0.003	0.0037	0.2333	0.0378	0.0379	0.0026
1989 Accord Sedan (Carb)	0.0188	0.0063	-0.6649	0.0184	0.0119	-0.3533	0.006	0.0054	-0.1000	0.0361	0.0307	-0.1496
1989 Chevy Suburban (TBI)	0.0071	0.0102	0.4366	0.006	0.0147	1.4500	0.0042	0.0042	0.0000	0.0633	0.0516	-0.1848
1989 Pontiac Grand Am (PFI)	0.0065	0.0094	0.4462	0.0186	0.0123	-0.3387	0.0084	0.0065	-0.2262	0.0401	0.0331	-0.1746
1989 Oldsmobile Delta 88 (PFI)	0.0053	0.0113	1.1321	0.0038	0.0239	5.2895	0.007	0.0077	0.1000	0.0396	0.0439	0.1086
Average	0.0059	0.0098	1.6484	0.0080	0.0123	3.8986	0.0050	0.0050	0.0263	0.0430	0.0414	-0.0326
Average (without Aerostar)			0.6043			1.5762						
Auto/Oil (Fuel U/Low ole., low T90, low arom., 10% EtOH, 10 RVP)												
1989 Plymouth Sundance (TBI)	0.0031	0.0061	0.9677	0.0034	0.0126	2.7059	0.0045	0.003	-0.3333	0.0458	0.0491	0.0721
1989 Dodge Shadow (TBI)	0.0054	0.0079	0.4630	0.0021	0.0064	2.0476	0.0059	0.0044	-0.2542	0.0514	0.0374	-0.2724
1989 Ford Mustang (PFI)	0.0038	0.008	1.1053	0.0029	0.0049	0.6897	0.0021	0.0014	-0.3333	0.0225	0.0155	-0.3111
1989 Ford Taurus (PFI)	0.003	0.0048	0.6000	0.0069	0.007	0.0145	0.0047	0.0029	-0.3830	0.0463	0.0259	-0.4406
1989 Ford Aerostar (PFI)	0.0022	0.0033	0.5000	0.0005	0.009	17.0000	0.0045	0.0027	-0.4000	0.0473	0.0529	0.1184
1989 Camry Sedan (PFI)	0.0037	0.0007	-0.8108	0.0173	0.0049	-0.7168	0.003	0.0028	-0.0667	0.0378	0.0302	-0.2011
1989 Accord Sedan (Carb)	0.0188	0.0094	-0.5000	0.0184	0.0065	-0.6467	0.006	0.0036	-0.4000	0.0361	0.0209	-0.4211
1989 Chevy Suburban (TBI)	0.0071	0.0003	-0.9577	0.006	0.003	-0.5000	0.0042	0.0009	-0.7857	0.0633	0.0438	-0.3081
1989 Pontiac Grand Am (PFI)	0.0065	0.0153	1.3538	0.0186	0.0157	-0.1559	0.0084	0.0076	-0.0952	0.0401	0.0319	-0.2045
1989 Oldsmobile Delta 88 (PFI)	0.0053	0.0109	1.0566	0.0038	0.0229	5.0263	0.007	0.0073	0.0429	0.0396	0.0421	0.0631
Average	0.0059	0.0067	0.3778	0.0080	0.0093	2.5465	0.0050	0.0037	-0.3391	0.0430	0.0350	-0.2187
Average (without Aerostar)						0.9405						
Auto/Oil (Fuel T/Low ole., low T90, low arom., 10% EtOH, 9 RVP)												
1989 Plymouth Sundance (TBI)	0.0031	0.0052	0.6774	0.0034	0.0104	2.0588	0.0045	0.004	-0.1111	0.0458	0.0658	0.4367
1989 Dodge Shadow (TBI)	0.0054	0.0028	-0.4815	0.0021	0.0079	2.7619	0.0059	0.0044	-0.2542	0.0514	0.0345	-0.3288
1989 Ford Mustang (PFI)	0.0038	0.0052	0.3684	0.0029	0.0084	1.8966	0.0021	0.0016	-0.2381	0.0225	0.0140	-0.3778
1989 Ford Taurus (PFI)	0.003	0.0082	1.7333	0.0069	0.0117	0.6957	0.0047	0.0036	-0.2340	0.0463	0.0397	-0.1425
1989 Ford Aerostar (PFI)	0.0022	0.004	0.8182	0.0005	0.008	15.0000	0.0045	0.0029	-0.3556	0.0473	0.0440	-0.0698
1989 Camry Sedan (PFI)	0.0037	0.0022	-0.4054	0.0173	0.0049	-0.7168	0.003	0.0021	-0.3000	0.0378	0.0308	-0.1852
1989 Accord Sedan (Carb)	0.0188	0.0068	-0.6383	0.0184	0.0073	-0.6033	0.006	0.0043	-0.2833	0.0361	0.0274	-0.2410
1989 Chevy Suburban (TBI)	0.0071	0.0134	0.8873	0.006	0.0047	-0.2167	0.0042	0.0042	0.0000	0.0633	0.0447	-0.2938
1989 Pontiac Grand Am (PFI)	0.0065	0.0148	1.2769	0.0186	0.0132	-0.2903	0.0084	0.0069	-0.1786	0.0401	0.0280	-0.3017
1989 Oldsmobile Delta 88 (PFI)	0.0053	0.009	0.6981	0.0038	0.0088	1.3158	0.007	0.0056	-0.2000	0.0396	0.0350	-0.1162
Average	0.0059	0.0072	0.4935	0.0080	0.0085	2.1902	0.0050	0.0040	-0.2155	0.0430	0.0364	-0.1620
Average (without Aerostar)						0.7669						

Change is defined by solving the equation:
TOG frac @ 0%EtOH * (1 + (change/3.5) * Ox) = TOG frac @ 10% EtOH
  where Ox = 3.5

LDGV (3-WAY -- FTP Data) Cont.

	Formaldehyde			Acetaldehyde			1,3-Butadiene			Benzene		
	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change
Colorado Department of Health, 1987												
1985 Mercury Marquis (TBI)	0.0077	0.0153	0.9870	0.0048	0.0139	1.8958	N/A	N/A	N/A	0.0268	0.0236	-0.1194
1985 Mercury Marquis (TBI)	0.0071	0.0194	1.7324	0.0056	0.0148	1.6429	N/A	N/A	N/A	0.0193	0.0297	0.5389
1986 Ford Escort (Carb)	0.026	0.0495	0.9038	0.0034	0.0073	1.1471	N/A	N/A	N/A	0.0381	0.0273	-0.2835
1984 Olds Stationwagon (Carb)	0.0105	0.0096	-0.0857	0.0038	0.0072	0.8947	N/A	N/A	N/A	0.0406	0.0326	-0.1970
1984 Buick Skylark (TBI)	0.0173	0.0175	0.0116	0.0061	0.0108	0.7705	N/A	N/A	N/A	0.0740	0.0689	-0.0689
1983 Chev Celebrity (TBI)	0.0276	0.0257	-0.0688	0.0192	0.0141	-0.2656	N/A	N/A	N/A	0.0450	0.0306	-0.3200
Average	0.0160	0.0228	0.5800	0.0072	0.0114	1.0142	N/A	N/A	N/A	0.0406	0.0355	-0.0750

Overall averages (weighted average of the averages)

Formaldehyde	0.4758
Acetaldehyde	1.1369
1,3-Butadiene	-0.1188
Benzene	-0.1299

02/23/1988

	DIURNAL				HOT SOAK			
	0% MTBE TOG Fraction	Benzene MTBE Blend TOG Fraction	Change MTBE Blend/ 0% MTBE	Average of Absolutes	0% MTBE TOG Fraction	Benzene MTBE Blend TOG Fraction	Change MTBE Blend/ 0% MTBE	Average of Absolutes
LDGV -- CARB								
9.0% MTBE								
Arco 91-03								
37 1981 Olsmobile Delta 88 (TWC)	0.0076	0.0071	-6.58		0.0280	0.0177	-36.79	
37 1981 Olsmobile Delta 88 (TWC)	0.0076	N.A.			0.0280	N.A.		
39 1984 Honda Accord (TWC)	0.0380	0.0293	-22.89		0.0599	0.0372	-37.90	
39 1984 Honda Accord (TWC)	0.0380	N.A.			0.0599	N.A.		
48 1985 Ford Mustang (TWC)	N.A.	N.A.			N.A.	N.A.		
48 1985 Ford Mustang (TWC)	N.A.	N.A.			N.A.	N.A.		
58 1989 Toyota Tercel (TWC)	N.A.	N.A.			N.A.	N.A.		
58 1989 Toyota Tercel (TWC)	N.A.	N.A.			N.A.	N.A.		
59 1982 Buick Regal (TWC)	N.A.	N.A.			N.A.	N.A.		
43 1985 Chevrolet Camaro (TWC + OX)	0.0152	0.0266	75.00		0.0117	0.0166	41.88	
Average	0.0106	0.0063	15.18	34.82	0.0188	0.0072	-10.93	38.85
15.0% MTBE								
Auto/Oil Study								
1989 Honda Accord (TWC)	0.0091	0.0118	29.67		0.0223	0.0194	-13.00	
1985 Plymouth Reliant (TWC)	0.0130	0.0126	-3.08		0.0263	0.0229	-12.93	
1985 Honda Accord (TWC)	0.0328	0.0270	-17.68		0.0284	0.0242	-14.79	
1985 Chevrolet Impala (TWC + OX)	0.0069	0.0058	-15.94		0.0293	0.0289	-1.37	
1984 Pontiac Grand Prix (TWC + OX)	0.0082	0.0097	18.29		0.0270	0.0274	1.48	
1984 Chevrolet Suburban (OX)	0.0079	0.0084	6.33		0.0274	0.0261	-4.74	
1983 Ford F-150 (OX)	0.0282	0.0238	-15.60		0.0319	0.0273	-14.42	
Average	0.0152	0.0142	0.28	15.23	0.0275	0.0252	-8.54	8.96
16.4% MTBE								
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)								
1977 Mercury Marquis (OX)	0.0052	0.0050	-3.85		0.0063	0.0086	36.51	
1974 Chevy Impala (Non-cat)	0.0094	0.0093	-1.06		0.0179	0.0130	-27.37	
Average	0.0073	0.0072	-2.45	2.45	0.0121	0.0108	4.57	31.94

LDGV -- TBI

9.0% MTBE

Arco 91-03

1983 Ford Thunderbird (TWC + OX)	0.0016	0.0029	81.25		0.0413	0.0232	-43.83	

15.0% MTBE

Arco 91-06 (Test Blend 1)

352 1990 Plymouth Sundance (TWC)	0.0066	0.0000	-100.00		0.0125	0.0118	-5.60	

Arco 91-06 (Test Blend 2)

352 1990 Plymouth Sundance (TWC)	0.0066	0.0331	401.52		0.0125	0.0324	159.20	

Auto/Oil Study

1989 Plymouth Sundance (TWC)	N.A.	N.A.			N.A.	N.A.		
1989 Chevrolet Suburban (TWC)	0.0295	0.0223	-24.41		0.0424	0.0365	-13.92	
1985 Ford Tempo (TWC + OX)	0.0197	0.0482	144.67		0.0271	0.0301	11.07	
Average	0.0164	0.0235	60.13	84.54	0.0232	0.0222	-1.42	12.49
AVERAGE (All vehicles)	0.0125	0.0207	105.44	167.65	0.0189	0.0222	37.69	47.45

LDGV -- PFI

5.5% MTBE (EC-1)

Arco 91-02 (Unleaded Reg)**

21 1989 Toyota Camry (TWC)	0.0286	0.0207	-27.62		0.0585	0.0309	-47.18	
22 1989 Pont. Grand Prix (TWC)	0.0021	0.0130	519.05		0.0270	0.0164	-39.26	
23 1989 Ford Taurus (TWC)	N.A.	N.A.			N.A.	N.A.		
24 1989 Dodge Dynasty (TWC)	0.0103	0.0020	-80.58		0.0102	0.0142	39.22	
25 1989 Nissan Maxima (TWC)	0.0207	0.0175	-15.46		0.0342	0.0221	-35.38	
Average	0.0123	0.0106	98.85	160.68	0.0260	0.0167	-20.65	40.26

9.0% MTBE

Arco 91-03

35 1988 Pontiac Grand Am (TWC)	N.A.	N.A.			N.A.	N.A.		
35 1988 Pontiac Grand Am (TWC)	N.A.	N.A.			N.A.	N.A.		
40 1987 Nissan 300ZX (TWC)	N.A.	N.A.			N.A.	N.A.		
40 1987 Nissan 300ZX (TWC)	0.0342	0.0322	-5.85		0.0647	0.0500	-22.72	
41 1988 Toyota 4WD Pickup (TWC)	N.A.	N.A.			N.A.	N.A.		
41 1988 Toyota 4WD Pickup (TWC)	0.0346	0.0380	9.83		0.0703	0.0730	3.84	
42 1988 Toyota Celica (TWC)	N.A.	N.A.			N.A.	N.A.		

42 1988 Toyota Celica (TWC)	0.0250	0.0338	35.20		0.0746	0.0574	-23.06
44 1986 Mercedes 190E (TWC)	N.A.	N.A.			N.A.	N.A.	
44 1986 Mercedes 190E (TWC)	0.0428	0.0291	-32.01		0.0478	0.0380	-20.50
45 1989 Lincoln Continental (TWC)	N.A.	N.A.			N.A.	N.A.	
45 1989 Lincoln Continental (TWC)	0.0309	0.0241	-22.01		0.0426	0.0430	0.94
46 1990 Plymouth Acclaim (TWC)	N.A.	N.A.			N.A.	N.A.	
46 1990 Plymouth Acclaim (TWC)	0.0292	0.0290	-0.68		0.0300	0.0382	27.33
47 1984 Toyota Celica (TWC)	N.A.	N.A.			N.A.	N.A.	
47 1984 Toyota Celica (TWC)	N.A.	N.A.			N.A.	N.A.	
49 1987 Oldsmobile Regency (TWC)	N.A.	N.A.			N.A.	N.A.	
56 1982 Nissan Maxima (TWC)	N.A.	N.A.			N.A.	N.A.	
57 1986 Buick Park Avenue (TWC)	N.A.	N.A.			N.A.	N.A.	
57 1986 Buick Park Avenue (TWC)	N.A.	N.A.			N.A.	N.A.	
Average	0.0098	0.0093	-2.59	17.60	0.0165	0.0150	-5.69	16.40

15.0% MTBE

Arco 91-06 (Test Blend 1)

350 1990 Ford Taurus (TWC)	0.0224	0.0000	-100.00		0.0221	0.0000	-100.00
351 1990 Toyota Camry (TWC)	0.0187	0.0000	-100.00		0.0178	0.0092	-48.31
353 1990 Honda Accord (TWC)	0.0102	0.0133	30.39		0.0157	0.0108	-31.21
354 1990 Nissan Stanza (TWC)	0.0260	0.0226	-13.08		0.0270	0.0171	-36.67
355 1990 Pontiac Grand Am (TWC)	0.0243	0.0266	9.47		0.0394	0.0436	10.66
356 1990 Ford Crown Victoria (TWC)	0.0178	0.0010	-94.38		0.0266	0.0284	6.77
357 1990 Plymouth Voyager (TWC)	0.0080	0.0000	-100.00		0.0380	0.0168	-55.79
358 1990 Nissan Pickup (TWC)	0.0256	0.0297	16.02		0.0343	0.0311	-9.33
359 1990 Buick LeSabre (TWC)	0.0116	0.0313	169.83		0.0496	N.A.	
Average	0.0183	0.0138	-20.20	70.35	0.0301	0.0174	-32.99	37.34

Arco 91-06 (Test Blend 2)

350 1990 Ford Taurus (TWC)	0.0224	0.0076	-66.07		0.0221	0.0299	35.29
351 1990 Toyota Camry (TWC)	0.0187	0.0224	19.79		0.0178	0.0206	15.73
353 1990 Honda Accord (TWC)	0.0102	0.0469	359.80		0.0157	0.0331	110.83
354 1990 Nissan Stanza (TWC)	0.0260	0.0200	-23.08		0.0270	0.0238	-11.85
355 1990 Pontiac Grand Am (TWC)	0.0243	0.0109	-55.14		0.0394	0.0395	0.25
356 1990 Ford Crown Victoria (TWC)	0.0178	0.0114	-35.96		0.0266	0.0000	-100.00
357 1990 Plymouth Voyager (TWC)	0.0080	0.0455	468.75		0.0380	0.0094	-75.26
358 1990 Nissan Pickup (TWC)	0.0256	0.0121	-52.73		0.0343	0.0245	-28.57
359 1990 Buick LeSabre (TWC)	0.0116	0.0437	276.72		0.0496	0.0426	-14.11
Average	0.0183	0.0245	99.12	150.89	0.0301	0.0248	-7.52	43.55

Auto/Oil Study

1989 Dodge Shadow (TWC)	0.0095	0.0292	207.37		0.0841	0.0566	-32.70
1989 Ford Taurus (TWC)	0.0507	0.0323	-36.29		0.0605	0.0401	-33.72

1989 Ford Aerostar (TWC)	0.0419	0.0391	-6.68		0.0392	0.0036	-90.82	
1989 Toyota Camry (TWC)	0.0157	0.0181	15.29		0.0381	0.0365	-4.20	
1989 Pontiac Grand Am (TWC)	0.0518	0.0360	-30.50		0.0587	0.0423	-27.94	
1989 Oldsmobile Delta 88 (TWC)	0.0278	0.0190	-31.65		0.0503	0.0351	-30.22	
1989 Ford Mustang (TWC + OX)	0.0190	0.0264	38.95		0.0893	0.0655	-26.65	
Average	0.0309	0.0286	22.35	52.39	0.0600	0.0400	-35.18	35.18
AVERAGE (All vehicles)	0.0218	0.0218	34.67	94.32	0.0384	0.0264	-24.08	39.04

02/23/1988

	DIURNAL						HOT SOA					
	0% EtOH TOG Fraction	Benzene EtOH Blend TOG Fraction	MTBE Blend* TOG Fraction	Change EtOH Blend/ 0% EtOH	Change MTBE Blend/ 0% EtOH	Change EtOH Blend/ MTBE Blend	0% EtOH TOG Fraction	Benzene EtOH Blend TOG Fraction	MTBE Blend* TOG Fraction	Change EtOH Blend/ 0% EtOH	Change MTBE Blend/ 0% EtOH	Change EtOH Blend/ MTBE Blend
LDGV -- CARB												
E10												
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)												
1983 Buick Regal (TWC)	0.0121	0.0093	0.0110	-0.2314	-0.0909	-0.1545	0.0313	0.0256	0.0283	-0.1821	-0.0958	-0.0954
1977 Mercury Marquis (Oxy)	0.0052	0.0064	0.0050	0.2308	-0.0385	0.2800	0.0063	0.0042	0.0086	-0.3333	0.3651	-0.5116
1974 Chevy Impala (Noncat)	0.0094	0.0119	0.0093	0.2660	-0.0106	0.2796	0.0179	0.0122	0.0130	-0.3184	-0.2737	-0.0615
Average	0.0089	0.0092	0.0084	0.0884	-0.0467	0.1350	0.0185	0.0140	0.0166	-0.2780	-0.0015	-0.2229
Auto/Oil (Fuel X/Industry + 10% EtOH, 10 RVP)												
1989 Accord Sedan (TWC)	0.0098	0.0101	0.0107	0.0306	0.0918	-0.0561	0.0282	0.0193	0.0171	-0.3156	-0.3936	0.1287
Auto/Oil (Fuel W/Industry + 10% EtOH, 9 RVP)												
1989 Accord Sedan (TWC)	0.0098	0.0121	0.0107	0.2347	0.0918	0.1308	0.0282	0.0207	0.0171	-0.2660	-0.3936	0.2105
Auto/Oil (Fuel U/low ole., low T90, low arom., 10% EtOH, 10 RVP)												
1989 Accord Sedan (TWC)	0.0098	0.0113	0.0107	0.1531	0.0918	0.0561	0.0282	0.0193	0.0171	-0.3156	-0.3936	0.1287
Auto/Oil (Fuel T/low ole., low T90, low arom., 10% EtOH, 9 RVP)												
1989 Accord Sedan (TWC)	0.0098	0.0139	0.0107	0.4184	0.0918	0.2991	0.0282	0.0255	0.0171	-0.0957	-0.3936	0.4912
Overall Avg. (Carb)	0.0094	0.0107	0.0097	0.1574	0.0325	0.1193	0.0240	0.0181	0.0169	-0.2610	-0.2256	0.0415
LDGV -- TBI												
8.1% EtOH												
Stump et al., 1990 (MSERB Oxyfuels Study)												
CO 665 1988 Chevy Corsica (TWC)	0.0032	0.0068	0.0036	1.1250	0.1364	0.8700	0.0209	0.0319	0.0046	0.5263	-0.7792	5.9117
CO322 1987 Chevy Corsica (TWC)	0.0134	0.0102	0.0066	-0.2388	-0.5099	0.5532	0.0217	0.0276	0.0104	0.2719	-0.5214	1.6578
Average	0.0083	0.0085	0.0051	0.4431	-0.1868	0.7116	0.0213	0.0298	0.0075	0.3991	-0.6503	3.7847
E10												
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)												
1990 Chevy Lumina (TWC)	0.0185	0.0097	0.0303	-0.4757	0.6378	-0.6799	0.0308	0.0368	0.0144	0.1948	-0.5325	1.5556
Auto/Oil (Fuel X/Industry + 10% EtOH, 10 RVP)												
1989 Plymouth Sundance (TWC)	0.0086	0.0141	0.0143	0.6395	0.6570	-0.0105	0.0572	0.0382	0.0361	-0.3322	-0.3698	0.0596
1989 Dodge Shadow (TWC)	0.0114	0.0060	0.0098	-0.4737	-0.1404	-0.3878	0.0456	0.0274	0.0522	-0.3991	0.1447	-0.4751
1989 Chevy Suburban (TWC)	0.0247	0.0259	0.0234	0.0486	-0.0526	0.1068	0.0454	0.0356	0.0339	-0.2159	-0.2544	0.0517
Average	0.0149	0.0153	0.0158	0.0715	0.1547	-0.0971	0.0494	0.0337	0.0407	-0.3157	-0.1598	-0.1213
Auto/Oil (Fuel W/Industry + 10% EtOH, 9 RVP)												
1989 Plymouth Sundance (TWC)	0.0086	0.0268	0.0143	2.1163	0.6570	0.8807	0.0572	0.0354	0.0361	-0.3811	-0.3698	-0.0180
1989 Dodge Shadow (TWC)	0.0114	0.0147	0.0098	0.2895	-0.1404	0.5000	0.0456	0.0394	0.0522	-0.1360	0.1447	-0.2452
1989 Chevy Suburban (TWC)	0.0247	0.0249	0.0234	0.0081	-0.0526	0.0641	0.0454	0.0324	0.0339	-0.2863	-0.2544	-0.0428
Average	0.0149	0.0221	0.0158	0.8046	0.1547	0.4816	0.0494	0.0357	0.0407	-0.2678	-0.1598	-0.1020
Auto/Oil (Fuel U/low ole., low T90, low arom., 10% EtOH, 10 RVP)												
1989 Plymouth Sundance (TWC)	0.0086	0.0241	0.0143	1.8023	0.6570	0.6912	0.0572	0.0356	0.0361	-0.3776	-0.3698	-0.0125
1989 Dodge Shadow (TWC)	0.0114	0.0065	0.0098	-0.4298	-0.1404	-0.3367	0.0456	0.0385	0.0522	-0.1557	0.1447	-0.2625
1989 Chevy Suburban (TWC)	0.0247	0.0196	0.0234	-0.2065	-0.0526	-0.1624	0.0454	0.0367	0.0339	-0.1916	-0.2544	0.0842
Average	0.0149	0.0167	0.0158	0.3887	0.1547	0.0640	0.0494	0.0369	0.0407	-0.2417	-0.1598	-0.0636

Auto/Oil (Fuel T/low ole., low T90, low arom., 10% EtOH, 9 RVP)

1989 Plymouth Sundance (TWC)	0.0086	0.0168	0.0143	0.9535	0.6570	0.1789	0.0572	0.0441	0.0361	-0.2290	-0.3698	0.2233
1989 Dodge Shadow (TWC)	0.0114	0.0110	0.0098	-0.0351	-0.1404	0.1224	0.0456	0.0415	0.0522	-0.0899	0.1447	-0.2050
1989 Chevy Suburban (TWC)	0.0247	0.0353	0.0234	0.4291	-0.0526	0.5085	0.0454	0.0437	0.0339	-0.0374	-0.2544	0.2910
Average	0.0149	0.0210	0.0158	0.4492	0.1547	0.2700	0.0494	0.0431	0.0407	-0.1188	-0.1598	0.1031
Overall Avg. (TBI)	0.0143	0.0168	0.0154	0.3702	0.1414	0.1932	0.0444	0.0363	0.0345	-0.1226	-0.0898	0.0630

LDGV -- PFI

E10

Warner-Selph and Smith, 1991 (EPA-460/3-91-02)

1990 Ford Probe (TWC)	0.0422	0.0458	0.0167	0.0853	-0.6043	1.7425	0.0450	0.0417	0.0379	-0.0733	-0.1578	0.1003

Auto/Oil (Fuel X/Industry + 10% EtOH, 10 RVP)

1989 Ford Mustang (TWC)	0.0074	0.0130	0.0122	0.7568	0.6486	0.0656	0.0692	0.0491	0.0527	-0.2905	-0.2384	-0.0683
1989 Ford Taurus (TWC)	0.0395	0.0396	0.0344	0.0025	-0.1304	0.1528	0.0527	0.0316	0.0375	-0.4004	-0.2894	-0.1562
1989 Ford Aerostar (TWC)	0.0401	0.0355	0.0328	-0.1147	-0.1833	0.0840	0.0384	0.0329	0.0323	-0.1432	-0.1602	0.0202
1989 Camry Sedan (TWC)	0.0130	0.0152	0.0358	0.1692	1.7538	-0.5754	0.0475	0.0361	0.0368	-0.2400	-0.2263	-0.0177
1989 Pontiac Grand Am (TWC)	0.0260	0.0254	0.0452	-0.0231	0.7385	-0.4381	0.0526	0.0431	0.0432	-0.1806	-0.1797	-0.0012
1989 Oldsmobile Delta 88 (TWC)	0.0196	0.0226	0.0302	0.1531	0.5383	-0.2504	0.0410	0.0309	0.0341	-0.2463	-0.1695	-0.0925
Average	0.0243	0.0252	0.0317	0.1573	0.5609	-0.1603	0.0502	0.0373	0.0394	-0.2502	-0.2106	-0.0526

Auto/Oil (Fuel W/Industry + 10% EtOH, 9 RVP)

1989 Ford Mustang (TWC)	0.0074	0.0292	0.0122	2.9459	0.6486	1.3934	0.0692	0.0442	0.0527	-0.3613	-0.2384	-0.1613
1989 Ford Taurus (TWC)	0.0395	0.0416	0.0344	0.0532	-0.1304	0.2111	0.0527	0.0334	0.0375	-0.3662	-0.2894	-0.1081
1989 Ford Aerostar (TWC)	0.0401	0.0398	0.0328	-0.0075	-0.1833	0.2153	0.0384	0.0410	0.0323	0.0677	-0.1602	0.2713
1989 Camry Sedan (TWC)	0.0130	0.0357	0.0358	1.7462	1.7538	-0.0028	0.0475	0.0000	0.0368	-1.0000	-0.2263	-1.0000
1989 Pontiac Grand Am (TWC)	0.0260	0.0367	0.0452	0.4115	0.7385	-0.1881	0.0526	0.0525	0.0432	-0.0019	-0.1797	0.2167
1989 Oldsmobile Delta 88 (TWC)	0.0196	0.0292	0.0302	0.4898	0.5383	-0.0315	0.0410	0.0413	0.0341	0.0073	-0.1695	0.2129
Average	0.0243	0.0354	0.0317	0.9399	0.5609	0.2662	0.0502	0.0354	0.0394	-0.2757	-0.2106	-0.0948

Auto/Oil (Fuel U/low ole., low T90, low arom., 10% EtOH, 10 RVP)

1989 Ford Mustang (TWC)	0.0074	0.0179	0.0122	1.4189	0.6486	0.4672	0.0692	0.0466	0.0527	-0.3266	-0.2384	-0.1157
1989 Ford Taurus (TWC)	0.0395	0.0347	0.0344	-0.1215	-0.1304	0.0102	0.0527	0.0332	0.0375	-0.3700	-0.2894	-0.1135
1989 Ford Aerostar (TWC)	0.0401	0.0357	0.0328	-0.1097	-0.1833	0.0901	0.0384	0.0185	0.0323	-0.5182	-0.1602	-0.4264
1989 Camry Sedan (TWC)	0.0130	0.0279	0.0358	1.1462	1.7538	-0.2207	0.0475	0.0455	0.0368	-0.0421	-0.2263	0.2381
1989 Pontiac Grand Am (TWC)	0.0260	0.0480	0.0452	0.8462	0.7385	0.0619	0.0526	0.0433	0.0432	-0.1768	-0.1797	0.0035
1989 Oldsmobile Delta 88 (TWC)	0.0196	0.0265	0.0302	0.3520	0.5383	-0.1211	0.0410	0.0431	0.0341	0.0512	-0.1695	0.2658
Average	0.0243	0.0318	0.0317	0.5887	0.5609	0.0479	0.0502	0.0384	0.0394	-0.2304	-0.2106	-0.0247

Auto/Oil (Fuel T/low ole., low T90, low arom., 10% EtOH, 9 RVP)

1989 Ford Mustang (TWC)	0.0074	0.0127	0.0122	0.7162	0.6486	0.0410	0.0692	0.0429	0.0527	-0.3801	-0.2384	-0.1860
1989 Ford Taurus (TWC)	0.0395	0.0364	0.0344	-0.0785	-0.1304	0.0597	0.0527	0.0364	0.0375	-0.3093	-0.2894	-0.0280
1989 Ford Aerostar (TWC)	0.0401	0.0382	0.0328	-0.0474	-0.1833	0.1664	0.0384	0.0369	0.0323	-0.0391	-0.1602	0.1442
1989 Camry Sedan (TWC)	0.0130	0.0293	0.0358	1.2538	1.7538	-0.1816	0.0475	0.0337	0.0368	-0.2905	-0.2263	-0.0830
1989 Pontiac Grand Am (TWC)	0.0260	0.0326	0.0452	0.2538	0.7385	-0.2788	0.0526	0.0512	0.0432	-0.0266	-0.1797	0.1866
1989 Oldsmobile Delta 88 (TWC)	0.0196	0.0327	0.0302	0.6684	0.5383	0.0846	0.0410	0.0431	0.0341	0.0512	-0.1695	0.2658
Average	0.0243	0.0303	0.0317	0.4611	0.5609	-0.0181	0.0502	0.0407	0.0394	-0.1657	-0.2106	0.0499
Overall Avg. (PFI)	0.0250	0.0313	0.0311	0.5187	0.5143	0.1023	0.0500	0.0381	0.0393	-0.2242	-0.2085	-0.0253
AVG., TBI and PFI	0.0210	0.0259	0.0252	0.4630	0.3745	0.1364	0.0479	0.0374	0.0375	-0.1861	-0.2241	0.1985

02/23/1988

*Auto/Oil data an average for 9 and 8 RVP blends (fuels MM and N2)

| | Formaldehyde | | | Acetaldehyde | | | 1,3-Butadiene | | | Benzene | | |
	0% MTBE TOG Fraction	15% MTBE TOG Fraction	Change	0% MTBE TOG Fraction	15% MTBE TOG Fraction	Change	0% MTBE TOG Fraction	15% MTBE TOG Fraction	Change	0% MTBE TOG Fraction	15% MTBE TOG Fraction	Change
LDGV (3-WAY+OX -- FTP Data)												
Auto/Oil Study												
1989 Ford Mustang (SFI)	0.0112	0.0175	0.5625	0.0039	0.0051	0.3077	0.0026	0.0025	-0.0385	0.0256	0.0290	0.1328
1985 Chevrolet Impala (Carb)	0.015	0.0107	-0.2867	0.0029	0.0024	-0.1724	0.0031	0.0033	0.0645	0.0376	0.0398	0.0585
1984 Pontiac Grand Prix (Carb)	0.0055	0.0089	0.6182	0.0021	0.0026	0.2381	0.0013	0.0012	-0.0769	0.0276	0.0296	0.0725
1985 Ford Tempo (TBI)	0.0103	0.0121	0.1748	0.0047	0.0032	-0.3191	0.0032	0.0032	0.0000	0.0435	0.0427	-0.0184
AVERAGE	0.0105	0.0123	0.2672	0.0034	0.0033	0.0136	0.0026	0.0026	-0.0127	0.0336	0.0353	0.0613
LDGV (OX-CAT --FTP Data)												
Auto/Oil Study												
1984 Chevrolet Suburban (Carb)	0.0391	0.0581	0.4859	0.0157	0.0183	0.1656	0.0033	0.0031	-0.0606	0.0351	0.0319	-0.0912
1983 Ford F-150 (Carb)	0.0231	0.0592	1.5628	0.007	0.0088	0.2571	0.0043	0.0061	0.4186	0.0300	0.0313	0.0433
AVERAGE	0.0311	0.0587	1.0244	0.0114	0.0136	0.2114	0.0038	0.0046	0.1790	0.0326	0.0316	-0.0239
LDGV (NON-CAT --FTP Data)												
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)												
1974 Chevy Impala (Carb)	0.0101	0.0159	0.5743	0.0023	0.0034	0.4783	0.0073	0.0092	0.2603	0.0507	0.0381	-0.2485
16.4% Adjusted to 15%		0.0154	0.5256		0.0033	0.4377		0.0090	0.2382		0.0392	-0.2275

Change is defined by solving the equation:
TOG frac @ 0%MTBE * (1 + (change/2.7) * Ox) = TOG frac @ 15% MTBE
  where Ox = 2.7

02/23/1988

| | Formaldehyde | | | Acetaldehyde | | | 1,3-Butadiene | | | Benzene | | |
	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change	0% EtOH TOG Fraction	10% EtOH TOG Fraction	Change
LDGV (3-WAY+OX -- FTP Data)												
Colorado Department of Health, 1987												
1981 Chev Caprice (Carb)	0.0153	0.0188	0.2288	0.0053	0.0119	1.2453	N/A	N/A	N/A	0.0618	0.0321	-0.4806
LDGV (OX-CAT --FTP Data)												
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)												
1977 Mercury Marquis	0.0346	0.0399	0.1532	0.0119	0.0201	0.6891	0.0016	0.0015	-0.0625	0.0189	0.0119	-0.3704
Colorado Department of Health, 1987												
1976 Dodge Cornet Wgn (Carb)	0.0056	0.0092	0.6429	0.0028	0.0117	3.1786	N/A	N/A	N/A	0.0439	0.0386	-0.1207
1977 Cadillac Seville (PFI)	0.0733	0.0673	-0.0819	0.0209	0.0369	0.7656	N/A	N/A	N/A	0.0344	0.0262	-0.2384
1978 Lincoln Cntinentl (Carb)	0.0111	0.0092	-0.1712	0.0024	0.0130	4.4167	N/A	N/A	N/A	0.0351	0.0293	-0.1652
1977 Chev MntCarlo (Carb)	0.0118	0.0147	0.2458	0.0042	0.0119	1.8333	N/A	N/A	N/A	0.0430	0.0264	-0.3860
1978 VW Rabbit (Carb)	0.0096	0.0133	0.3854	0.0022	0.0084	2.8182	N/A	N/A	N/A	0.0349	0.0190	-0.4556
1978 Chev Impala (Carb)	0.0293	0.0404	0.3788	0.0081	0.0173	1.1358	N/A	N/A	N/A	0.0260	0.0221	-0.1500
1978 Chevy MntCarlo (Carb)	0.0278	0.0377	0.3561	0.0055	0.0093	0.6909	N/A	N/A	N/A	0.0276	0.0205	-0.2572
Average	0.0241	0.0274	0.2509	0.0066	0.0155	2.1199	N/A	N/A	N/A	0.0350	0.0260	-0.2533
AVERAGE			0.2400			1.9609			-0.0625			-0.2663
LDGV (NON-CAT --FTP Data)												
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)												
1974 Chevy Impala (Carb)	0.0101	0.0097	-0.0396	0.0023	0.0032	0.3913	0.0073	0.0082	0.1233	0.0507	0.0400	-0.2110
Colorado Department of Health, 1987												
1974 Ford Squire (Carb)	0.0060	0.0055	-0.0833	0.0033	0.0083	1.5152	N/A	N/A	N/A	0.0316	0.0258	-0.1835
1974 Ford Pinto (Carb)	0.0033	0.0053	0.6061	0.0023	0.0055	1.3913	N/A	N/A	N/A	0.0296	0.0192	-0.3514
1973 VW Fastback (EFI)	0.0089	0.0092	0.0337	0.0038	0.0100	1.6316	N/A	N/A	N/A	0.0248	0.0234	-0.0565
1979 Honda Civic (Carb)	0.0097	0.0097	0.0000	0.0029	0.0052	0.7931	N/A	N/A	N/A	0.0258	0.0194	-0.2481
Average	0.0070	0.0074	0.1391	0.0031	0.0073	1.3328	N/A	N/A	N/A	0.0280	0.0220	-0.2099
AVERAGE	0.0076	0.0079	0.1034	0.0029	0.0064	1.1445			0.1233	0.0325	0.0256	-0.2101

Change is defined by solving the equation:
TOG frac @ 10%EtOH * (1 + (change/3.5) * Ox) = TOG frac @ 10%EtOH
  where Ox = 3.5

02/23/1988

Actual Versus Predicted Benzene Comparisons

Study	Catalyst	Fuel	# of Vehicles	RVP, psi	Fuel Specifications Aromatics	Benzene	Olefins	% Benzene Adjustment	Benzene (% Total) Predicted (w/adj.)*	Benzene (% Total) Predicted (no adj.)*	Actual	% Change (w/adj.)	% Change (no adj.)
Gasoline/MTBE Blends													
ARCO 91-02	TWC	Unleaded Reg.	5	8.8	33.6	1.8	12.3	0.0000	5.61	5.61	5.24	-6.55	-6.55
ARCO 91-03	TWC	Unleaded Prem.	16	8.9	41.5	1.9	6.8	0.0000	6.45	6.45	5.42	-16.03	-16.03
ARCO 91-06	TWC	Ind. Avg.	10	8.6	34.4	1.0	9.7	0.0000	5.15	5.15	5.79	12.50	12.50
Auto/Oil (TB#1)	TWC	A	7	8.7	32.0	1.5	9.2	0.0000	5.25	5.25	5.71	8.82	8.82
Auto/Oil (TB#6)	TWC	A	10	8.7	32.0	1.5	9.2	0.0000	5.25	5.25	4.30	-18.05	-18.05
Colorado Dept. of Health, 1987	TWC	UL Base	6	10.7	30.4	1.7	14.4	0.0000	5.20	5.20	4.06	-21.88	-21.88
ARCO 91-02	TWC	5.5% MTBE	5	7.6	33.6	0.8	12.0	-0.0505	4.68	4.93	2.92	-37.66	-40.81
ARCO 91-03	TWC	9.0% MTBE	16**	7.6	32.9	1.2	11.0	-0.0826	4.71	5.13	4.16	-11.67	-18.97
ARCO 91-03	TWC	12.5% MTBE	13	8.1	23.6	1.0	12.5	-0.1148	3.61	4.08	3.17	-12.25	-22.32
ARCO 91-06	TWC	15% MTBE (Blend 1)	10	7.9	21.2	0.7	5.1	-0.1377	3.14	3.64	3.70	17.83	1.60
ARCO 91-06	TWC	15% MTBE (Blend 2)	10	6.7	21.6	0.7	5.5	-0.1377	3.17	3.68	4.48	41.14	21.70
Auto/Oil (TB#1)	TWC	15% MTBE (Pooled)**	9	8.4-8.8	18.0-47.6(34.43)	1.28-1.52(1.43)	3.3-21.8	-0.1377	4.69	5.44	5.17	10.22	-4.96
Auto/Oil (TB#6)	TWC	15% MTBE (N2)	10	8.8	21.4	1.3	4.9	-0.1377	3.51	4.07	3.49	-0.46	-14.17
Auto/Oil (TB#6)	TWC	15% MTBE (MM)	10	8.0	22.2	1.4	5.4	-0.1377	3.63	4.21	3.34	-8.05	-20.71
Urban, 1980b, 1980c (EPA-460/3-80-004, 005)	TWC+Oxy	Gasoline	2	8.9	28.0	2.0	8.0	0.0000	2.92	2.92	3.11	6.54	6.54
Sigsby, et al., 1987 (46 car study)	TWC+Oxy	Gasoline	8	8.8-12.2	31.23-44.2	1.76-1.96	9.33-10.54	0.0000					
Stump et al., 1989 (MSERB Low Temp.)	TWC+Oxy	Gasoline	6	11.5	30.3	0.9	4.6	0.0000	2.33	2.33	2.67	14.68	14.68
Stump et al., unpublished (MSERB High Temp.)	TWC+Oxy	Gasoline	1	10.2	37.9	1.4	10.3	0.0000	3.19	3.19	1.65	-48.20	-48.20
Warner-Selph and DeVita, 1989 (Carb Toxics Study)	TWC+Oxy	Gasoline	1	8.4	37.0	2.4	7.9	0.0000	3.83	3.83	4.76	24.25	24.25
ARCO 91-03	TWC+Oxy	Unleaded Prem.	3	8.9	41.5	1.9	6.8	0.0000	3.77	3.77	3.83	1.58	1.58
Auto/Oil (TB#1)	TWC+Oxy	A	4	8.7	32.0	1.5	9.2	0.0000	2.85	2.85	3.36	17.82	17.82
Colorado Dept. of Health, 1987	TWC+Oxy	UL Base	1	10.7	30.4	1.7	14.4	0.0000	2.85	2.85	6.18	116.72	116.72
ARCO 91-03	TWC+Oxy	9% MTBE	4***	7.6	32.9	1.2	11.0	0.0368	2.81	2.71	2.69	-4.23	-0.71
ARCO 91-03	TWC+Oxy	12.5% MTBE	1	8.1	23.6	1.0	12.5	0.0511	2.04	1.94	1.92	-5.84	-1.03
Auto/Oil (TB#1)	TWC+Oxy	15% MTBE (Pooled)**	4	8.4-8.8	18.0-47.6(34.43)	1.28-1.52(1.43)	3.3-21.8	0.0613	3.15	2.97	3.53	12.00	18.87
Urban, 1980a (EPA-460/3-80-003)	Oxy	Gasoline	3	8.9	28.0	2.0	8.0	0.0000	3.96	3.96	4.16	4.97	4.97
Sigsby et al., 1987 (46 car study)	Oxy	Gasoline	24	8.8-12.2	31.23-44.2	1.76-1.96	9.33-10.54	0.0000					
Smith, 1981 (EPA-460/3-81-024)	Oxy	Gasoline	7****	8.9	28.0	2.0	8.0	0.0000	3.96	3.96	4.27	7.75	7.75
Stump et al., 1989 (MSERB Low Temp.)	Oxy	Gasoline	1	11.5	30.3	0.9	4.6	0.0000	3.30	3.30	3.95	19.59	19.59
Auto/Oil (TB#1)	Oxy	A	2	8.7	32.0	1.5	9.2	0.0000	4.02	4.02	3.25	-19.22	-19.22
ARCO 91-03	Oxy	Unleaded Prem.	1	8.9	41.5	1.9	6.8	0.0000	5.52	5.52	2.96	-46.42	-46.42
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	Oxy	Gasoline	1	9.5	35.1	1.6	6.8	0.0000	4.49	4.49	1.89	-57.88	-57.88
Colorado Dept. of Health, 1987	Oxy	UL Base	7	10.7	30.4	1.7	14.4	0.0000	3.97	3.97	3.50	-11.73	-11.73
ARCO 91-03	Oxy	9.0% MTBE	1	7.6	32.9	1.2	11.0	-0.0143	3.82	3.88	2.92	-23.58	-24.68
Auto/Oil (TB#1)	Oxy	15% MTBE (Pooled)**	2	8.4-8.8	18.0-47.6(34.43)	1.28-1.52(1.43)	3.3-21.8	-0.0239	4.16	4.26	3.16	-24.00	-25.82
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	Oxy	16.4% MTBE	1	9.5	20.4	1.2	5.4	-0.0261	2.32	2.39	1.13	-51.37	-52.64
Urban, 1980a, 1981 (EPA-460/3-80-003)	Non-cat	Gasoline	5	8.9	28.0	2.0	8.0	0.0000	3.96	3.96	3.33	-15.97	-15.97
Sigsby et al., 1987 (46 car study)	Non-cat	Gasoline	5	8.8-12.2	31.23-44.2	1.76-1.96	9.33-10.54	0.0000					
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	Non-cat	Gasoline	1	9.5	35.1	1.6	6.8	0.0000	4.49	4.49	5.07	12.99	12.99
Colorado Dept. of Health, 1987	Non-cat	UL Base	4	10.7	30.4	1.7	14.4	0.0000	3.97	3.97	2.80	-29.38	-29.38
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	Non-cat	16.4% MTBE	1	9.5	20.4	1.2	5.4	-0.2485	1.79	2.39	3.81	112.47	59.67
EtOH													
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	TWC	10% EtOH	3	10.2	22.8	1.4	7.3	-0.1299	3.69	4.24	3.56	-3.46	-16.00
Auto/Oil (TB#6)	TWC	X	10	9.6	27.2	1.5	8.1	-0.1299	4.15	4.77	3.91	-5.86	-18.09
Auto/Oil (TB#6)	TWC	W	10	9.0	29.0	1.4	8.1	-0.1299	4.25	4.88	4.14	-2.57	-15.23
Auto/Oil (TB#6)	TWC	U	10	9.6	19.1	1.4	3.1	-0.1299	3.40	3.91	3.50	2.97	-10.41
Auto/Oil (TB#6)	TWC	T	10	9.3	18.1	1.4	3.6	-0.1299	3.31	3.81	3.64	9.87	-4.41
Colorado Dept. of Health, 1987	TWC	10% EtOH	6	10.1	25.4	1.3	14.7	-0.1299	3.85	4.43	3.55	-7.84	-19.81

Stump et al., 1990 (MSERB Oxyfuels Study)	TWC	8.1% EtOH	2	10.5	34.9	1.3	9.5	-0.1052	4.81	4.18	4.17	-13.39	-0.34
Colorado Dept. of Health, 1987	TWC+Oxy	10% EtOH	1	10.1	25.4	1.3	14.7	-0.4806	1.16	2.23	3.21	176.84	43.79
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	Oxy	10% EtOH	1	10.2	22.8	1.4	7.3	-0.2633	2.04	2.77	1.19	-41.75	-57.09
Colorado Dept. of Health, 1987	Oxy	10% EtOH	7	10.1	25.4	1.3	14.7	-0.2633	2.21	3.00	2.60	17.46	-13.47
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	Non-cat	10% EtOH	1	10.2	22.8	1.4	7.3	-0.2101	2.19	2.77	4.00	82.62	44.25
Colorado Dept. of Health, 1987	Non-cat	10% EtOH	4	10.1	25.4	1.3	14.7	-0.2101	2.37	3.00	2.20	-7.30	-26.78

*The exhaust benzene fractions were calculated using the following equations:
   TWC Bz%HC = [1.077+0.7732*(volume % benzene) + 0.0987*(volume % aromatics - volume % benzene)]*(1 + % benzene adjustment)
   TWC+Oxy Bz%HC = [0.6796*(volume % benzene) + 0.0681*(volume % aromatics) - 0.3468]*(1 + % benzene adjustment)
   other Bz%HC = 0.8551*(volume % benzene) + 0.12198*(volume % aromatics) - 1.1626]*(1 + % benzene adjustment)

**Vehicles were tested for several 15% MTBE blends, and data was pooled. A range of fuel specifications is given here, with mean values in parentheses.

***Some vehicles were tested twice, in two separate experiments.

****Two sets of measurements were taken for each vehicle.

02/23/1987

Actual Versus Predicted Benzene Comparisons

| Study | Fuel System | Fuel | # of Vehicles | Fuel Specifications | | | | | Diurnal Benzene (% Total) | | Hot Soak Benzene (% Total) | |
				RVP, psi	Aromatics	Benzene	Paraffins	Olefins	Predicted*	Actual	Predicted**	Actual
Arco 91-03	Carb	Gasoline	6	8.9	41.5	1.9	50.5	6.8	1.26	2.03	1.39	3.32
Auto/Oil	Carb	Gasoline	7	8.5-8.9	20.0-47.8	0.52-1.53	34.5-76.8	3.2-22.3	0.34-1.06	1.52	0.38-1.17	2.75
Stump et al., unpublished (MSERB High Temp.)	Carb	Gasoline	1	10.2	37.93	1.39	48.62	10.33	0.77	0.75	0.87	1.08
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	Carb	Gasoline	2	9.5	35.1	1.6	58.1	6.8	0.98	0.73	1.10	1.21
Arco 91-03	Carb	9.0% MTBE	3	7.6	32.9	1.2	41.1	11	0.86	1.97	0.93	2.38
Auto Oil	Carb	15.0% MTBE	7	8.4-8.8	18.0-46.7	1.28-1.52	19.4-59.7	3.3-21.8	0.76-0.95	1.42	0.83-1.03	2.52
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	Carb	16.4% MTBE	2	9.45	20.4	1.24	56.8	6.4	0.66	0.72	0.72	1.08
Arco 91-03	TBI	Gasoline	1	8.9	41.5	1.9	50.5	6.8	1.26	0.16	1.39	4.13
Arco 91-06	TBI	Gasoline	1	8.6	34.4	1	49.4	9.7	0.69	0.66	0.75	1.25
Auto/Oil	TBI	Gasoline	3	8.5-8.9	20.0-47.8	0.52-1.53	34.5-76.8	3.2-22.3	0.34-1.06	2.46	0.38-1.17	3.47
Arco 91-03	TBI	9.0% MTBE	1	7.6	32.9	1.2	41.1	11	0.86	0.29	0.93	2.32
Arco 91-06 (Blend 1)	TBI	15.0% MTBE	1	7.9	21.2	0.7	60.4	5.1	0.46	0.00	0.50	1.18
Arco 91-06 (Blend 2)	TBI	15.0% MTBE	1	6.7	21.6	0.7	61.2	5.5	0.53	3.31	0.57	3.24
Auto/Oil	TBI	15.0% MTBE	3	8.4-8.8	18.0-46.7	1.28-1.52	19.4-59.7	3.3-21.8	0.76-0.95	3.53	0.83-1.03	3.33
Arco 91-02	PFI	Gasoline	5	8.8	33.6	1.8	43.7	12.3	1.20	1.54	1.33	3.25
Arco 91-03	PFI	Gasoline	6	8.9	41.5	1.9	50.5	6.8	1.26	3.28	1.39	5.50
Arco 91-06	PFI	Gasoline	9	8.6	34.4	1	49.4	9.7	0.69	1.83	0.75	3.01
Auto/Oil***	PFI	Gasoline	7	8.7	32	1.53	58.8	9.2	1.04	3.09	1.14	6.00
Arco 91-02	PFI	5.5% MTBE	5	7.6	20.9	0.8	50.7	12	0.59	1.33	0.64	2.09
Arco 91-03	PFI	9.0% MTBE	6	7.6	32.9	1.2	41.1	11	0.86	3.10	0.93	4.99
Arco 91-06 (Blend 1)	PFI	15.0% MTBE	9	7.9	21.2	0.7	60.4	5.1	0.46	1.38	0.50	1.96
Arco 91-06 (Blend 2)	PFI	15.0% MTBE	9	6.7	21.6	0.7	61.2	5.5	0.53	2.45	0.57	2.48
Auto/Oil	PFI	15.0% MTBE	7	8.4-8.8	18.0-46.7	1.28-1.52	19.4-59.7	3.3-21.8	0.76-0.95	2.86	0.83-1.03	4.00

*Predictions obtained using diurnal evaporative emissions equation from SDSB's reformulated gasoline NPRM:
 Diurnal Benzene = [(1.3758-(0.0579*(Weight % Oxygen/2.0)-(0.080274*RVP)]*(Volume % Benzene)

**Predictions obtained using hot soak evaporative emissions equation from SDSB's reformulated gasoline NPRM:
 Hot Soak Benzene = [(1.4448-(0.0684*(Weight % Oxygen/2.0)-(0.080274*RVP)]*(Volume % Benzene)

***Only data for fuel A considered.

02/23/1988

Actual Versus Predicted Benzene Comparisons

Study	Fuel System	Fuel	# of Vehicles	RVP, psi	Fuel Specifications Aromatics	Benzene	Olefins	Diurnal Benzene (% Total) Predicted*	Actual	% Change	Hot Soak Benzene (% Total) Predicted**	Actual	% Change
Auto/Oil	Carb	A (Indust. Avg.	1	8.7	32.0	1.5	9.2	1.02	0.98	-3.56	1.12	0.91	-18.72
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	Carb	Gasoline	3	9.5	35.1	1.6	6.8	0.98	0.92	-6.23	1.09	1.40	28.26
Auto/Oil	Carb	X	1	9.6	27.2	1.5	8.1	0.91	1.01	11.26	1.01	1.93	90.85
Auto/Oil	Carb	W	1	9.0	29.0	1.4	8.1	0.91	1.21	32.29	1.01	2.07	104.69
Auto/Oil	Carb	U	1	9.6	19.1	1.4	3.1	0.85	1.13	33.37	0.94	1.93	104.48
Auto/Oil	Carb	T	1	9.3	18.1	1.4	3.6	0.88	1.39	57.78	0.98	2.55	160.86
Auto/Oil	TBI	A (Indust. Avg.	3	8.7	32.0	1.5	9.2	1.02	1.49	46.64	1.12	4.94	341.22
Stump et al., 1990 (MSERB Oxyfuels Study)	TBI	8.1% EtOH	2	10.5	34.9	1.3	9.5	0.68	0.85	24.61	0.77	2.98	286.78
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	TBI	10% EtOH	1	10.2	22.8	1.4	7.3	0.76	0.97	28.07	0.85	3.68	332.67
Auto/Oil	TBI	X	3	9.6	27.2	1.5	8.1	0.91	1.53	68.55	1.01	3.37	233.25
Auto/Oil	TBI	W	3	9.0	29.0	1.4	8.1	0.91	2.21	141.62	1.01	3.57	253.02
Auto/Oil	TBI	U	3	9.6	19.1	1.4	3.1	0.85	1.67	97.11	0.94	3.69	290.96
Auto/Oil	TBI	T	3	9.3	18.1	1.4	3.6	0.88	2.10	138.38	0.98	4.31	340.90
Auto/Oil	PFI	A (Indust. Avg.	6	8.7	32.0	1.5	9.2	1.02	2.43	139.14	1.12	5.02	348.36
Warner-Selph and Smith, 1991 (EPA-460/3-91-02)	PFI	10% EtOH	1	10.2	22.8	1.4	7.3	0.76	4.58	504.72	0.85	4.17	390.29
Auto/Oil	PFI	X	6	9.6	27.2	1.5	8.1	0.91	2.52	177.61	1.01	3.73	268.85
Auto/Oil	PFI	W	6	9.0	29.0	1.4	8.1	0.91	3.54	287.03	1.01	3.54	250.06
Auto/Oil	PFI	U	6	9.6	19.1	1.4	3.1	0.85	3.18	275.34	0.94	3.84	306.85
Auto/Oil	PFI	T	6	9.3	18.1	1.4	3.6	0.88	3.03	243.95	0.98	4.07	316.35

*Predictions obtained using diurnal evaporative emissions equation from SDSB's reformulated gasoline NPRM:
 Diurnal Benzene = [1.3758-(0.080274*RVP)]*(Volume % Benzene)

**Predictions obtained using hot soak evaporative emissions equation from SDSB's reformulated gasoline NPRM:
 Hot Soak Benzene = [(1.4448-(0.080274*RVP)]*(Volume % Benzene)

02/23/1988

13-BUTAD

Vehicle class/ technology	Baseline (gasoline)(1) Exhaust	Evap	Winter oxygenates(2) Exhaust	Evap	10% Ethanol(3) Exhaust	Evap	Reformulated gasoline CY 1995-1999(4) Exhaust	Evap	CY 2000+(5) Exhaust	Evap	California NMOG standards (exhaust only)(6) 0.3900	0.2500	TLEV 0.1250	LEV 0.0750	ULEV 0.0400	ZEV 0.0000
LDGV																
3-way																
CARB	0.0057	0.0000	0.0056	0.0000	0.0050	0.0000	0.0056	0.0000	0.0056	0.0000	0.0056	0.0056	0.0056	0.0056	0.0056	0.0000
TBI	0.0057	0.0000	0.0056	0.0000	0.0050	0.0000	0.0056	0.0000	0.0056	0.0000	0.0056	0.0056	0.0056	0.0056	0.0056	0.0000
PFI	0.0057	0.0000	0.0056	0.0000	0.0050	0.0000	0.0056	0.0000	0.0056	0.0000	0.0056	0.0056	0.0056	0.0056	0.0056	0.0000
3-way + ox																
CARB	0.0044	0.0000	0.0043	0.0000	0.0041	0.0000	0.0044	0.0000	0.0044	0.0000	0.0044	0.0044	0.0044	0.0044	0.0044	0.0000
TBI	0.0044	0.0000	0.0043	0.0000	0.0041	0.0000	0.0044	0.0000	0.0044	0.0000	0.0044	0.0044	0.0044	0.0044	0.0044	0.0000
PFI	0.0044	0.0000	0.0043	0.0000	0.0041	0.0000	0.0044	0.0000	0.0044	0.0000	0.0044	0.0044	0.0044	0.0044	0.0044	0.0000
ox cat																
CARB	0.0044	0.0000	0.0052	0.0000	0.0041	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
TBI	0.0044	0.0000	0.0052	0.0000	0.0041	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
PFI	0.0044	0.0000	0.0052	0.0000	0.0041	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
non-cat																
CARB	0.0098	0.0000	0.0121	0.0000	0.0110	0.0000	0.0115	0.0000	0.0115	0.0000	0.0115	0.0115	0.0115	0.0115	0.0115	0.0000
LDGT																
3-way																
CARB	0.0057	0.0000	0.0056	0.0000	0.0050	0.0000	0.0056	0.0000	0.0056	0.0000	0.0056	0.0056	0.0056	0.0056	0.0056	0.0000
TBI	0.0057	0.0000	0.0056	0.0000	0.0050	0.0000	0.0056	0.0000	0.0056	0.0000	0.0056	0.0056	0.0056	0.0056	0.0056	0.0000
PFI	0.0057	0.0000	0.0056	0.0000	0.0050	0.0000	0.0056	0.0000	0.0056	0.0000	0.0056	0.0056	0.0056	0.0056	0.0056	0.0000
3-way + ox																
CARB	0.0044	0.0000	0.0043	0.0000	0.0041	0.0000	0.0044	0.0000	0.0044	0.0000	0.0044	0.0044	0.0044	0.0044	0.0044	0.0000
TBI	0.0044	0.0000	0.0043	0.0000	0.0041	0.0000	0.0044	0.0000	0.0044	0.0000	0.0044	0.0044	0.0044	0.0044	0.0044	0.0000
PFI	0.0044	0.0000	0.0043	0.0000	0.0041	0.0000	0.0044	0.0000	0.0044	0.0000	0.0044	0.0044	0.0044	0.0044	0.0044	0.0000
ox cat																
CARB	0.0044	0.0000	0.0052	0.0000	0.0041	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
TBI	0.0044	0.0000	0.0052	0.0000	0.0041	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
PFI	0.0044	0.0000	0.0052	0.0000	0.0041	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
non-cat																
CARB	0.0098	0.0000	0.0121	0.0000	0.0110	0.0000	0.0115	0.0000	0.0115	0.0000	0.0115	0.0115	0.0115	0.0115	0.0115	0.0000
HDGV																
TWC	0.0057	0.0000	0.0056	0.0000	0.0050	0.0000	0.0056	0.0000	0.0056	0.0000	0.0056	0.0056	0.0056	0.0056	0.0056	0.0000
non-cat	0.0098	0.0000	0.0121	0.0000	0.0110	0.0000	0.0115	0.0000	0.0115	0.0000	0.0115	0.0115	0.0115	0.0115	0.0115	0.0000
LDDV	0.0103	0.0000														
LDDT	0.0103	0.0000														
HDDV	0.0158	0.0000														

Footnotes given on the following page.

Footnotes:

(1) For baseline gasoline, the LDGV 3-way fraction was based on FTP data from 55 in-use vehicles tested by CARB.
THC as measured by the FID in these studies was converted to TOG using a single TOG/THC conversion factor of 1.0125, which is consistent with what is used in MOBILE 4.1.
This was done because the fractions are applied to MOBILE 4.1 TOG.

The LDGV 3-way + ox fraction was assumed to equal the LDGV ox cat fraction.

The LDGV ox cat fraction was based on FTP data from 7 in-use vehicles tested by CARB.
A TOG/THC conversion factor of 1.0170 was used.

The LDGV non cat fraction was based on FTP data from 16 in-use vehicles tested by CARB.
A TOG/THC conversion factor of 1.0333 was used.

LDGTs are assumed to have the same fractions as LDGVs.

The LDDV fraction was based on FTP data from two vehicles tested by CARB. A TOG/THC conversion factor of 1.0490 was used.
LDDTs are assumed to have the same fractions as LDDVs.

The HDDV fraction was based on transient data from one engine tested by CARB. A TOG/THC conversion factor of 1.0342 was used.

The HDGV non-cat and 3-way fractions were assumed to equal those for LDGV, due to lack of data. A TOG/THC conversion factor of 1.0358 was used.

(2) For the winter oxygenate program, it is assumed that 15% MTBE (2.7 weight % O2) will be the major oxygenate used in the CO non-attainment areas.
Changes in the 1,3-butadiene TOG fraction from 0% to 15% MTBE were estimated based on vehicle data. (As with baseline gasoline, MOBILE4.1 TOG/THC conversion factors
were used to calculate 1,3-butadiene TOG fractions. The conversion factors for baseline gasoline by catalyst type were each multiplied by 1.0197 to estimate
TOG/THC conversion factors by catalyst type for 15% MTBE blends.) The baseline gasoline fractions were multiplied by the following to estimate fractions
for 15% MTBE: 0.9798 for 3-way, 0.9873 for 3-way + ox, 1.1790 for ox cat, and 1.2382 for non- cat. These numbers were estimated using data from Auto/Oil and
Arco 91-06 for 3-way, Auto/Oil for 3-way+ox and ox cat, and the 1990 SwRI report for non-cat.

(3) Changes in the 1,3-butadiene TOG fraction from 0% to 10% ethanol were estimated based on vehicle data. (As with baseline gasoline, MOBILE4.1 TOG/THC conversion factors
were used to calculate 1,3-butadiene TOG fractions. The conversion factors for baseline gasoline by catalyst type were each multiplied by 1.0232 to estimate
TOG/THC conversion factors by catalyst type for 10% ethanol.) The baseline gasoline fractions were multiplied by the following to estimate fractions
for 10% ethanol: 0.8812 for 3-way, 0.9375 for 3-way + ox, 0.9375 for ox cat, and 1.1233 for non- cat. These numbers were estimated using data from Auto/Oil,
and the 1990 SwRI report for 3-way, the 1990 SwRI report for ox cat, and the 1990 SwRI report for non-cat. Due to lack of data, 3-way+ox was assumed to equal ox cat.

(4) The composition of reformulated gasoline in CY1995-1999 is assumed to be 2% oxygen, 1.0% benzene and 25% aromatics.
The percent changes in the 1,3-butadiene TOG fractions from 0% to 15% MTBE (2.7 wt% O2) were multiplied by 2/2.7, the ratio of oxygen contents by weight for
reformulated gasoline and 15% MTBE, to obtain changes in the 1,3-butadiene TOG fractions with reformulated gasoline.

(5) For reformulated gasoline in CY 2000+, the fraction of 1,3-butadiene is assumed to remain the same relative to CY1995-1999.
However, the mass of TOG will be reduced. As a result, the mass of 1,3-butadiene is assumed to be reduced proportionately to TOG.

(6) The 1,3-butadiene fractions for the California vehicles are assumed to be those for reformulated gasoline. As a result, the mass of 1,3-butadiene is assumed
to be reduced proportionately to TOG.

Remaining CAAA provisions not included in this table are the clean fuel fleet program and the California pilot program. These will not be considered.

03/02/1988

ACETALDE

Vehicle class/ technology	Baseline (gasoline)(1) Exhaust	Evap	Winter oxygenates(2) Exhaust	Evap	10% Ethanol(3) Exhaust	Evap	Reformulated gasoline CY 1995-1999(4) Exhaust	Evap	CY 2000+(5) Exhaust	Evap	California NMOG standards (exhaust only)(6) 0.3900	0.2500	TLEV 0.1250	LEV 0.0750	ULEV 0.0400	ZEV 0.0000
LDGV																
3-way																
CARB	0.0047	0.0000	0.0051	0.0000	0.0100	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
TBI	0.0047	0.0000	0.0051	0.0000	0.0100	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
PFI	0.0047	0.0000	0.0051	0.0000	0.0100	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
3-way + ox																
CARB	0.0045	0.0000	0.0046	0.0000	0.0101	0.0000	0.0045	0.0000	0.0045	0.0000	0.0045	0.0045	0.0045	0.0045	0.0045	0.0000
TBI	0.0045	0.0000	0.0046	0.0000	0.0101	0.0000	0.0045	0.0000	0.0045	0.0000	0.0045	0.0045	0.0045	0.0045	0.0045	0.0000
PFI	0.0045	0.0000	0.0046	0.0000	0.0101	0.0000	0.0045	0.0000	0.0045	0.0000	0.0045	0.0045	0.0045	0.0045	0.0045	0.0000
ox cat																
CARB	0.0044	0.0000	0.0053	0.0000	0.0130	0.0000	0.0051	0.0000	0.0051	0.0000	0.0051	0.0051	0.0051	0.0051	0.0051	0.0000
TBI	0.0044	0.0000	0.0053	0.0000	0.0130	0.0000	0.0051	0.0000	0.0051	0.0000	0.0051	0.0051	0.0051	0.0051	0.0051	0.0000
PFI	0.0044	0.0000	0.0053	0.0000	0.0130	0.0000	0.0051	0.0000	0.0051	0.0000	0.0051	0.0051	0.0051	0.0051	0.0051	0.0000
non-cat																
CARB	0.0062	0.0000	0.0089	0.0000	0.0133	0.0000	0.0082	0.0000	0.0082	0.0000	0.0082	0.0082	0.0082	0.0082	0.0082	0.0000
LDGT																
3-way																
CARB	0.0047	0.0000	0.0051	0.0000	0.0100	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
TBI	0.0047	0.0000	0.0051	0.0000	0.0100	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
PFI	0.0047	0.0000	0.0051	0.0000	0.0100	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
3-way + ox																
CARB	0.0045	0.0000	0.0046	0.0000	0.0101	0.0000	0.0045	0.0000	0.0045	0.0000	0.0045	0.0045	0.0045	0.0045	0.0045	0.0000
TBI	0.0045	0.0000	0.0046	0.0000	0.0101	0.0000	0.0045	0.0000	0.0045	0.0000	0.0045	0.0045	0.0045	0.0045	0.0045	0.0000
PFI	0.0045	0.0000	0.0046	0.0000	0.0101	0.0000	0.0045	0.0000	0.0045	0.0000	0.0045	0.0045	0.0045	0.0045	0.0045	0.0000
ox cat																
CARB	0.0044	0.0000	0.0053	0.0000	0.0130	0.0000	0.0051	0.0000	0.0051	0.0000	0.0051	0.0051	0.0051	0.0051	0.0051	0.0000
TBI	0.0044	0.0000	0.0053	0.0000	0.0130	0.0000	0.0051	0.0000	0.0051	0.0000	0.0051	0.0051	0.0051	0.0051	0.0051	0.0000
PFI	0.0044	0.0000	0.0053	0.0000	0.0130	0.0000	0.0051	0.0000	0.0051	0.0000	0.0051	0.0051	0.0051	0.0051	0.0051	0.0000
non-cat																
CARB	0.0062	0.0000	0.0089	0.0000	0.0133	0.0000	0.0082	0.0000	0.0082	0.0000	0.0082	0.0082	0.0082	0.0082	0.0082	0.0000
HDGV																
TWC	0.0047	0.0000	0.0051	0.0000	0.0100	0.0000	0.0050	0.0000	0.0050	0.0000	0.0050	0.0050	0.0050	0.0050	0.0050	0.0000
non-cat	0.0063	0.0000	0.0091	0.0000	0.0135	0.0000	0.0083	0.0000	0.0083	0.0000	0.0083	0.0083	0.0083	0.0083	0.0083	0.0000
LDDV	0.0125	0.0000														
LDDT	0.0125	0.0000														
HDDV	0.0075	0.0000														

Footnotes given on the following page.

Footnotes:

(1) For baseline gasoline, the LDGV 3-way fraction was based on FTP data from 38 vehicles tested in four studies (Arco 91-02, Arco 91-03, Arco 91-06, and Auto/Oil).
     THC as measured by the FID in these studies was converted to TOG using a single TOG/THC conversion factor of 1.0125, which is consistent with what is used in MOBILE 4.1.
     This was done because the fractions are applied to MOBILE 4.1 TOG.

     The LDGV 3-way + ox fraction was based on FTP data from 25 vehicles tested in eight studies (SwRI 80-004, SwRI 80-005, MSERB 46 car study, MSERB low temp study,
     MSERB high temp study, 1989 CARB toxics study, Arco 91-03, and Auto/Oil). A TOG/THC conversion factor of 1.0175 was used.

     The LDGV ox cat fraction was based on FTP data from 41 vehicles tested in eight studies (SwRI 80-003, SwRI 79-007, MSERB 46 car study, SwRI 81-024,
     MSERB low temp study, Auto/Oil study, Arco 91-03, and 1990 SwRI report). A TOG/THC conversion factor of 1.0170 was used.

     The LDGV non cat fraction was based on FTP data from ll vehicles tested in four studies (SwRI 81-020, SwRI 80-003, MSERB 46 car study, and 1990 SwRI report).
     A TOG/THC conversion factor of 1.0333 was used.

     LDGTs are assumed to have the same fractions as LDGVs.

     The LDDV fraction was based on FTP data from seven vehicles tested in two studies (SwRI 76-034 and SwRI 79-007). A TOG/THC conversion factor of 1.0490 was used.
     LDDTs are assumed to have the same fractions as LDDVs.

     The HDDV fraction was based on 13-mode data from two engines tested in one study (SwRI 79-007). A TOG/THC conversion factor of 1.0342 was used.

     The HDGV non-cat fraction was based on 13-mode data from one engine tested in one study (SwRI 79-007). A TOG/THC conversion factor of 1.0358 was used.
     The HDGV 3-way fraction is assumed to equal the LDGV 3-way fraction.

(2) For the winter oxygenate program, it is assumed that 15% MTBE (2.7 weight % O2) will be the major oxygenate used in the CO non-attainment areas.
     Changes in the acetaldehyde TOG fraction from 0% to 15% MTBE were estimated based on vehicle data. (As with baseline gasoline, MOBILE4.1 TOG/THC conversion factors
     were used to calculate acetaldehyde TOG fractions. The conversion factors for baseline gasoline by catalyst type were each multiplied by 1.0197 to estimate
     TOG/THC conversion factors by catalyst type for 15% MTBE blends.) The baseline gasoline fractions were multiplied by the following to estimate fractions
     for 15% MTBE: 1.0826 for 3-way, 1.0136 for 3-way + ox, 1.2114 for ox cat, and 1.4377 for non- cat. These numbers were estimated using data from Auto/Oil and
     Arco 91-06 for 3-way, Auto/Oil for 3-way+ox and ox cat, and the 1990 SwRI report for non-cat.

(3) Changes in the acetaldehyde TOG fraction from 0% to 10% ethanol were estimated based on vehicle data. (As with baseline gasoline, MOBILE4.1 TOG/THC conversion factors
     were used to calculate acetaldehyde TOG fractions. The conversion factors for baseline gasoline by catalyst type were each multiplied by 1.0232 to estimate
     TOG/THC conversion factors by catalyst type for 10% ethanol.) The baseline gasoline fractions were multiplied by the following to estimate fractions
     for 10% ethanol: 2.1369 for 3-way, 2.2453 for 3-way + ox, 2.9609 for ox cat, and 2.1445 for non- cat. These numbers were estimated using data from Auto/Oil,
     1990 SwRI report, and CDH data for 3-way, CDH data for 3-way+ox, 1990 SwRI report and CDH data for ox cat, and the 1990 SwRI report and CDH data for non-cat.

(4) The composition of reformulated gasoline in CY1995-1999 is assumed to be 2% oxygen, 1.0% benzene and 25% aromatics.
     The percent changes in the acetaldehyde TOG fractions from 0% to 15% MTBE (2.7 wt% O2) were multiplied by 2/2.7, the ratio of oxygen contents by weight for
     reformulated gasoline and 15% MTBE, to obtain changes in the acetaldehyde TOG fractions with reformulated gasoline.

(5) For reformulated gasoline in CY 2000+, the fraction of acetaldehyde is assumed to remain the same relative to CY1995-1999.
     However, the mass of TOG will be reduced. As a result, the mass of acetaldehyde is assumed to be reduced proportionately to TOG.

(6) The acetaldehyde fractions for the California vehicles are assumed to be those for reformulated gasoline. As a result, the mass of acetaldehyde is assumed
     to be reduced proportionately to TOG.

Remaining CAAA provisions not included in this table are the clean fuel fleet program and the California pilot program. These will not be considered.   03/02/1988

BENZENET

Vehicle class/ technology	Baseline (gasoline) (1) Exhaust(2)	Diurnal(3)	Hot Soak(3)	Winter oxygenates (4) Exhaust	Diurnal	Hot Soak	10% Ethanol (5) Exhaust	Diurnal	Hot Soak	CY 1995-1999 (6) Exhaust	Diurnal	Hot Soak	CY 2000+ (7) Exhaust	Diurnal	Hot Soak	California NMOG standards (exhaust only)(8) 0.3900	0.2500	TLEV 0.1250	LEV 0.0750	ULEV 0.0400	ZEV 0.0000
LDGV																					
3-way																					
CARB	0.0527	0.0104	0.0114	0.0396	0.0000	0.0000	0.0486	0.0084	0.0093	0.0422	0.0067	0.0073	0.0422	0.0069	0.0075	0.0422	0.0422	0.0422	0.0422	0.0422	0.0000
TBI	0.0527	0.0104	0.0114	0.0396	0.0000	0.0000	0.0486	0.0084	0.0093	0.0422	0.0067	0.0073	0.0422	0.0069	0.0075	0.0422	0.0422	0.0422	0.0422	0.0422	0.0000
PFI	0.0527	0.0104	0.0114	0.0396	0.0000	0.0000	0.0486	0.0084	0.0093	0.0422	0.0067	0.0073	0.0422	0.0069	0.0075	0.0422	0.0422	0.0422	0.0422	0.0422	0.0000
3-way + ox																					
CARB	0.0287	0.0104	0.0114	0.0186	0.0000	0.0000	0.0257	0.0084	0.0093	0.0204	0.0067	0.0073	0.0204	0.0069	0.0075	0.0204	0.0204	0.0204	0.0204	0.0204	0.0000
TBI	0.0287	0.0104	0.0114	0.0186	0.0000	0.0000	0.0257	0.0084	0.0093	0.0204	0.0067	0.0073	0.0204	0.0069	0.0075	0.0204	0.0204	0.0204	0.0204	0.0204	0.0000
PFI	0.0287	0.0104	0.0114	0.0186	0.0000	0.0000	0.0257	0.0084	0.0093	0.0204	0.0067	0.0073	0.0204	0.0069	0.0075	0.0204	0.0204	0.0204	0.0204	0.0204	0.0000
ox cat																					
CARB	0.0405	0.0104	0.0114	0.0242	0.0000	0.0000	0.0355	0.0084	0.0093	0.0274	0.0067	0.0073	0.0274	0.0069	0.0075	0.0274	0.0274	0.0274	0.0274	0.0274	0.0000
TBI	0.0405	0.0104	0.0114	0.0242	0.0000	0.0000	0.0355	0.0084	0.0093	0.0274	0.0067	0.0073	0.0274	0.0069	0.0075	0.0274	0.0274	0.0274	0.0274	0.0274	0.0000
PFI	0.0405	0.0104	0.0114	0.0242	0.0000	0.0000	0.0355	0.0084	0.0093	0.0274	0.0067	0.0073	0.0274	0.0069	0.0075	0.0274	0.0274	0.0274	0.0274	0.0274	0.0000
non-cat																					
CARB	0.0405	0.0104	0.0114	0.0242	0.0000	0.0000	0.0355	0.0084	0.0093	0.0274	0.0067	0.0073	0.0274	0.0069	0.0075	0.0274	0.0274	0.0274	0.0274	0.0274	0.0000
LDGT																					
3-way																					
CARB	0.0527	0.0104	0.0114	0.0396	0.0000	0.0000	0.0486	0.0084	0.0093	0.0422	0.0067	0.0073	0.0422	0.0069	0.0075	0.0422	0.0422	0.0422	0.0422	0.0422	0.0000
TBI	0.0527	0.0104	0.0114	0.0396	0.0000	0.0000	0.0486	0.0084	0.0093	0.0422	0.0067	0.0073	0.0422	0.0069	0.0075	0.0422	0.0422	0.0422	0.0422	0.0422	0.0000
PFI	0.0527	0.0104	0.0114	0.0396	0.0000	0.0000	0.0486	0.0084	0.0093	0.0422	0.0067	0.0073	0.0422	0.0069	0.0075	0.0422	0.0422	0.0422	0.0422	0.0422	0.0000
3-way + ox																					
CARB	0.0287	0.0104	0.0114	0.0186	0.0000	0.0000	0.0257	0.0084	0.0093	0.0204	0.0067	0.0073	0.0204	0.0069	0.0075	0.0204	0.0204	0.0204	0.0204	0.0204	0.0000
TBI	0.0287	0.0104	0.0114	0.0186	0.0000	0.0000	0.0257	0.0084	0.0093	0.0204	0.0067	0.0073	0.0204	0.0069	0.0075	0.0204	0.0204	0.0204	0.0204	0.0204	0.0000
PFI	0.0287	0.0104	0.0114	0.0186	0.0000	0.0000	0.0257	0.0084	0.0093	0.0204	0.0067	0.0073	0.0204	0.0069	0.0075	0.0204	0.0204	0.0204	0.0204	0.0204	0.0000
ox cat																					
CARB	0.0405	0.0104	0.0114	0.0242	0.0000	0.0000	0.0355	0.0084	0.0093	0.0274	0.0067	0.0073	0.0274	0.0069	0.0075	0.0274	0.0274	0.0274	0.0274	0.0274	0.0000
TBI	0.0405	0.0104	0.0114	0.0242	0.0000	0.0000	0.0355	0.0084	0.0093	0.0274	0.0067	0.0073	0.0274	0.0069	0.0075	0.0274	0.0274	0.0274	0.0274	0.0274	0.0000
PFI	0.0405	0.0104	0.0114	0.0242	0.0000	0.0000	0.0355	0.0084	0.0093	0.0274	0.0067	0.0073	0.0274	0.0069	0.0075	0.0274	0.0274	0.0274	0.0274	0.0274	0.0000
non-cat																					
CARB	0.0405	0.0104	0.0114	0.0242	0.0000	0.0000	0.0355	0.0084	0.0093	0.0274	0.0067	0.0073	0.0274	0.0069	0.0075	0.0274	0.0274	0.0274	0.0274	0.0274	0.0000
HDGV																					
TWC	0.0527	0.0104	0.0114	0.0396	0.0000	0.0000	0.0486	0.0084	0.0093	0.0422	0.0067	0.0073	0.0422	0.0069	0.0075	0.0422	0.0422	0.0422	0.0422	0.0422	0.0000
Non-Cat	0.0405	0.0104	0.0114	0.0242	0.0000	0.0000	0.0355	0.0084	0.0093	0.0274	0.0067	0.0073	0.0274	0.0069	0.0075	0.0274	0.0274	0.0274	0.0274	0.0274	0.0000
LDDV	0.0229	0.0000	0.0000																		
LDDT	0.0229	0.0000	0.0000																		
HDDV	0.0106	0.0000	0.0000																		

Footnotes given on the following page.

Footnotes:

(1) The composition of baseline gasoline in 1990+ is assumed to be 1.53% benzene, 32.0% aromatics, 0% oxygen, and 8.7 psi RVP, as given in Section 219 of the Clean Air Act
    and used as the baseline fuel (Fuel A) in the Auto/Oil program.

(2) The baseline gasoline exhaust benzene fractions for LDGV and LDGT were calculated using the following equations:
    3-way: Bz%HC = 1.077 + 0.7732*(volume % benzene) + 0.0987*(volume % aromatics - volume % benzene)
    3-way + ox: Bz%HC = 0.6796*(volume % benzene) + 0.0681*(volume % aromatics) - 0.3468
    other: Bz%HC = 0.8551*(volume % benzene) + 0.12198*(volume % aromatics) - 1.1626
The equation for 3-way catalysts was taken from the reformulated gasoline NPRM (July 9, 1991). The equations for the other catalyst types were taken from the RIA for the RVP regulations.
It was assumed that the benzene exhaust fractions for the HDGV categories were equal to those corresponding LDGV categories.
The benzene fractions for LDDV, LDDT, and HDDV are based on the benzene fractions of THC used in the 1987 EPA motor vehicle air toxics report (0.0240 for LDDV and LDDT; 0.0110 for HDDV).
These were then adjusted to give benzene fractions of TOG using TOG/THC ratios (1.0490 for LDD; 1.0342 for HDD).

(3) The baseline gasoline diurnal and hot soak benzene fractions are based on the following equations taken from the reformulated gasoline NPRM.
    Diurnal benzene = [(1.3758 - (0.0579*(weight %oxygen/2.0) - (0.080274*RVP)]*(volume % benzene)
    Hot soak benzene = [(1.4448 - (0.0684*(weight % oxygen/2.0) - (0.080274*RVP)]*(volume % benzene)

(4) For the winter oxygenate program, it is assumed that 15% MTBE will be the major oxygenate used in the CO non-attainment areas. The composition of the fuel is assumed to be 2.7% oxygen, 22% aromatics,
    1.05% benzene, and an arbitrary 8.7 psi RVP (although a winter fuel would have higher RVP, RVP has a minor effect on this fraction). The estimate of 22% aromatics resulted from an examination
    of the composition of 15% MTBE fuels from existing test programs. The %aromatic level was fairly consistent.
    The % reduction in aromatics from the baseline of 32% to 22% was then applied to the baseline benzene level of 1.53% to obtain the estimate of 1.05% benzene.
    The equations for calculating benzene fractions for exhaust are the same as those used for baseline gasoline. It is assumed that evaporative emissions are zero, since it is a winter program.

(5) The composition of 10% ethanol 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, applied to the baseline gasoline specifications.
    The equations for calculating benzene fractions for exhaust, diurnal, and hot soak are the same as those used for baseline gasoline,
    except the weight % oxygen term is eliminated in the hot soak and diurnal equations.

(6) The composition of reformulated gasoline in CY1995-1999 is assumed to be 2% oxygen, 1.0% benzene, 25% aromatics, and 8.1 psi RVP. The oxygen, benzene, and aromatic contents are minimum requirements
    specified in the Clean Air Act. The RVP level is an estimate for Class C areas.

(7) For reformulated gasoline in CY 2000+, the fraction of exhaust benzene (and the other toxics mentioned in CAAA Section 219) is assumed to remain the same relative to CY1995-1999. However, the
    mass of TOG will be reduced.   As a result, the mass of benzene is assumed to be reduced proportionately to TOG for exhaust. An RVP of 7.8 psi is used, which is an estimate for Class C areas.
    This different RVP assumption results in slightly higher diurnal and hot soak benzene fractions for CY 2000+ compared to 1995-1999. However, evaporative TOG will be reduced by a greater amount,
    resulting in an overall decrease in the mass of evaporative benzene.

(8) The benzene exhaust fractions for the California vehicles are assumed to be those for reformulated gasoline, since the fuel characteristics are similar.
    The benzene diurnal and hot soak fractions for the vehicles meeting the 0.39, 0.25, and 0.125 standards are assumed to equal those for 1995-1999 reformulated gasoline.
    The benzene diurnal and hot soak fractions for LEVs and ULEVs are handled differently, since these vehicles will be phased in after CARB's proposed Phase 2 gasoline specifications take effect.
    Phase 2 gasoline includes limits of 7.0 psi RVP, 1.8-2.2 wt% O2, 25% aromatics, and 1% benzene. Therefore, the diurnal and hot soak fractions for LEVs and ULEVs are 0.0076 and 0.0081, respectively,
    based on the Phase 2 gasoline specifications, assuming 2% O2.

Remaining CAAA provisions not included in this table are the clean fuel fleet program and the California pilot program. These will not be considered.

03/02/1988

HCHOTOGF Vehicle class/technology	Baseline (gasoline)(1) Exhaust	Evap	Winter oxygenates(2) Exhaust	Evap	10% Ethanol(3) Exhaust	Evap	Reformulated gasoline CY 1995-1999(4) Exhaust	Evap	CY 2000+(5) Exhaust	Evap	California NMOG standards (exhaust only)(6) 0.3900	0.2500	TLEV 0.1250	LEV 0.0750	ULEV 0.0400	ZEV 0.0000
LDGV																
3-way																
CARB	0.0087	0.0000	0.0146	0.0000	0.0128	0.0000	0.0130	0.0000	0.0130	0.0000	0.0130	0.0130	0.0130	0.0130	0.0130	0.0000
TBI	0.0087	0.0000	0.0146	0.0000	0.0128	0.0000	0.0130	0.0000	0.0130	0.0000	0.0130	0.0130	0.0130	0.0130	0.0130	0.0000
PFI	0.0087	0.0000	0.0146	0.0000	0.0128	0.0000	0.0130	0.0000	0.0130	0.0000	0.0130	0.0130	0.0130	0.0130	0.0130	0.0000
3-way + ox																
CARB	0.0137	0.0000	0.0174	0.0000	0.0168	0.0000	0.0164	0.0000	0.0164	0.0000	0.0164	0.0164	0.0164	0.0164	0.0164	0.0000
TBI	0.0137	0.0000	0.0174	0.0000	0.0168	0.0000	0.0164	0.0000	0.0164	0.0000	0.0164	0.0164	0.0164	0.0164	0.0164	0.0000
PFI	0.0137	0.0000	0.0174	0.0000	0.0168	0.0000	0.0164	0.0000	0.0164	0.0000	0.0164	0.0164	0.0164	0.0164	0.0164	0.0000
ox cat																
CARB	0.0139	0.0000	0.0281	0.0000	0.0172	0.0000	0.0244	0.0000	0.0244	0.0000	0.0244	0.0244	0.0244	0.0244	0.0244	0.0000
TBI	0.0139	0.0000	0.0281	0.0000	0.0172	0.0000	0.0244	0.0000	0.0244	0.0000	0.0244	0.0244	0.0244	0.0244	0.0244	0.0000
PFI	0.0139	0.0000	0.0281	0.0000	0.0172	0.0000	0.0244	0.0000	0.0244	0.0000	0.0244	0.0244	0.0244	0.0244	0.0244	0.0000
non-cat																
CARB	0.0269	0.0000	0.0410	0.0000	0.0297	0.0000	0.0374	0.0000	0.0374	0.0000	0.0374	0.0374	0.0374	0.0374	0.0374	0.0000
LDGT																
3-way																
CARB	0.0087	0.0000	0.0146	0.0000	0.0128	0.0000	0.0130	0.0000	0.0130	0.0000	0.0130	0.0130	0.0130	0.0130	0.0130	0.0000
TBI	0.0087	0.0000	0.0146	0.0000	0.0128	0.0000	0.0130	0.0000	0.0130	0.0000	0.0130	0.0130	0.0130	0.0130	0.0130	0.0000
PFI	0.0087	0.0000	0.0146	0.0000	0.0128	0.0000	0.0130	0.0000	0.0130	0.0000	0.0130	0.0130	0.0130	0.0130	0.0130	0.0000
3-way + ox																
CARB	0.0137	0.0000	0.0174	0.0000	0.0168	0.0000	0.0164	0.0000	0.0164	0.0000	0.0164	0.0164	0.0164	0.0164	0.0164	0.0000
TBI	0.0137	0.0000	0.0174	0.0000	0.0168	0.0000	0.0164	0.0000	0.0164	0.0000	0.0164	0.0164	0.0164	0.0164	0.0164	0.0000
PFI	0.0137	0.0000	0.0174	0.0000	0.0168	0.0000	0.0164	0.0000	0.0164	0.0000	0.0164	0.0164	0.0164	0.0164	0.0164	0.0000
ox cat																
CARB	0.0139	0.0000	0.0281	0.0000	0.0172	0.0000	0.0244	0.0000	0.0244	0.0000	0.0244	0.0244	0.0244	0.0244	0.0244	0.0000
TBI	0.0139	0.0000	0.0281	0.0000	0.0172	0.0000	0.0244	0.0000	0.0244	0.0000	0.0244	0.0244	0.0244	0.0244	0.0244	0.0000
PFI	0.0139	0.0000	0.0281	0.0000	0.0172	0.0000	0.0244	0.0000	0.0244	0.0000	0.0244	0.0244	0.0244	0.0244	0.0244	0.0000
non-cat																
CARB	0.0269	0.0000	0.0410	0.0000	0.0297	0.0000	0.0374	0.0000	0.0374	0.0000	0.0374	0.0374	0.0374	0.0374	0.0374	0.0000
HDGV																
TWC	0.0100	0.0000	0.0167	0.0000	0.0148	0.0000	0.0150	0.0000	0.0150	0.0000	0.0150	0.0150	0.0150	0.0150	0.0150	0.0000
non-cat	0.0310	0.0000	0.0473	0.0000	0.0342	0.0000	0.0431	0.0000	0.0431	0.0000	0.0431	0.0431	0.0431	0.0431	0.0431	0.0000
LDDV	0.0391	0.0000														
LDDT	0.0391	0.0000														
HDDV	0.0280	0.0000														

Footnotes given on the following page.

Footnotes:

(1) For baseline gasoline, the LDGV 3-way fraction was based on FTP data from 38 vehicles tested in four studies (Arco 91-02, Arco 91-03, Arco 91-06, and Auto/Oil).
THC as measured by the FID in these studies was converted to TOG using a single TOG/THC conversion factor of 1.0125, which is consistent with what is used in MOBILE 4.1.
This was done because the fractions are applied to MOBILE 4.1 TOG.

The LDGV 3-way + ox fraction was based on FTP data from 25 vehicles tested in eight studies (SwRI 80-004, SwRI 80-005, MSERB 46 car study, MSERB low temp study,
MSERB high temp study, 1989 CARB toxics study, Arco 91-03, and Auto/Oil). A TOG/THC conversion factor of 1.0175 was used.

The LDGV ox cat fraction was based on FTP data from 41 vehicles tested in eight studies (SwRI 80-003, SwRI 79-007, MSERB 46 car study, SwRI 81-024,
MSERB low temp study, Auto/Oil study, Arco 91-03, and 1990 SwRI report). A TOG/THC conversion factor of 1.0170 was used.

The LDGV non cat fraction was based on FTP data from ll vehicles tested in four studies (SwRI 81-020, SwRI 80-003, MSERB 46 car study, and 1990 SwRI report).
A TOG/THC conversion factor of 1.0333 was used.

LDGTs are assumed to have the same fractions as LDGVs.

The LDDV fraction was based on FTP data from seven vehicles tested in two studies (SwRI 76-034 and SwRI 79-007). A TOG/THC conversion factor of 1.0490 was used.
LDDTs are assumed to have the same fractions as LDDVs.

The HDDV fraction was based on 13-mode data from two engines tested in one study (SwRI 79-007). A TOG/THC conversion factor of 1.0342 was used.

The HDGV non-cat fraction was based on 13-mode data from one engine tested in one study (SwRI 79-007). A TOG/THC conversion factor of 1.0358 was used.
To estimate the 3-way fraction, the non-cat to 3-way fraction reduction for LDGV was applied to the HDGV non-cat fraction.

(2) For the winter oxygenate program, it is assumed that 15% MTBE (2.7 weight % O2) will be the major oxygenate used in the CO non-attainment areas.
Changes in the formaldehyde TOG fraction from 0% to 15% MTBE were estimated based on vehicle data. (As with baseline gasoline, MOBILE4.1 TOG/THC conversion factors
were used to calculate formaldehyde TOG fractions. The conversion factors for baseline gasoline by catalyst type were each multiplied by 1.0197 to estimate
TOG/THC conversion factors by catalyst type for 15% MTBE blends.) The baseline gasoline fractions were multiplied by the following to estimate fractions
for 15% MTBE: 1.6746 for 3-way, 1.2672 for 3-way + ox, 2.0244 for ox cat, and 1.5256 for non- cat. These numbers were estimated using data from Auto/Oil and
Arco 91-06 for 3-way, Auto/Oil for 3-way+ox and ox cat, and the 1990 SwRI report for non-cat.

(3) Changes in the formaldehyde TOG fraction from 0% to 10% ethanol were estimated based on vehicle data. (As with baseline gasoline, MOBILE4.1 TOG/THC conversion factors
were used to calculate formaldehyde TOG fractions. The conversion factors for baseline gasoline by catalyst type were each multiplied by 1.0232 to estimate
TOG/THC conversion factors by catalyst type for 10% ethanol.) The baseline gasoline fractions were multiplied by the following to estimate fractions
for 10% ethanol: 1.4758 for 3-way, 1.2288 for 3-way + ox, 1.2400 for ox cat, and 1.1034 for non- cat. These numbers were estimated using data from Auto/Oil,
1990 SwRI report, and CDH data for 3-way, CDH data for 3-way+ox, 1990 SwRI report and CDH data for ox cat, and the 1990 SwRI report and CDH data for non-cat.

(4) The composition of reformulated gasoline in CY1995-1999 is assumed to be 2% oxygen, 1.0% benzene and 25% aromatics.
The percent changes in the formaldehyde TOG fractions from 0% to 15% MTBE (2.7 wt% O2) were multiplied by 2/2.7, the ratio of oxygen contents by weight for
reformulated gasoline and 15% MTBE, to obtain changes in the formaldehyde TOG fractions with reformulated gasoline.

(5) For reformulated gasoline in CY 2000+, the fraction of formaldehyde is assumed to remain the same relative to CY1995-1999.
However, the mass of TOG will be reduced. As a result, the mass of formaldehyde is assumed to be reduced proportionately to TOG.

(6) The formaldehyde fractions for the California vehicles are assumed to be those for reformulated gasoline. As a result, the mass of formaldehyde is assumed
to be reduced proportionately to TOG.

Remaining CAAA provisions not included in this table are the clean fuel fleet program and the California pilot program. These will not be considered.

03/02/1988

APPENDIX C

Ambient Monitoring Data for Benzene, Formaldehyde,
1,3-Butadiene, and Acetaldehyde

Table C-1.
EPA Aerometric Information Retrieval System (AIRS)   11/6/91

Pollutant Name	Average Concentration			Sampling Sites	Land Use of Monitor Location	Sampling Time/Frequency	Num. of Samples		
	1987	1988	1989				87	88	89
		(ppb)							
Benzene	3.17	1.68		Birmingham, AL	Residential/Suburban	24 hour/Every 12 Days	7	18	
Benzene		1.40		Birmingham, AL	Residential/Suburban	24 hour/Every 12 Days		6	
Benzene	1.66			Oakland, CA	Residential/Suburban	24 hour/Every 12 Days	15		
Benzene	2.09			Oakland, CA	Residential/Suburban	24 hour/Every 12 Days	14		
Benzene	1.59			Oakland, CA	Commercial/Suburban	24 hour/Every 12 Days	13		
Benzene	3.53			Fresno, CA	Residential/Suburban	24 hour/Every 12 Days	11		
Benzene	2.04			Bakersfield, CA	Residential/Urban & Center City	24 hour/Every 12 Days	13		
Benzene	3.26			Los Angeles, CA	Commercial/Suburban	24 hour/Every 12 Days	23		
Benzene	3.49			Los Angeles, CA	Residential/Urban & Center City	24 hour/Every 12 Days	20		
Benzene	3.19			Los Angeles, CA	Residential/Suburban	24 hour/Every 12 Days	25		
Benzene	1.29			Merced, CA	Commercial/Suburban	24 hour/Every 12 Days	9		
Benzene	2.67			Riverside, CA	Residential/Suburban	24 hour/Every 12 Days	22		
Benzene	1.92			Sacramento, CA	Residential/Suburban	24 hour/Every 12 Days	10		
Benzene	2.65			San Bernardino, CA	Residential/Urban & Center City	24 hour/Every 12 Days	25		
Benzene	1.66			San Diego, CA	Residential/Suburban	24 hour/Every 12 Days	26		
Benzene	1.84			San Diego, CA	Commercial/Suburban	24 hour/Every 12 Days	21		
Benzene	1.08			San Francisco, CA	Commercial/Urban & Center City	24 hour/Every 12 Days	10		
Benzene	1.89			Stockton, CA	Residential/Urban & Center City	24 hour/Every 12 Days	15		
Benzene	2.13			Santa Barbara, CA	Mobile/Urban & Center City	24 hour/Every 12 Days	12		
Benzene	3.70			Santa Barbara, CA	Mobile/Urban & Center City	24 hour/Every 12 Days	11		
Benzene	2.04			San Jose, CA	Residential/Urban & Center City	24 hour/Every 12 Days	14		
Benzene	1.35			Modesto, CA	Commerical/Urban & Center City	24 hour/Every 12 Days	11		
Benzene	1.97			Oxnard, CA	Residential/Suburban	24 hour/Every 12 Days	23		
Benzene	1.41	0.73		Miami, FL	Commercial/Urban & Center City	24 hour/Every 12 Days	6	20	
Benzene		0.85		Miami, FL	Commercial/Urban & Center City	24 hour/Every 12 Days		5	
Benzene	1.25	0.56		Jacksonville, FL	Commercial/Suburban	24 hour/Every 12 Days	5	17	
Benzene		0.56		Jacksonville, FL	Commercial/Suburban	24 hour/Every 12 Days		5	
Benzene	1.02	0.91		Atlanta, GA	Commercial/Urban & Center City	24 hour/Every 12 Days	5	17	
Benzene		0.98		Atlanta, GA	Commercial/Urban & Center City	24 hour/Every 12 Days		4	
Benzene	1.44	1.04	1.37	Chicago, IL	Residential/Urban & Center City	24 hour/Every 12 Days	4	28	18
Benzene		0.95	0.88	Chicago, IL	Residential/Suburban	24 hour/Every 12 Days		19	20
Benzene	0.93	2.19	1.92	Chicago, IL	Industrial/Suburban	24 hour/Every 12 Days	4	28	16
Benzene	9.54	2.78		St. Louis, MO	Industrial/Suburban	24 hour/Every 12 Days	5	14	
Benzene	1.72	1.55		Louisville, KY	Commercial/Urban & Center City	24 hour/Every 12 Days	4	16	

Table C-1 Con't.

Compound				City	Type	Frequency			
Benzene	1.66	0.80		Baton Rouge, LA	Commercial/Urban & Center City	24 hour/Every 12 Days	8	19	
Benzene		0.92		Baton Rouge, LA	Commercial/Urban & Center City	24 hour/Every 12 Days		5	
Benzene		1.10	0.70	Lowell, MA	Residential/Suburban	24 hour/Every 12 Days		28	17
Benzene	1.03	1.19	1.13	Boston, MA	Industrial/Urban & Center City	24 hour/Every 12 Days	4	24	20
Benzene	0.77	1.10	0.90	Boston, MA	Industrial/Urban & Center City	24 hour/Every 12 Days	4	24	18
Benzene		1.79		Detroit, MI	Industrial/Urban & Center City	24 hour/Every 12 Days		11	
Benzene		0.78		Dearborn, MI	Industrial/Suburban	24 hour/Every 12 Days		16	
Benzene		0.50		Dearborn, MI	Industrial/Suburban	24 hour/Every 12 Days		6	
Benzene		0.85		Port Huron, MI	Residential/Suburban	24 hour/Every 12 Days		14	
Benzene		1.37		Port Huron, MI	Residential/Suburban	24 hour/Every 12 Days		4	
Benzene		0.60		Lansing/E. Lansing, MI	Commercial/Urban & Center City	24 hour/Every 12 Days		16	
Benzene			1.18	New York, NY	Residential/Suburban	24 hour/Every 12 Days			62
Benzene			1.17	New York, NY	Residential/Suburban	24 hour/Every 12 Days			62
Benzene			1.05	New York, NY	Residential/Urban & Center City	24 hour/Every 12 Days			62
Benzene	1.50	2.39		Cleveland, OH	Commercial/Urban & Center City	24 hour/Every 12 Days	5	18	
Benzene		3.46		Cleveland, OH	Commercial/Urban & Center City	24 hour/Every 12 Days		4	
Benzene		0.67		Dallas, TX	Commercial/Urban & Center City	24 hour/Every 12 Days		21	
Benzene		0.63		Dallas, TX	Commercial/Urban & Center City	24 hour/Every 12 Days		4	
Benzene		1.67	1.61	Houston, TX	Residential/Suburban	24 hour/Every 12 Days		21	19
Benzene		1.43		Houston, TX	Residential/Suburban	24 hour/Every 12 Days		23	
Benzene		0.79		Houston, TX	Residential/Suburban	24 hour/Every 12 Days		5	
Benzene		2.19	1.95	Houston, TX	Residential/Suburban	24 hour/Every 12 Days		14	20
Benzene	2.11			Houston, TX	Residential/Suburban	24 hour/Every 12 Days	7		
Benzene		1.73	1.46	Deer Park, TX	No info. listed	24 hour/Every 12 Days		28	20
Benzene	1.05	1.17		Burlington, VT	Commercial/Urban & Center City	24 hour/Every 12 Days	7	28	
Benzene	1.01	0.87		Burlington, VT	Commercial/Urban & Center City	24 hour/Every 12 Days	4	10	
Benzene		1.52	1.32	Tacoma, WA	Industrial/Suburban	24 hour/Every 12 Days		20	19

Benzene Average 2.13ppb 1.27ppb 1.28ppb
Annual Concentrations

Table C-1. Con't.

Formaldehyde		3.90	Birmingham, AL	Residential/Suburban	24 hour/Every 12 Days		19
Formaldehyde	1.43	2.02	Miami, FL	Commercial/Urban & Center City	24 hour/Every 12 Days	6	19
Formaldehyde		2.43	Jacksonville, FL	Commercial/Suburban	24 hour/Every 12 Days		16
Formaldehyde	3.28	3.11	Atlanta, GA	Commercial/Urban & Center City	24 hour/Every 12 Days	5	^20
Formaldehyde	2.21	2.09	Chicago, IL	Residential/Suburban	24 hour/Every 12 Days	7	7
Formaldehyde	1.51	1.93	St. Louis, MO	Industrial/Suburban	24 hour/Every 12 Days	9	19
Formaldehyde	4.68	5.03	Louisville, KY	Commerical/Urban & Center City	24 hour/Every 12 Days	4	20
Formaldehyde	2.27	2.13	Baton Rouge, LA	Commercial/Urban & Center City	24 hour/Every 12 Days	8	20
Formaldehyde	3.70	3.56	Dearborn, MI	Industrial/Suburban	24 hour/Every 12 Days	8	18
Formaldehyde	3.11	2.56	Detroit, MI	Industrial/Urban & Center City	24 hour/Every 12 Days	8	18
Formaldehyde	2.46	1.20	Port Huron, MI	Residential/Suburban	24 hour/Every 12 Days	7	19
Formaldehyde	1.90	1.51	Lansing/E. Lansing, MI	Commercial/Suburban	24 hour/Every 12 Days	8	19
Formaldehyde	4.72	3.94	Cleveland, OH	Commercial/Urban & Center City	24 hour/Every 12 Days	6	18
Formaldehyde	2.26	1.80	Dallas, TX	Commercial/Urban & Center City	24 hour/Every 12 Days	9	19
Formaldehyde	3.11	2.94	Houston, TX	Residential/Suburban	24 hour/Every 12 Days	7	22
Formaldehyde	2.49	2.19	Burlington, VT	Commercial/Urban & Center City	24 hour/Every 12 Days	8	20

Formaldehyde   2.79ppb   2.65ppb
Average
Annual Concentrations:

Table C-1. Con't.

Acetaldehyde		2.49	Birmingham, AL	Residential/Suburban	24 hour/Every 12 Days		17
Acetaldehyde	1.06	1.59	Miami, FL	Commercial/Urban & Center City	24 hour/Every 12 Days	5	17
Acetaldehyde		1.50	Jacksonville, Fl	Commercial/Suburban	24 hour/Every 12 Days		12
Acetaldehyde	1.63	1.95	Atlanta, GA	Commercial/Urban & Center City	24 hour/Every 12 Days	4	18
Acetaldehyde	1.17	0.98	Chicago, IL	Residential/Suburban	24 hour/Every 12 Days	6	5
Acetaldehyde	0.83	1.36	St. Louis, MO	Industrial/Suburban	24 hour/Every 12 Days	7	17
Acetaldehyde		1.88	Louisville, KY	Commercial/Urban & Center City	24 hour/Every 12 Days		16
Acetaldehyde	1.54	1.42	Baton Rouge, LA	Commercial/Urban & Center City		8	17
Acetaldehyde	0.78	1.06	Port Huron, MI	Residential/Suburban	24 hour/Every 12 Days	5	17
Acetaldehyde	1.22	1.74	Dearborn, MI	Industrial/Suburban	24 hour/Every 12 Days	5	16
Acetaldehyde	1.35	1.84	Detroit, MI	Industrial/Urban & Center City	24 hour/Every 12 Days	8	16
Acetaldehyde	1.12	1.05	Lansing/E.Lansing, MI	Commercial/Urban & Center City	24 hour/Every 12 Days	6	16
Acetaldehyde	2.08	1.91	Cleveland, OH	Commercial/Urban & Center City	24 hour/Every 12 Days	6	15
Acetaldehyde	1.29	1.35	Dallas, TX	Commercial/Urban & Center City	24 hour/Every 12 Days	9	18
Acetaldehyde	2.15	2.90	Houston, TX	Residential/Suburban	24 hour/Every 12 Days	6	19
Acetaldehyde	1.23	1.06	Burlington, VT	Commercial/Urban & Center City	24 hour/Every 12 Days	7	17
Acetaldehyde Average Annual Concentrations:	1.34ppb	1.63ppb					

C-4

Table C-1. Con't.

Pollutant Name	Average Concentration (ppb)			Sampling Sites	Land Use of Monitor Location	Sampling Time/Frequency	Num. of Samples		
	1988	1990	1991				88	90	91
1,3-Butadiene	0.44			Louisville, KY	Commercial/Urban & Center City	24 hour/Every 12 Days	6		
1,3-Butadiene	1.11	0.715		Houston, TX	Residential/Suburban	24 hour/Every 12 Days	6	4	
1,3-Butadiene	0.47			Burlington, VT	Commercial/Urban & Center City	24 hour/Every 12 Days	6		
1,3-Butadiene		0.33	0.07	Detroit, MI	Commercial/Urban & Center City	24 hour/Every 12 Days		19	28
1,3-Butadiene		0.20	0.11	Arlington County, VA	Commercial/Urban & Center City	24 hour/Every 12 Days		13	18
1,3-Butadiene		0.16	0.12	Henrico County, VA	Residential/Suburban	24 hour/Every 12 Days		21	12
1,3-Butadiene		0.22	0.11	Hampton, VA	Residential/Suburban	24 hour/Every 12 Days		14	22
1,3-Butadiene		0.13	0.06	Hopewell, VA	Residential/Suburban	24 hour/Every 12 Days		16	15
1,3-Butadiene		0.24	0.12	Roanoke, VA	Residential/Suburban	24 hour/Every 12 Days		14	22

1,3-Butadiene
Average Annual
Concentration: 0.67 ppb 0.29 ppb 0.10 ppb

Table C-2.
TAMS--Toxic Air Monitoring System   9/5/91

Pollutant Name	Sample Year(s)	Average Concentration (ppb)	Sampling Sites	Monitor Location/Land Use	Sampling Time/Frequency
Benzene	November 1987 to September 1989	0.99	Boston-1	Urban Area-Industrial	24 hour/Every 12 Days
	November 1987 to September 1989	1.15	Boston-2	Urban Area-Industrial	24 hour/Every 12 Days
	January 1987 to September 1989	0.95	Boston-3	Suburban Area-Residential	24 hour/Every 12 Days
		1.03			
Benzene	November 1987 to September 1989	1.19	Chicago-1	Urban Area-Industrial	24 hour/Every 12 Days
	November 1987 to September 1989	1.99	Chicago-2	Urban Area-Industrial	24 hour/Every 12 Days
	May 1988 to September 1989	0.91	Chicago-3	O'Hare Airport-Industrial	24 hour/Every 12 Days
		1.36			
Benzene	February 1988 to September 1989	2.05	Houston-1	Urban Area-Industrial	24 hour/Every 12 Days
	December 1987 to September 1989	1.59	Houston-2	Urban Area-Industrial	24 hour/Every 12 Days
	February 1988 to September 1989	1.64	Houston-3	Suburban Area-Residential	24 hour/Every 12 Days
		1.76			
Benzene	May 1988 to September 1989	0.62	Seattle/Tacoma	Port Of Tacoma-Industrial	24 hour/Every 12 Days
Formaldehyde	November 1987 to September 1989	1.41	Boston-1	Urban Area-Industrial	24 hour/Every 12 Days
	November 1987 to September 1989	1.83	Boston-2	Urban Area-Industrial	24 hour/Every 12 Days
	January 1987 to September 1989	1.44	Boston-3	Suburban Area-Residential	24 hour/Every 12 Days
		1.56			
Formaldehyde	November 1987 to September 1989	1.93	Chicago-1	Urban Area-Industrial	24 hour/Every 12 Days
	November 1987 to September 1989	2.27	Chicago-2	Urban Area-Industrial	24 hour/Every 12 Days
	May 1988 to September 1989	2.18	Chicago-3	O'Hare Airport-Industrial	24 hour/Every 12 Days
		2.13			

Table C-2 Con't.

Formaldehyde	February 1988 to September 1989	1.78	Houston-1	Urban Area-Industrial	24 hour/Every 12 Days
	December 1987 to September 1989	1.85	Houston-2	Urban Area-Industrial	24 hour/Every 12 Days
	February 1988 to September 1989	1.23	Houston-3	Suburban Area-Residential	24 hour/Every 12 Days
		1.62			
Formaldehyde	May 1988 to September 1989	1.54	Seattle/Tacoma	Port of Tacoma-Industrial	24 hour/Every 12 Days

Table C-3.
Urban Air Toxics Monitoring Program (UATMP)   9/5/91

Pollutant Name	Average Concentration (ppb)** 1989	1990	Sampling Sites	Monitor Location//Land Use	Sampling Time/Frequency	Num. Samples Compound Detected/Maximum Total 1989	1990
Benzene	1.62	1.37	Baton Rouge, LA	Urban/Center City-Commercial	24-Hour/Every 12 Days	31/31	29/29
Benzene	3.37	1.55	Chicago, IL	Suburban Area-Residential	24-Hour/Every 12 Days	27/27	29/29
Benzene	1.83	1.40	Camden, NJ	Suburban Area-Residential	24-Hour/Every 12 Days	32/32	30/30
Benzene	0.92		Dallas, TX	Urban/Center City-Commercial	24-Hour/Every 12 Days	25/25	
Benzene	1.83		Fort Lauderdale, FL	Urban/Center City-Commercial	24-Hour/Every 12 Days	31/31	
Benzene	2.35	2.69	Houston, TX	Suburban Area-Residential	24-Hour/Every 12 Days	34/34	28/28
Benzene	1.47		Miami, FL	Urban/Center City-Commercial	24-Hour/Every 12 Days	33/33	
Benzene	0.60	1.24	Pensacola, FL	Suburban Area-Industrial	24-Hour/Every 12 Days	7/7	42/42
Benzene	3.97		St. Louis, MO	Urban/Center City-Commercial	24-Hour/Every 12 Days	30/30	
Benzene	2.93	1.94	Sauget, IL	Suburban Area-Industrial	24-Hour/Every 12 Days	31/31	27/27
Benzene	1.38	1.28	Washington, DC-1	Urban/Center City-Commercial	24-Hour/Every 12 Days	27/27	30/30
Benzene	2.08	1.78	Washington, DC-2	Urban/Center City-Commercial	24-Hour/Every 12 Days	27/27	27/27
Benzene	0.91	1.18	Wichita, KS-1	Urban/Center City-Residential	24-Hour/Every 12 Days	31/31	30/30
Benzene	1.07		Wichita, KS-2	Suburban Area-Residential	24-Hour/Every 12 Days	31/31	
Benzene		1.39	Port Neches, TX	Suburban Area-Residential	24-Hour/Every 12 Days		28/28
Benzene		1.00	Orlando, FL	Urban/Center City-Commercial	24-Hour/Every 12 Days		28/28
Benzene		0.84	Toledo, OH	Suburban Area-Residential	24-Hour/Every 12 Days		21/21

Benzene Average Concentration ppb*: 1.96   1.47

* calculated by averaging equally all 397 samples taken from 14 sites
  in 1989 and 349 samples from 12 sites in 1990.
** The arithmetic average concentration of all samples using half
   Model Detection Limit (MDL) value for samples in which the
   compound was not found.

Table C-3. Con't. UATMP-1,3-Butadiene Results

1,3-butadiene	0.39	0.36	Baton Rouge, LA	Urban/Center City-Commercial	24-Hour/Every 12 Days	11/31	8/29
1,3-butadiene	0.24	0.06	Chicago, IL	Suburban Area-Residential	24-Hour/Every 12 Days	10/27	4/29
1,3-butadiene	0.20	0.10	Camden, NJ	Suburban Area-Residential	24-Hour/Every 12 Days	19/32	9/30
1,3-butadiene	0.08		Dallas, TX	Urban/Center City-Commercial	24-Hour/Every 12 Days	8/25	
1,3-butadiene	0.20		Fort Lauderdale, FL	Urban/Center City-Commercial	24-Hour/Every 12 Days	18/31	
1,3-butadiene	0.60	0.47	Houston, TX	Suburban Area-Residential	24-Hour/Every 12 Days	23/34	17/28
1,3-butadiene	0.11		Miami, FL	Urban/Center City-Commercial	24-Hour/Every 12 Days	7/33	
1,3-butadiene		0.06	Pensacola, FL	Suburban Area-Industrial	24 Hour/Every 12 Days		6/42
1,3-butadiene	0.09		St. Louis, MO	Urban/Center City-Commercial	24-Hour/Every 12 Days	12/30	
1,3-butadiene	0.20	0.06	Sauget, IL	Suburban Area-Industrial	24-Hour/Every 12 Days	7/31	2/27
1,3-butadiene	0.11	0.10	Washington, DC-1	Urban/Center City-Commercial	24-Hour/Every 12 Days	9/27	11/30
1,3-butadiene	0.29	0.15	Washington, DC-2	Urban/Center City-Commercial	24-Hour/Every 12 Days	19/27	12/27
1,3-butadiene	0.16	0.06	Wichita, KS-1	Urban/Center City-Residential	24-Hour/Every 12 Days	10/31	1/30
1,3-butadiene	0.09		Wichita, KS-2	Suburban Area-Residential	24-Hour/Every 12 Days	7/31	
1,3-butadiene		11.09	Port Neches, TX	Suburban Area-Residential	24-Hour/Every 12 Days		24/28
1,3-butadiene		0.10	Orlando, FL	Urban/Center City-Commercial	24 Hours/Every 12 Days		8/28
1,3-butadiene		0.06	Toledo, OH	Suburban Area-Residential	24 Hours/Every 12 Days		4/21
1,3-butadiene	0.21	1.02					

Average
Concentration: ppb*

* Calculated by averaging all 390 samples taken from 13 sites equally
  in 1989 and 349 samples from 12 sites in 1990.
** The arithmetic average concentration of all samples using half
   Model Detection Limit (MDL) value for samples in which the
   compound was not found.

Table C-3 Con't. UATMP-Aldehyde Results

Acetaldehyde	1.25	1.88	Baton Rouge, LA	Urban/Center City-Commercial	24-Hour/Every 12 Days	30/31	22/30
Acetaldehyde	1.36	2.04	Chicago, IL	Suburban Area-Residential	24-Hour/Every 12 Days	27/30	28/28
Acetaldehyde	1.61	2.34	Camden, NJ	Suburban Area-Residential	24-Hour/Every 12 Days	32/32	30/30
Acetaldehyde	1.32		Dallas, TX	Urban/Center City-Commercial	24-Hour/Every 12 Days	29/31	
Acetaldehyde	1.28		Ft. Lauderdale, FL	Urban/Center City-Commercial	24-Hour/Every 12 Days	32/32	
Acetaldehyde	1.86	0.68	Houston, TX	Suburban Area-Residential	24-Hour/Every 12 Days	38/38	23/27
Acetaldehyde	1.16		Miami, FL	Urban/Center City-Commercial	24-Hour/Every 12 Days	31/32	
Acetaldehyde	1.17	0.81	Pensacola, FL	Suburban Area-Industrial	24-Hour/Every 12 Days	7/7	40/42
Acetaldehyde	1.45		St. Louis, MO	Urban/Center City-Commercial	24-Hour/Every 12 Days	30/31	
Acetaldehyde	0.92	1.03	Sauget, IL	Suburban Area-Industrial	24-Hour/Every 12 Days	29/30	28/29
Acetaldehyde	1.82	1.70	Washington, DC-1	Urban/Center City-Commercial	24-Hour/Every 12 Days	28/28	30/30
Acetaldehyde	1.61	2.47	Washington, DC-2	Urban/Center City-Commercial	24-Hour/Every 12 Days	30/30	30/30
Acetaldehyde	0.98	1.86	Wichita, KS-1	Urban/Center City-Residential	24-Hour/Every 12 Days	28/30	30/30
Acetaldehyde	1.02		Wichita, KS-2	Suburban Area-Residential	24-Hour/Every 12 Days	35/36	
Acetaldehyde		2.36	Port Neches, TX	Suburban Area-Residential	24-Hour/Every 12 Days		29/30
Acetaldehyde		1.43	Orlando, FL	Urban/Center City-Commercial	24 Hour/Every 12 Days		25/28
Acetaldehyde		0.91	Toledo, OH	Suburban Area-Residential	24 Hour/Every 12 Days		17/22

Acetaldehyde   1.36   1.72***
Average
Concentration: ppb*

   * Calculated by averaging equally all 418 samples taken from 14 sites
     for 1989 and all 356 samples taken from 12 sites in 1990.
  ** The arithmetic average concentration of all samples using half
     Model Detection Limit (MDL) value for samples in which the
     compound was not found.
 *** Sampling technique in 1990 accounted for the interference of ozone
     resulting in higher exposure numbers.

Table C-3 Con't. UATMP-Formaldehyde Results

Formaldehyde	1.52	4.99	Baton Rouge, LA	Urban/Center City-Commercial	24-Hour/Every 12 Days	31/31	30/30
Formaldehyde	2.07	4.75	Chicago, IL	Suburban Area-Residential	24-Hour/Every 12 Days	30/30	28/28
Formaldehyde	2.41	4.90	Camden, NJ	Suburban Area-Residential	24-Hour/Every 12 Days	32/32	30/30
Formaldehyde	2.01		Dallas, TX	Urban/Center City-Commercial	24-Hour/Every 12 Days	29/31	
Formaldehyde	2.24		Ft. Lauderdale, FL	Urban/Center City-Commercial	24-Hour/Every 12 Days	32/32	
Formaldehyde	2.32	1.46	Houston, TX	Suburban Area-Residential	24-Hour/Every 12 Days	38/38	26/27
Formaldehyde	1.76		Miami, FL	Urban/Center City-Commercial	24-Hour/Every 12 Days	32/32	
Formaldehyde	1.59	2.47	Pensacola, FL	Suburban Area-Industrial	24-Hour/Every 12 Days	7/7	42/42
Formaldehyde	2.41		St. Louis, MO	Urban/Center City-Commercial	24-Hour/Every 12 Days	31/31	
Formaldehyde	1.45	2.49	Sauget, IL	Suburban Area-Industrial	24-Hour/Every 12 Days	30/30	29/29
Formaldehyde	3.77	4.86	Washington, DC-1	Urban/Center City-Commercial	24-Hour/Every 12 Days	28/28	30/30
Formaldehyde	3.09	7.92	Washington, DC-2	Urban/Center City-Commercial	24-Hour/Every 12 Days	30/30	30/30
Formaldehyde	1.46	4.73	Wichita, KS-1	Urban/Center City-Residential	24-Hour/Every 12 Days	30/30	30/30
Formaldehyde	1.40		Wichita, KS-2	Suburban Area-Residential	24-Hour/Every 12 Days	36/36	
Formaldehyde		4.82	Port Neches, TX	Suburban Area-Residential	24 Hour/Every 12 Days		29/30
Formaldehyde		4.39	Orlando, TX	Urban/Center City-Commercial	24 Hour/Every 12 Days		28/28
Formaldehyde		2.40	Toledo, OH	Suburban Area-Residential	24 Hour/Every 12 Days		22/22

Formaldehyde   2.12   4.21***
Average
Concentration: ppb*

    * Calculated by averaging equally all 418 samples taken from 14 sites
      in 1989 and all 356 samples taken from 12 sites in 1990.
    ** The arithmetic average concentration of all samples using half
       Model Detection Limit (MDL) value for samples in which the
       compound was not found.
    *** Sampling technique in 1990 accounted for the interference of ozone
       resulting in higher exposure numbers.

Table C-4.
National Ambient Volatile Organic (VOC's) Database Update   10/4/91

Pollutant Name	Sample Year(s)	Average Concentration (ppb)	# of Samples Used For Average	Sampling Sites	Land Use of Monitor Location	# of Samples/ Sampling Time
Benzene	1/14/87 to 9/22/87	2.04	13	Bakersfield, CA	Urban	1/24-Hours
Benzene	1/8/87 to 9/27/87	1.91	26	Chula Vista, CA	Urban	1/24-Hours
Benzene	1/25/87 to 9/13/87	1.89	10	Citrus Heights, CA	Urban	1/24-Hours
Benzene	1/5/87 to 9/28/87	2.06	14	Concord, CA	Urban	1/24-Hours
Benzene	2/2/87 to 9/22/87	1.83	21	El Cajon, CA	Urban	1/24-Hours
Benzene	1/5/87 to 9/29/87	3.26	23	El Monte, CA	Urban	1/24-Hours
Benzene	1/7/87 to 9/30/87	1.66	15	Fremont, CA	Urban	1/24-Hours
Benzene	1/6/87 to 9/14/87	3.53	11	Fresno, CA	Urban	1/24-Hours
Benzene	1/16/87 to 9/28/87	3.19	25	Long Beach, CA	Urban	1/24-Hours
Benzene	1/7/87 to 9/21/87	3.49	20	Los Angeles, CA	Urban	1/24-Hours
Benzene	1/13/87 to 6/21/87	1.29	9	Merced, CA	Urban	1/24-Hours
Benzene	1/12/87 to 8/30/87	1.36	11	Modesto, CA	Urban	1/24-Hours
Benzene	1/6/87 to 8/12/87	1.58	13	Richmond, CA	Urban	1/24-Hours
Benzene	1/7/87 to 9/13/87	2.67	22	Rubidoux, CA	Urban	1/24-Hours
Benzene	2/3/87 to 2/3/87	3.60	1	San Francisco, CA	Urban	1/24-Hours
Benzene	1/27/87 to 9/21/87	2.04	14	San Jose, CA	Urban	1/24-Hours
Benzene	1/28/87 to 9/15/87	2.88	23	Santa Barbara, CA	Urban	1/24-Hours
Benzene	1/1/87 to 9/30/87	1.97	23	Simi Valley, CA	Urban	1/24-Hours
Benzene	1/11/87 to 9/29/87	1.89	15	Stockton, CA	Urban	1/24-Hours
Benzene	1/13/87 to 9/23/87	2.65	25	Upland, CA	Urban	1/24-Hours
Benzene	1/3/87 to 2/20/87	2.61	6	Philadelphia, PA	Suburban	1/24-Hours
Benzene	1/8/87 to 4/28/87	1.19	8	San Leandro, CA	Urban	1/24-Hours
Benzene	1/8/87 to 4/28/87	1.08	8	Livermore, CA	Urban	1/24-Hours
Benzene	1/19/87 to 4/15/87	1.35	8	San Rafael, CA	Urban	1/24-Hours
Benzene	1/12/87 to 4/22/87	1.93	8	Napa, CA	Urban	1/24-Hours
Benzene	1/12/87 to 4/22/87	2.20	8	Vallejo, CA	Urban	1/24-Hours
Benzene	1/19/87 to 4/15/87	2.01	8	Santa Rosa, CA	Urban	1/24-Hours

Table C-4 Con't.

Benzene	1/6/87 to 4/26/87	2.31	8	Redwood City, CA	Urban	1/24-Hours
Benzene	1/6/87 to 4/26/87	1.51	8	Mountain View, CA	Urban	1/24-Hours
Benzene	4/26/87 to 4/26/87	0.800	1	Oakland, CA	Urban	1/24-Hours
Benzene	1/1/87 to 6/30/87	2.54	159	Baton Rouge, LA	Urban	Varies

Overall Benzene Average Concentration: 2.21 ppb
* Calculated by averaging all 564 samples taken from 31 cities equally
Zero values included in average.

Formaldehyde	1/3/87 to 6/8/87	3.25	36	Philadelphia, PA	Suburban	1/24-Hours

Overall Formaldehyde Average Concentration: 3.25 ppb
* Calculated by averaging all 36 samples taken from Philadelphia equally
Zero values included in average

1,3-Butadiene	9/22/87 to 10/4/87	0.30	2	Bakersfield, CA	Urban	1/24-Hours
1,3-Butadiene	9/16/87 to 9/28/87	0.35	2	Concord, CA	Urban	1/24-Hours
1,3-Butadiene	10/4/87 to 10/4/87	0.60	1	Fremont, CA	Urban	1/24-Hours
1,3-Butadiene	10/4/87 to 10/4/87	0.40	1	Richmond, CA	Urban	1/24-Hours
1,3-Butadiene	9/9/87 to 10/7/87	0.25	2	San Jose, CA	Urban	1/24-Hours
1,3-Butadiene	9/29/87 to 9/27/87	0.30	1	Stockton, CA	Urban	1/24-Hours

Overall 1,3-Butadiene Average Concentration: 0.344 ppb
* calculated by averaging all 9 samples taken from 6 cities equally

APPENDIX D

Time Series Plots Data for Benzene, Formaldehyde,
1,3-Butadiene, and Acetaldehyde

APPENDIX E

Benzene Unit Risk Estimates Based on 21 Models

**Table E-1. Benzene Unit Risk Estimates
Results From Relative Risk Model Using Cumulative Dose**

Data set	Unit risk estimate $(ppm)^{-1}$		
	95% lower bound	MLE	95% upper bound

I
Rinsky et al.
  (follow-up from 1950,
  Table 6)[a]
	1.65×10^{-2}	5.10×10^{-2}	1.08×10^{-1}

II
Rinsky et al.
  (follow-up from 1940,
  Table 2)
	1.53×10^{-2}	5.10×10^{-2}	1.13×10^{-1}

III
Rinsky et al.
  (revised exposures,
  Table 7)
	1.78×10^{-2}	6.43×10^{-2}	1.44×10^{-1}

IV Rinsky et al. (Table 6) and Ott et al. (Table 1)	7.78×10^{-3}	2.89×10^{-2}	6.87×10^{-2}
V Same as IV except two questionable cases added	4.45×10^{-3}	2.04×10^{-2}	5.10×10^{-2}

VI
Wong et al. (Table 3,
  SMR=50 assumed for
  unexposed
	8.76×10^{-4}	1.04×10^{-1}	2.42×10^{-1}

VII
Rinsky et al. (Table 6)
  Ott et al. (Table 1)
  and Wong et al. (Table 3)
	2.67×10^{-2}	7.32×10^{-2}	1.60×10^{-1}

VIII
Same as VII except
  SMR=100 assumed for
  unexposed
	1.56×10^{-2}	3.55×10^{-2}	6.43×10^{-2}

[a]The tables referred to are those given in EPA (1985).

**Table E-2. Benzene Unit Risk Estimates (Continued)
Results From Absolute Risk Model Using Cumulative Dose**

Data set	Unit risk estimate (ppm^{-1})		
	95% lower bound	MLE	95% upper bound

I
Rinsky et al.
  (follow-up from 1950,
  Table 6)[a]
	1.02×10^{-2}	2.45×10^{-2}	4.77×10^{-1}

II
Rinsky et al.
  (follow-up from 1940,
  Table 2)
	9.31×10^{-3}	2.20×10^{-2}	4.40×10^{-2}

III
Rinsky et al.
  (revised exposures,
  Table 7)
	1.22×10^{-2}	3.06×10^{-2}	5.99×10^{-2}

IV Rinsky et al. (Table 6) and Ott et al. (Table 1)	7.23×10^{-3}	1.96×10^{-2}	3.79×10^{-2}
V Same as IV except two questionable cases added	7.11×10^{-3}	2.08×10^{-2}	4.04×10^{-2}

[a]The tables referred to are those given in EPA (1985).

**Table E-3. Benzene Unit Risk Estimates (Continued)
Results From Relative Risk Model Using Cumulative Dose**

Data set	Unit risk estimate (ppm^{-1})		
	95% lower bound	MLE	95% upper bound
I Rinsky et al. (Table 8)[a]	1.30×10^{-2}	4.10×10^{-2}	8.79×10^{-2}
II Rinsky et al. (Table 8) and Ott et al. (Table 10)	7.29×10^{-3}	2.49×10^{-2}	5.59×10^{-2}

[a]The tables referred to are those given in EPA (1985).

**Table E-4. Benzene Unit Risk Estimates (Continued)
Results From Relative Risk Model Using Cumulative Dose**

Data set	Unit risk estimate (ppm^{-1})		
	95% lower bound	MLE	95% upper bound
I Rinsky et al. 1940–1970 follow-up (Table 8)[a]	7.47×10^{-3}	1.76×10^{-2}	3.36×10^{-2}
II Rinsky et al. (Table 8) and Ott et al. (Table 10)	5.43×10^{-3}	1.40×10^{-2}	2.71×10^{-2}

[a]The tables referred to are those given in EPA (1985).

**Table E-5. Benzene Unit Risk Estimates (Continued)
Results From Relative Risk Model Using Cumulative Dose**

Data set	Unit risk estimate (ppm^{-1})		
	95% lower bound	MLE	95% upper bound
I Rinsky et al. (Table 9)[a]	5.56×10^{-3}	1.67×10^{-2}	2.48×10^{-2}
II Rinsky et al. (Table 9) and Ott et al. (Table 11)	3.60×10^{-3}	1.15×10^{-2}	2.48×10^{-2}

[a]The tables referred to are those given in EPA (1985).

**Table E-6. Benzene Unit Risk Estimates (Continued)
Results From Relative Risk Model Using Cumulative Dose**

Data set	Unit risk estimate (ppm^{-1})		
	95% lower bound	MLE	95% upper bound
I Rinsky et al. (Table 9)[a]	4.34×10^{-3}	1.10×10^{-2}	2.17×10^{-2}
II Rinsky et al. (Table 9) and Ott et al. (Table 11)	3.48×10^{-3}	8.98×10^{-3}	2.52×10^{-2}

[a]The tables referred to are those given in EPA (1985).

**Table E-7. Effect of Modifications of EPA's Assumptions
on Unit Risk Estimate for Benzene[a]**

Modification	Excess risk per 1 ppm lifetime exposure[b]
None (Combined EPA Estimate)	2.7×10^{-2} (8.3×10^{-6})
Model restricted to absolute risk and weighted cumulative dose form; data restricted to Rinsky cohort	1.8×10^{-2} (5.5×10^{-6})
Three years of follow-up added	1.7×10^{-2} (5.2×10^{-6})
Job code errors corrected	1.8×10^{-2} (5.5×10^{-6})
New weighted cumulative dose form from epidemiological latency data; new statistical method for estimating transition rate parameter	3.2×10^{-3} (9.8×10^{-7})
New definition of diseases induced by benzene; new estimate of background rates in U.S. population	3.5×10^{-3} (1.1×10^{-6})
Quadratic model: Two molecules of benzene metabolite required to induce initial event producing malignant cell	1.4×10^{-4} (4.3×10^{-8})
Linear-quadratic model: Upper bound on quadratic model	1.0×10^{-3} (3.1×10^{-7})

[a]Source: Clement Associates, Inc. 1988
[b]Numbers in parentheses are excess risk per $\mu g/m^3$

APPENDIX F

Lay Description of the Linearized Multistage Model

Lay Description of the Linear Multistage Model

The assessment of human cancer risk associated with some specified chemical exposure is a complex process that requires careful review of all the pertinent information by appropriately trained individuals including statisticians, toxicologists, epidemiologists, and pathologists. In a small number of instances, epidemiological data are suitable for quantitative estimates of risk and permit a dose-response relationship to be developed directly from human data. Should epidemiological data be available from either occupational or case-control studies, these studies should be evaluated for their applicability in establishing causal relationships and their suitability for inclusion in quantitative risk assessment.

In the majority of cases, the available epidemiological studies are inadequate and assessment of human cancer risk is based on animal bioassays. Carcinogenicity bioassays are usually designed as screening procedures with the primary focus being hazard identification, rather than risk assessment. In such studies, a limited number of animals may be exposed to a maximum tolerated dose that is several orders of magnitude higher than that encountered by humans. That being the case, two extrapolations are necessary to convert the animal data to appropriate human risk estimates: the first extrapolation is from animals to humans and the second is from high experimental doses to the low doses encountered by humans.

In extrapolation from animal data to humans, the appropriate route, species, tumor type, and dose units (i.e., those which provide an adequate model of human carcinogenicity) are not always known with certainty. When several bioassays of a chemical exist, it is necessary to select for analysis those experiments that are most appropriate for making quantitative estimates. Toxicological and statistical considerations apply in that selection.

Ideally, the process of selecting a key study from among the various available bioassays, in which different species, strains, sexes, or routes of administration may have been tested, should maximize the biological correlations between animal species and humans. Available information on comparative metabolism, pharmacokinetics, and mechanisms of action should be considered when making a choice of data to use. Specific guidelines for evaluating studies for use in risk assessment have been proposed by the EPA (1989). Those studies with suitable dose-response data that meet statistical and toxicological criteria are then included in a quantitative risk assessment.

Once particular experiments have been selected for analysis, it is necessary to select the specific tumor responses that are used to estimate a dose-response relationship. Tumor

response that may be considered include: tumors located at sites related to the metabolism, storage, or elimination of the chemical; tumor types related to chemical exposure in epidemiological studies; or, tumors that show a statistically significant dose-related trend or significant increased incidence in treated animals when compared to control. In the absence of quantitative information describing differences in metabolism, pharmacokinetics, or pharmacodynamics between animals and humans, quantitative estimates of human cancer risk are usually based on those tumor responses that show a statistically significant increased incidence in specific organs or tissues.

There are a number of statistical issues in the analysis and interpretation of animal carcinogenicity studies that should be considered. For example, in the analysis of tumor incidence data, survival differences among groups should be taken into account. However, no rigid statistical "decision rule" should be employed in the interpretation of carcinogenicity data. Even if a study has been carefully designed and appropriate statistical methodology employed, interpretation of results is a complex process. Carcinogenic responses should be evaluated carefully as to their biological relevance with respect to human carcinogenic risks. Special consideration should be given to the evaluation of rare tumors or to tumors at sites with a high spontaneous background.

Extrapolation from high to low dose is based on a presumed dose-response relationship, with parameters estimated from the experimental data. The mathematical form of the dose-response model selected is an important consideration, as different models can provide very different estimates of risk outside of the experimental range of exposure levels. It has been argued that the dose-response function for carcinogenicity could be linear or that it is unlikely to exceed linearity in the low dose region. The dose-response model used most commonly is the multistage model for quantal data (i.e., data indicating only the number of animals with cancer) (Crump et al. 1977; Crump 1984). This model expresses upper confidence limits on cancer risk as a linear function of dose in the low dose range.

The multistage model is based on the Armitage and Doll (1961) model that assumes that a cell line goes through a number of distinct stages (k) in its progression to becoming cancerous. For a spontaneous tumor, the rate at which it progresses through a specific stage is assumed to be constant. Different cell lines are assumed to compete independently in producing tumors. The underlying basis for the multistage model is that cancer incidence will increase as a function of age ($[age]^{k-1}$), which agrees with the observation that the age-specific incidence rates of many human cancers, particularly carcinomas in organs other than sex organs, increase as $(age)^x$, where x ranges between 3 and 6 (Crump and Howe 1984). Crump and Howe (1984) have extended the

Armitage-Doll model to include the effect of exposure to a carcinogen by assuming that the transition rate at which a cell goes through each stage is linearly related to the dose rate (i.e., that

$$\gamma_i = \alpha_i + \beta_i d$$

where d is the dose rate of a continuously applied carcinogen). Here α_i is the background transition rate in the absence of an applied dose, and β_i represents the increase in the transition rate per unit dose. Through a series of complicated mathematical steps, this formula is transformed into the linearized multistage model. The mathematical form of the linearized multistage model is

$$P(d) = 1 - \exp(-q_0 - q_1 d - \ldots q_k d^k)$$

where q_1, which is called the linear term, is equal to or greater than zero, d is the average lifetime daily dose of the chemical in mg/kg/day, P(d) is the lifetime probability of cancer from the dose level d, and $q_0, \ldots, q_k$ are nonnegative parameters estimated by fitting the model to experimental animal carcinogenicity data. The input into this model is the experimental dose, the number of animals with the specific tumor, and the number of animals at risk or examined for that specific tumor. This is often referred to as quantal data.

The quantity of principal interest is not the absolute probability of a cancer P(d), but rather the extra lifetime risk of cancer resulting from exposure to dose d. This risk is defined as

$$[P(d)-P(0)]/[1-P(0)],$$

and can be interpreted as the probability of the occurrence of a tumor at a dose of d, given that no tumor would have occurred in the absence of the dose.

Parameters (q values) are estimated by fitting the model to experimental animal carcinogenicity data using the maximum likelihood method. In addition to maximum likelihood estimates of model parameters, an upper bound or upper confidence limit[1] on

---

[1] Since, there is an inherent, mathematical uncertainty in an extrapolation from high doses to low doses using a small number of data points, confidence limits are estimated. A confidence limit is a statistical term that describes the degree of confidence that the estimated risk is not likely to differ by more than a specified amount from the risk that would be predicted by the model if more data were available. The EPA generally uses the 95% upper confidence limit as an upper bound on low-dose cancer risks. By using the 95% upper confidence limit, there is only a 5% chance that the risk predicted by the model would be higher than the risk value that is used. The confidence limit gives an indication of how well the data fit the model at high-dose levels but cannot

the dose-response curve is calculated, reflecting the uncertainty
of extrapolating the curve to low doses at which human exposures
are anticipated to occur. This upper bound or confidence limit
can be considered to represent the largest reasonable linear
extrapolation to low doses consistent with the data. The method
for determining the upper confidence limits for extra risk and
the lower confidence limits for risk-related doses is based on
the largest value for the linear term q_1 that is consistent with
the data. This new term is the q_1^*, also referred to as the unit
potency estimate or unit cancer estimate. The estimated dose-
response curve will be linear at low doses whenever the estimate
of the linear coefficient, q_1, is greater than zero. The upper
bound, specifically the q_1^*, is always linear, since there is
always some model with a positive coefficient that is consistent
with the data.

   The output value from the multistage model, q_1^*, is the 95%
upper confidence limit on the linear term q_1, and represents the
unit risk expressed in units of $(mg/kg/day)^{-1}$ that is directly
applicable to humans, when appropriate "scaling up" dose
conversions are applied to the experimental data prior to
application of the dose-response models. Similarly, the output
Maximum Likelihood Estimate (MLE) and statistical lower bounds on
risk related doses are directly applicable to humans. At low
doses, estimates of the upper bound on extra cancer risk can be
obtained using the equation

   $Risk = q_1^* (mg/kg/day)^{-1} *$ exposure dose $(mg/kg/day)$.

   In addition, animal-to-human extrapolation is accomplished
by assuming that animals and humans are equally susceptible (in
terms of extra risk) when the dose is measured in the same units
for both species. EPA methodology assumes that doses measured in
units of mg/m^2 surface area/day ("surface area" equivalency) give
equal risks in animals and humans.

   Some of the difficulties in risk assessment, whether
bioassay or epidemiologically-based, arise when exposures are
intermittent. In this case, it is problematical whether or not
an average dose adequately reflects the exposure history or if
less than lifetime human exposures can be estimated from
experiments dosing animals for their entire lifespan.

---

indicate how well the model reflects the true low-dose risks.

APPENDIX G

Diesel Particulate Emission Factor Inputs

Diesel Market Shares

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	0.03	0.01	1	28	46	51	20	60	84	99.9	100	100
1989	0.04	0.01	1.5	26.1	0	50	77	56	74	99.9	100	100
1988	0	0.15	1.5	20.4	0	56	28	33	70	98	100	100
1987	0.1	0.5	2	21.7	0	55	7	22	63	96	100	100
1986	1	0.5	2	28.9	0	0	0	22	56	96	100	100
1985	1.7	0.8	2.5	31.9	30	0	0	31	58	96	100	100
1984	2.1	1	4.1	30.9	26.5	26.5	26.5	26	55	98	100	100
1983	2.2	2	8.8	16.6	23.4	23.4	23.4	23	55	92	100	100
1982	4.5	5.1	4.8	19.7	19.8	19.8	19.8	21	49	94	100	100
1981	6.1	8.2	3.6	0	0	0	0	16	60	94	100	100
1980	4.3	12.6	0.9	0	0	0	0	11	60	95	100	100
1979	2	3.3	0	0	0	0	0	8	54	90	100	100
1978	1.2	3	0	0	0	0	0	7	60	88	100	100
1977	0.4	0.2	0.1	0	0	0	0	2	57	90	100	100
1976	0.3	0	0.2	0	0	0	0	3	45	84	100	100
1975	0	0	0	0	0	0	0	1	48	70	100	100
1974	0	0	0	0	0	0	0	2	36	74	100	100
1973	0	0	0	0	0	0	0	2	40	78	100	100
1972	0	0	0	0	0	0	0	2	40	78	100	100
1971	0	0	0	0	0	0	0	2	40	78	100	100

Gasoline Annual Vehicle Miles Travelled

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	13118	15640	17608	18211	18211	18211	18211	18211	18211	18211	18211	58500
1989	12408	14590	16217	16767	16767	16767	16767	16767	16767	16767	16767	55400
1988	11737	13610	14937	15437	15437	15437	15437	15437	15437	15437	15437	54200
1987	11103	12696	13758	14213	14213	14213	14213	14213	14213	14213	14213	53400
1986	10503	11843	12671	13086	13086	13086	13086	13086	13086	13086	13086	53000
1985	9935	11048	11671	12048	12048	12048	12048	12048	12048	12048	12048	47600
1984	9389	10306	10749	11093	11093	11093	11093	11093	11093	11093	11093	37800
1983	8889	9614	9901	10213	10213	10213	10213	10213	10213	10213	10213	36400
1982	8409	8968	9119	9403	9403	9403	9403	9403	9403	9403	9403	32500
1981	7954	8366	8399	8657	8657	8657	8657	8657	8657	8657	8657	31500
1980	7524	7804	7736	7971	7971	7971	7971	7971	7971	7971	7971	26000
1979	7117	7280	7125	7339	7339	7339	7339	7339	7339	7339	7339	22750
1978	6733	6791	6562	6757	6757	6757	6757	6757	6757	6757	6757	20600
1977	6369	6335	6044	6221	6221	6221	6221	6221	6221	6221	6221	21000
1976	6024	5909	5567	5728	5728	5728	5728	5728	5728	5728	5728	20500
1975	5698	5512	5127	5273	5273	5273	5273	5273	5273	5273	5273	20600
1974	5390	5142	4723	4855	4855	4855	4855	4855	4855	4855	4855	14800
1973	5099	4797	4350	4470	4470	4470	4470	4470	4470	4470	4470	17000
1972	4823	4475	4006	4116	4116	4116	4116	4116	4116	4116	4116	15500
1971	4562	4174	3690	3789	3789	3789	3789	3789	3789	3789	3789	13000

Diesel Annual Vehicle Miles Travelled

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	17825	20140	20140	17608	23611	23611	23611	43946	43946	43946	86375	58500
1989	16478	17572	17572	16217	20947	20947	20947	40504	40504	40504	79434	55400
1988	15233	15432	15432	14937	18583	18583	18583	37332	37332	37332	73051	54200
1987	14081	13639	13639	13758	16486	16486	16486	34408	34408	34408	67181	53400
1986	13017	12133	12133	12671	14625	14625	14625	31713	31713	31713	61782	53000
1985	12033	10863	10863	11671	12975	12975	12975	29229	29229	29229	56817	47600
1984	11124	9788	9788	10749	11511	11511	11511	26939	26939	26939	52252	37800
1983	10283	8877	8877	9901	10212	10212	10212	24929	24929	24929	48053	36400
1982	9506	8103	8103	9119	9059	9059	9059	22885	22885	22885	44191	32500
1981	8788	7444	7444	8399	8037	8037	8037	21092	21092	21092	40640	31500
1980	8123	6883	6883	7736	7130	7130	7130	19440	19440	19440	37374	26000
1979	7509	6405	6405	7125	6325	6325	6325	17918	17918	17918	34371	22750
1978	7509	5999	5999	6562	5612	5612	5612	16514	16514	16514	31609	20600
1977	6842	5655	5655	6044	4978	4978	4978	15221	15221	15221	29069	21000
1976	5932	5365	5365	5567	4416	4416	4416	14029	14029	14029	26733	20500
1975	5484	5123	5123	5127	3918	3918	3918	12930	12930	12930	24585	20600
1974	5069	4923	4923	4723	3476	3476	3476	11917	11917	11917	22609	14800
1973	4686	4763	4763	4350	3084	3084	3084	10984	10984	10984	20792	17000
1972	4332	4637	4637	4006	2736	2736	2736	10123	10123	10123	19121	15500
1971	4005	4543	4543	3690	2427	2427	2427	9931	9931	9931	17585	13000

Ratio of Gasoline Annual VMT to Diesel Annual VMT

Year	LDV	I	IIA	IIB	III-V	VI	VII-VIII	VIIIA	VIIIB	BUSES
1990	0.7359	0.7766	0.8743	1.0342	0.77129	0.4144	0.4144	0.4144	0.2108	1
1989	0.753	0.8303	0.9229	1.0339	0.80045	0.414	0.414	0.414	0.2111	1
1988	0.7705	0.8819	0.9679	1.0335	0.83071	0.4135	0.4135	0.4135	0.2113	1
1987	0.7885	0.9309	1.0087	1.0331	0.86213	0.4131	0.4131	0.4131	0.2116	1
1986	0.8069	0.9761	1.0443	1.0328	0.89477	0.4126	0.4126	0.4126	0.2118	1
1985	0.8256	1.017	1.0744	1.0323	0.92855	0.4122	0.4122	0.4122	0.212	1
1984	0.844	1.0529	1.0982	1.032	0.96369	0.4118	0.4118	0.4118	0.2123	1
1983	0.8644	1.083	1.1154	1.0315	1.0001	0.4097	0.4097	0.4097	0.2125	1
1982	0.8846	1.1068	1.1254	1.0311	1.03797	0.4109	0.4109	0.4109	0.2128	1
1981	0.9051	1.1239	1.1283	1.0307	1.07714	0.4104	0.4104	0.4104	0.213	1
1980	0.9263	1.1338	1.1239	1.0304	1.11795	0.41	0.41	0.41	0.2133	1
1979	0.9478	1.1366	1.1124	1.03	1.16032	0.4096	0.4096	0.4096	0.2135	1
1978	0.8967	1.132	1.0938	1.0297	1.20403	0.4092	0.4092	0.4092	0.2138	1
1977	0.9309	1.1202	1.0688	1.0293	1.2497	0.4087	0.4087	0.4087	0.214	1
1976	1.0155	1.1014	1.0377	1.0289	1.2971	0.4083	0.4083	0.4083	0.2143	1
1975	1.039	1.0759	1.0008	1.0285	1.34584	0.4078	0.4078	0.4078	0.2145	1
1974	1.0633	1.0445	0.9594	1.0279	1.39672	0.4074	0.4074	0.4074	0.2147	1
1973	1.0881	1.0071	0.9133	1.0276	1.44942	0.407	0.407	0.407	0.215	1
1972	1.1133	0.9651	0.8639	1.0275	1.50439	0.4066	0.4066	0.4066	0.2153	1
1971	1.1391	0.9188	0.8122	1.0268	1.56119	0.3815	0.3815	0.3815	0.2155	1

Diesel Mile Fractions

Year	LDV	LDT I	LDT IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	0.0004	0.0001	0.0114	0.2733	0.52482	0.5744	0.2448	0.7835	0.9268	0.9996	1	1
1989	0.0005	0.0001	0.0162	0.2546	0	0.5554	0.807	0.7546	0.873	0.9996	1	1
1988	0	0.0017	0.0155	0.1987	0	0.6051	0.3189	0.5436	0.8495	0.9916	1	1
1987	0.0013	0.0054	0.0198	0.2115	0	0.5864	0.0803	0.4058	0.8048	0.9831	1	1
1986	0.0124	0.0051	0.0192	0.2824	0	0	0	0.406	0.7552	0.9831	1	1
1985	0.0205	0.0079	0.0233	0.3121	0.31579	0	0	0.5215	0.7701	0.9831	1	1
1984	0.0248	0.0095	0.0375	0.3023	0.27227	0.2723	0.2723	0.4604	0.748	0.9917	1	1
1983	0.0254	0.0185	0.0796	0.1617	0.23398	0.234	0.234	0.4217	0.749	0.9656	1	1
1982	0.0506	0.0463	0.0429	0.1922	0.19215	0.1921	0.1921	0.3928	0.7005	0.9744	1	1
1981	0.067	0.0736	0.032	0	0	0	0	0.317	0.7852	0.9745	1	1
1980	0.0463	0.1128	0.008	0	0	0	0	0.2316	0.7853	0.9789	1	1
1979	0.0211	0.0291	0	0	0	0	0	0.1751	0.7413	0.9565	1	1
1978	0.0134	0.0266	0	0	0	0	0	0.1554	0.7857	0.9472	1	1
1977	0.0043	0.0018	0.0009	0	0	0	0	0.0476	0.7643	0.9566	1	1
1976	0.003	0	0.0019	0	0	0	0	0.0704	0.6671	0.9278	1	1
1975	0	0	0	0	0	0	0	0.0242	0.6936	0.8512	1	1
1974	0	0	0	0	0	0	0	0.0477	0.58	0.8748	1	1
1973	0	0	0	0	0	0	0	0.0478	0.621	0.897	1	1
1972	0	0	0	0	0	0	0	0.0478	0.6212	0.8971	1	1
1971	0	0	0	0	0	0	0	0.0508	0.636	0.9028	1	1

3/26/1992

Diesel Market Shares

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	0.13	0.1	2	35	50	63	20	70	89	99.9	100	100
1994	0.13	0.05	1.6	34	50	62	20	68	88	99.9	100	100
1993	0.11	0.01	1.4	33	49	61	20	66	87	99.9	100	100
1992	0.09	0.01	1.2	32	48	60	20	64	86	99.9	100	100
1991	0.09	0.01	1	30	47	55	20	62	85	99.9	100	100
1990	0.03	0.01	1	28	46	51	20	60	84	99.9	100	100
1989	0.04	0.01	1.5	26.1	0	50	77	56	74	99.9	100	100
1988	0	0.15	1.5	20.4	0	56	28	33	70	98	100	100
1987	0.1	0.5	2	21.7	0	55	7	22	63	96	100	100
1986	1	0.5	2	28.9	0	0	0	22	56	96	100	100
1985	1.7	0.8	2.5	31.9	30	0	0	31	58	96	100	100
1984	2.1	1	4.1	30.9	26.5	26.5	26.5	26	55	98	100	100
1983	2.2	2	8.8	16.6	23.4	23.4	23.4	23	55	92	100	100
1982	4.5	5.1	4.8	19.7	19.8	19.8	19.8	21	49	94	100	100
1981	6.1	8.2	3.6	0	0	0	0	16	60	94	100	100
1980	4.3	12.6	0.9	0	0	0	0	11	60	95	100	100
1979	2	3.3	0	0	0	0	0	8	54	90	100	100
1978	1.2	3	0	0	0	0	0	7	60	88	100	100
1977	0.4	0.2	0.1	0	0	0	0	2	57	90	100	100
1976	0.3	0	0.2	0	0	0	0	3	45	84	100	100

Gasoline Annual Vehicle Miles Travelled

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	13118	15640	17608	18211	18211	18211	18211	18211	18211	18211	18211	58500
1994	12408	14590	16217	16767	16767	16767	16767	16767	16767	16767	16767	55400
1993	11737	13610	14937	15437	15437	15437	15437	15437	15437	15437	15437	54200
1992	11103	12696	13758	14213	14213	14213	14213	14213	14213	14213	14213	53400
1991	10503	11843	12671	13086	13086	13086	13086	13086	13086	13086	13086	53000
1990	9935	11048	11671	12048	12048	12048	12048	12048	12048	12048	12048	47600
1989	9389	10306	10749	11093	11093	11093	11093	11093	11093	11093	11093	37800
1988	8889	9614	9901	10213	10213	10213	10213	10213	10213	10213	10213	36400
1987	8409	8968	9119	9403	9403	9403	9403	9403	9403	9403	9403	32500
1986	7954	8366	8399	8657	8657	8657	8657	8657	8657	8657	8657	31500
1985	7524	7804	7736	7971	7971	7971	7971	7971	7971	7971	7971	26000
1984	7117	7280	7125	7339	7339	7339	7339	7339	7339	7339	7339	22750
1983	6733	6791	6562	6757	6757	6757	6757	6757	6757	6757	6757	20600
1982	6369	6335	6044	6221	6221	6221	6221	6221	6221	6221	6221	21000
1981	6024	5909	5567	5728	5728	5728	5728	5728	5728	5728	5728	20500
1980	5698	5512	5127	5273	5273	5273	5273	5273	5273	5273	5273	20600
1979	5390	5142	4723	4855	4855	4855	4855	4855	4855	4855	4855	14800
1978	5099	4797	4350	4470	4470	4470	4470	4470	4470	4470	4470	17000
1977	4823	4475	4006	4116	4116	4116	4116	4116	4116	4116	4116	15500
1976	4562	4174	3690	3789	3789	3789	3789	3789	3789	3789	3789	13000

Diesel Annual Vehicle Miles Travelled

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	17825	20140	20140	17608	23611	23611	23611	43946	43946	43946	86375	58500
1994	16478	17572	17572	16217	20947	20947	20947	40504	40504	40504	79434	55400
1993	15233	15432	15432	14937	18583	18583	18583	37332	37332	37332	73051	54200
1992	14081	13639	13639	13758	16486	16486	16486	34408	34408	34408	67181	53400
1991	13017	12133	12133	12671	14625	14625	14625	31713	31713	31713	61782	53000
1990	12033	10863	10863	11671	12975	12975	12975	29229	29229	29229	56817	47600
1989	11124	9788	9788	10749	11511	11511	11511	26939	26939	26939	52252	37800
1988	10283	8877	8877	9901	10212	10212	10212	24929	24929	24929	48053	36400
1987	9506	8103	8103	9119	9059	9059	9059	22885	22885	22885	44191	32500
1986	8788	7444	7444	8399	8037	8037	8037	21092	21092	21092	40640	31500
1985	8123	6883	6883	7736	7130	7130	7130	19440	19440	19440	37374	26000
1984	7509	6405	6405	7125	6325	6325	6325	17918	17918	17918	34371	22750
1983	7509	5999	5999	6562	5612	5612	5612	16514	16514	16514	31609	20600
1982	6842	5655	5655	6044	4978	4978	4978	15221	15221	15221	29069	21000
1981	5932	5365	5365	5567	4416	4416	4416	14029	14029	14029	26733	20500
1980	5484	5123	5123	5127	3918	3918	3918	12930	12930	12930	24585	20600
1979	5069	4923	4923	4723	3476	3476	3476	11917	11917	11917	22609	14800
1978	4686	4763	4763	4350	3084	3084	3084	10984	10984	10984	20792	17000
1977	4332	4637	4637	4006	2736	2736	2736	10123	10123	10123	19121	15500
1976	4005	4543	4543	3690	2427	2427	2427	9931	9931	9931	17585	13000

Ratio of Gasoline Annual VMT to Diesel Annual VMT

Year	LDV	I	IIA	IIB	III-V	VI	VII-VIII	VIIIA	VIIIB	BUSES
1995	0.7359	0.7766	0.8743	1.0342	0.77129	0.4144	0.4144	0.4144	0.2108	1
1994	0.753	0.8303	0.9229	1.0339	0.80045	0.414	0.414	0.414	0.2111	1
1993	0.7705	0.8819	0.9679	1.0335	0.83071	0.4135	0.4135	0.4135	0.2113	1
1992	0.7885	0.9309	1.0087	1.0331	0.86213	0.4131	0.4131	0.4131	0.2116	1
1991	0.8069	0.9761	1.0443	1.0328	0.89477	0.4126	0.4126	0.4126	0.2118	1
1990	0.8256	1.017	1.0744	1.0323	0.92855	0.4122	0.4122	0.4122	0.212	1
1989	0.844	1.0529	1.0982	1.032	0.96369	0.4118	0.4118	0.4118	0.2123	1
1988	0.8644	1.083	1.1154	1.0315	1.0001	0.4097	0.4097	0.4097	0.2125	1
1987	0.8846	1.1068	1.1254	1.0311	1.03797	0.4109	0.4109	0.4109	0.2128	1
1986	0.9051	1.1239	1.1283	1.0307	1.07714	0.4104	0.4104	0.4104	0.213	1
1985	0.9263	1.1338	1.1239	1.0304	1.11795	0.41	0.41	0.41	0.2133	1
1984	0.9478	1.1366	1.1124	1.03	1.16032	0.4096	0.4096	0.4096	0.2135	1
1983	0.8967	1.132	1.0938	1.0297	1.20403	0.4092	0.4092	0.4092	0.2138	1
1982	0.9309	1.1202	1.0688	1.0293	1.2497	0.4087	0.4087	0.4087	0.214	1
1981	1.0155	1.1014	1.0377	1.0289	1.2971	0.4083	0.4083	0.4083	0.2143	1
1980	1.039	1.0759	1.0008	1.0285	1.34584	0.4078	0.4078	0.4078	0.2145	1
1979	1.0633	1.0445	0.9594	1.0279	1.39672	0.4074	0.4074	0.4074	0.2147	1
1978	1.0881	1.0071	0.9133	1.0276	1.44942	0.407	0.407	0.407	0.215	1
1977	1.1133	0.9651	0.8639	1.0275	1.50439	0.4066	0.4066	0.4066	0.2153	1
1976	1.1391	0.9188	0.8122	1.0268	1.56119	0.3815	0.3815	0.3815	0.2155	1

Diesel Mile Fractions

Year	LDV	LDT I	LDT IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	0.0018	0.0013	0.0228	0.3424	0.56456	0.6882	0.2448	0.8492	0.9513	0.9996	1	1
1994	0.0017	0.0006	0.0173	0.3326	0.55542	0.6709	0.238	0.837	0.9466	0.9996	1	1
1993	0.0014	0.0001	0.0145	0.3228	0.5363	0.6531	0.2313	0.8244	0.9418	0.9996	1	1
1992	0.0011	0.0001	0.0119	0.313	0.51707	0.635	0.2248	0.8115	0.937	0.9996	1	1
1991	0.0011	0.0001	0.0096	0.2933	0.49776	0.5773	0.2184	0.7981	0.9321	0.9996	1	1
1990	0.0004	0.0001	0.0093	0.2736	0.47846	0.5285	0.2121	0.7844	0.9272	0.9996	1	1
1989	0.0005	0.0001	0.0137	0.255	0	0.5092	0.7765	0.7555	0.8736	0.9996	1	1
1988	0	0.0014	0.0135	0.199	0	0.56	0.28	0.5459	0.8506	0.9917	1	1
1987	0.0011	0.0045	0.0178	0.2118	0	0.5408	0.0676	0.407	0.8056	0.9832	1	1
1986	0.011	0.0045	0.0178	0.2828	0	0	0	0.4073	0.7562	0.9832	1	1
1985	0.0183	0.0071	0.0223	0.3125	0.27712	0	0	0.5228	0.7711	0.9832	1	1
1984	0.0221	0.0088	0.037	0.3027	0.23707	0.2371	0.2371	0.4617	0.749	0.9917	1	1
1983	0.0245	0.0177	0.0811	0.162	0.20237	0.2024	0.2024	0.422	0.7492	0.9656	1	1
1982	0.0482	0.0458	0.045	0.1925	0.16496	0.165	0.165	0.3941	0.7016	0.9746	1	1
1981	0.0601	0.075	0.0347	0	0	0	0	0.3181	0.786	0.9746	1	1
1980	0.0415	0.1182	0.009	0	0	0	0	0.2326	0.7862	0.979	1	1
1979	0.0188	0.0316	0	0	0	0	0	0.1759	0.7424	0.9567	1	1
1978	0.011	0.0298	0	0	0	0	0	0.1561	0.7866	0.9474	1	1
1977	0.0036	0.0021	0.0012	0	0	0	0	0.0478	0.7653	0.9568	1	1
1976	0.0026	0	0.0025	0	0	0	0	0.075	0.682	0.9323	1	1

3/26/1992

Diesel Market Shares

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	0.2	0.6	4.2	40	50	68	20	80	90	99.9	100	100
1999	0.2	0.5	4	39	50	67	20	78	90	99.9	100	100
1998	0.2	0.4	3.5	38	50	66	20	76	90	99.9	100	100
1997	0.2	0.3	3	37	50	65	20	74	90	99.9	100	100
1996	0.14	0.2	2.5	36	50	64	20	72	90	99.9	100	100
1995	0.13	0.1	2	35	50	63	20	70	89	99.9	100	100
1994	0.13	0.05	1.6	34	50	62	20	68	88	99.9	100	100
1993	0.11	0.01	1.4	33	49	61	20	66	87	99.9	100	100
1992	0.09	0.01	1.2	32	48	60	20	64	86	99.9	100	100
1991	0.09	0.01	1	30	47	55	20	62	85	99.9	100	100
1990	0.03	0.01	1	28	46	51	20	60	84	99.9	100	100
1989	0.04	0.01	1.5	26.1	0	50	77	56	74	99.9	100	100
1988	0	0.15	1.5	20.4	0	56	28	33	70	98	100	100
1987	0.1	0.5	2	21.7	0	55	7	22	63	96	100	100
1986	1	0.5	2	28.9	0	0	0	22	56	96	100	100
1985	1.7	0.8	2.5	31.9	30	0	0	31	58	96	100	100
1984	2.1	1	4.1	30.9	26.5	26.5	26.5	26	55	98	100	100
1983	2.2	2	8.8	16.6	23.4	23.4	23.4	23	55	92	100	100
1982	4.5	5.1	4.8	19.7	19.8	19.8	19.8	21	49	94	100	100
1981	6.1	8.2	3.6	0	0	0	0	16	60	94	100	100

Gasoline Annual Vehicle Miles Travelled

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	13118	15640	17608	18211	18211	18211	18211	18211	18211	18211	18211	58500
1999	12408	14590	16217	16767	16767	16767	16767	16767	16767	16767	16767	55400
1998	11737	13610	14937	15437	15437	15437	15437	15437	15437	15437	15437	54200
1997	11103	12696	13758	14213	14213	14213	14213	14213	14213	14213	14213	53400
1996	10503	11843	12671	13086	13086	13086	13086	13086	13086	13086	13086	53000
1995	9935	11048	11671	12048	12048	12048	12048	12048	12048	12048	12048	47600
1994	9389	10306	10749	11093	11093	11093	11093	11093	11093	11093	11093	37800
1993	8889	9614	9901	10213	10213	10213	10213	10213	10213	10213	10213	36400
1992	8409	8968	9119	9403	9403	9403	9403	9403	9403	9403	9403	32500
1991	7954	8366	8399	8657	8657	8657	8657	8657	8657	8657	8657	31500
1990	7524	7804	7736	7971	7971	7971	7971	7971	7971	7971	7971	26000
1989	7117	7280	7125	7339	7339	7339	7339	7339	7339	7339	7339	22750
1988	6733	6791	6562	6757	6757	6757	6757	6757	6757	6757	6757	20600
1987	6369	6335	6044	6221	6221	6221	6221	6221	6221	6221	6221	21000
1986	6024	5909	5567	5728	5728	5728	5728	5728	5728	5728	5728	20500
1985	5698	5512	5127	5273	5273	5273	5273	5273	5273	5273	5273	20600
1984	5390	5142	4723	4855	4855	4855	4855	4855	4855	4855	4855	14800
1983	5099	4797	4350	4470	4470	4470	4470	4470	4470	4470	4470	17000
1982	4823	4475	4006	4116	4116	4116	4116	4116	4116	4116	4116	15500
1981	4562	4174	3690	3789	3789	3789	3789	3789	3789	3789	3789	13000

Diesel Annual Vehicle Miles Travelled

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	17825	20140	20140	17608	23611	23611	23611	43946	43946	43946	86375	58500
1999	16478	17572	17572	16217	20947	20947	20947	40504	40504	40504	79434	55400
1998	15233	15432	15432	14937	18583	18583	18583	37332	37332	37332	73051	54200
1997	14081	13639	13639	13758	16486	16486	16486	34408	34408	34408	67181	53400
1996	13017	12133	12133	12671	14625	14625	14625	31713	31713	31713	61782	53000
1995	12033	10863	10863	11671	12975	12975	12975	29229	29229	29229	56817	47600
1994	11124	9788	9788	10749	11511	11511	11511	26939	26939	26939	52252	37800
1993	10283	8877	8877	9901	10212	10212	10212	24929	24929	24929	48053	36400
1992	9506	8103	8103	9119	9059	9059	9059	22885	22885	22885	44191	32500
1991	8788	7444	7444	8399	8037	8037	8037	21092	21092	21092	40640	31500
1990	8123	6883	6883	7736	7130	7130	7130	19440	19440	19440	37374	26000
1989	7509	6405	6405	7125	6325	6325	6325	17918	17918	17918	34371	22750
1988	7509	5999	5999	6562	5612	5612	5612	16514	16514	16514	31609	20600
1987	6842	5655	5655	6044	4978	4978	4978	15221	15221	15221	29069	21000
1986	5932	5365	5365	5567	4416	4416	4416	14029	14029	14029	26733	20500
1985	5484	5123	5123	5127	3918	3918	3918	12930	12930	12930	24585	20600
1984	5069	4923	4923	4723	3476	3476	3476	11917	11917	11917	22609	14800
1983	4686	4763	4763	4350	3084	3084	3084	10984	10984	10984	20792	17000
1982	4332	4637	4637	4006	2736	2736	2736	10123	10123	10123	19121	15500
1981	4005	4543	4543	3690	2427	2427	2427	9931	9931	9931	17585	13000

Ratio of Gasoline Annual VMT to Diesel Annual VMT

Year	LDV	I	IIA	IIB	III-V	VI	VII-VIII	VIIIA	VIIIB	BUSES
1995	0.7359	0.7766	0.8743	1.0342	0.77129	0.4144	0.4144	0.4144	0.2108	1
1994	0.753	0.8303	0.9229	1.0339	0.80045	0.414	0.414	0.414	0.2111	1
1993	0.7705	0.8819	0.9679	1.0335	0.83071	0.4135	0.4135	0.4135	0.2113	1
1992	0.7885	0.9309	1.0087	1.0331	0.86213	0.4131	0.4131	0.4131	0.2116	1
1991	0.8069	0.9761	1.0443	1.0328	0.89477	0.4126	0.4126	0.4126	0.2118	1
1990	0.8256	1.017	1.0744	1.0323	0.92855	0.4122	0.4122	0.4122	0.212	1
1989	0.844	1.0529	1.0982	1.032	0.96369	0.4118	0.4118	0.4118	0.2123	1
1988	0.8644	1.083	1.1154	1.0315	1.0001	0.4097	0.4097	0.4097	0.2125	1
1987	0.8846	1.1068	1.1254	1.0311	1.03797	0.4109	0.4109	0.4109	0.2128	1
1986	0.9051	1.1239	1.1283	1.0307	1.07714	0.4104	0.4104	0.4104	0.213	1
1985	0.9263	1.1338	1.1239	1.0304	1.11795	0.41	0.41	0.41	0.2133	1
1984	0.9478	1.1366	1.1124	1.03	1.16032	0.4096	0.4096	0.4096	0.2135	1
1983	0.8967	1.132	1.0938	1.0297	1.20403	0.4092	0.4092	0.4092	0.2138	1
1982	0.9309	1.1202	1.0688	1.0293	1.2497	0.4087	0.4087	0.4087	0.214	1
1981	1.0155	1.1014	1.0377	1.0289	1.2971	0.4083	0.4083	0.4083	0.2143	1
1980	1.039	1.0759	1.0008	1.0285	1.34584	0.4078	0.4078	0.4078	0.2145	1
1979	1.0633	1.0445	0.9594	1.0279	1.39672	0.4074	0.4074	0.4074	0.2147	1
1978	1.0881	1.0071	0.9133	1.0276	1.44942	0.407	0.407	0.407	0.215	1
1977	1.1133	0.9651	0.8639	1.0275	1.50439	0.4066	0.4066	0.4066	0.2153	1
1976	1.1391	0.9188	0.8122	1.0268	1.56119	0.3815	0.3815	0.3815	0.2155	1

Diesel Mile Fractions

Year	LDV	LDT I	LDT IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	0.0027	0.0077	0.0478	0.3919	0.56456	0.7337	0.2448	0.9061	0.956	0.9996	1	1
1994	0.0027	0.006	0.0432	0.3821	0.55542	0.7172	0.238	0.8954	0.956	0.9996	1	1
1993	0.0026	0.0045	0.0361	0.3723	0.54624	0.7003	0.2313	0.8845	0.9561	0.9996	1	1
1992	0.0025	0.0032	0.0297	0.3624	0.53702	0.683	0.2248	0.8733	0.9561	0.9996	1	1
1991	0.0017	0.002	0.024	0.3526	0.52777	0.6652	0.2184	0.8617	0.9562	0.9996	1	1
1990	0.0016	0.001	0.0186	0.3428	0.51852	0.6471	0.2121	0.8499	0.9515	0.9996	1	1
1989	0.0015	0.0005	0.0146	0.333	0.50925	0.6287	0.206	0.8377	0.9468	0.9996	1	1
1988	0.0013	0.0001	0.0126	0.3232	0.48998	0.61	0.2	0.8257	0.9423	0.9996	1	1
1987	0.001	0.0001	0.0107	0.3134	0.47071	0.591	0.1941	0.8123	0.9373	0.9996	1	1
1986	0.001	0.0001	0.0089	0.2937	0.45154	0.5315	0.1884	0.799	0.9325	0.9996	1	1
1985	0.0003	0.0001	0.0089	0.274	0.43246	0.4821	0.1828	0.7853	0.9276	0.9996	1	1
1984	0.0004	0.0001	0.0135	0.2553	0	0.4629	0.7426	0.7565	0.8742	0.9996	1	1
1983	0	0.0013	0.0137	0.1993	0	0.5139	0.2441	0.5462	0.8508	0.9917	1	1
1982	0.0011	0.0045	0.0187	0.2121	0	0.4944	0.0568	0.4083	0.8064	0.9833	1	1
1981	0.0098	0.0045	0.0193	0.2832	0	0	0	0.4086	0.7571	0.9833	1	1
1980	0.0164	0.0074	0.025	0.3129	0.24153	0	0	0.5242	0.772	0.9833	1	1
1979	0.0198	0.0096	0.0427	0.3031	0.20517	0.2052	0.2052	0.4631	0.75	0.9918	1	1
1978	0.0203	0.0199	0.0956	0.1623	0.17407	0.1741	0.1741	0.4233	0.7502	0.9658	1	1
1977	0.0406	0.0527	0.0551	0.1927	0.14097	0.141	0.141	0.3953	0.7026	0.9747	1	1
1976	0.054	0.0886	0.044	0	0	0	0	0.333	0.7972	0.9762	1	1

4/28/1992

Diesel Market Shares

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	0.3	1.5	6.2	47	50	75	20	80	90	99.9	100	100
2009	0.3	1.4	6	47	50	75	20	80	90	99.9	100	100
2008	0.3	1.3	5.6	47	50	75	20	80	90	99.9	100	100
2007	0.3	1.2	5.6	46	50	75	20	80	90	99.9	100	100
2006	0.3	1.1	5.4	46	50	74	20	80	90	99.9	100	100
2005	0.3	1.1	5.2	45	50	73	20	80	90	99.9	100	100
2004	0.3	1	5	44	50	72	20	80	90	99.9	100	100
2003	0.3	0.9	4.8	43	50	71	20	80	90	99.9	100	100
2002	0.3	0.8	4.6	42	50	70	20	80	90	99.9	100	100
2001	0.3	0.7	4.4	41	50	69	20	80	90	99.9	100	100
2000	0.2	0.6	4.2	40	50	68	20	80	90	99.9	100	100
1999	0.2	0.5	4	39	50	67	20	78	90	99.9	100	100
1998	0.2	0.4	3.5	38	50	66	20	76	90	99.9	100	100
1997	0.2	0.3	3	37	50	65	20	74	90	99.9	100	100
1996	0.14	0.2	2.5	36	50	64	20	72	90	99.9	100	100
1995	0.13	0.1	2	35	50	63	20	70	89	99.9	100	100
1994	0.13	0.05	1.6	34	50	62	20	68	88	99.9	100	100
1993	0.11	0.01	1.4	33	49	61	20	66	87	99.9	100	100
1992	0.09	0.01	1.2	32	48	60	20	64	86	99.9	100	100
1991	0.09	0.01	1	30	47	55	20	62	85	99.9	100	100

Gasoline Annual Vehicle Miles Travelled

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	13118	15640	17608	18211	18211	18211	18211	18211	18211	18211	18211	58500
2009	12408	14590	16217	16767	16767	16767	16767	16767	16767	16767	16767	55400
2008	11737	13610	14937	15437	15437	15437	15437	15437	15437	15437	15437	54200
2007	11103	12696	13758	14213	14213	14213	14213	14213	14213	14213	14213	53400
2006	10503	11843	12671	13086	13086	13086	13086	13086	13086	13086	13086	53000
2005	9935	11048	11671	12048	12048	12048	12048	12048	12048	12048	12048	47600
2004	9389	10306	10749	11093	11093	11093	11093	11093	11093	11093	11093	37800
2003	8889	9614	9901	10213	10213	10213	10213	10213	10213	10213	10213	36400
2002	8409	8968	9119	9403	9403	9403	9403	9403	9403	9403	9403	32500
2001	7954	8366	8399	8657	8657	8657	8657	8657	8657	8657	8657	31500
2000	7524	7804	7736	7971	7971	7971	7971	7971	7971	7971	7971	26000
1999	7117	7280	7125	7339	7339	7339	7339	7339	7339	7339	7339	22750
1998	6733	6791	6562	6757	6757	6757	6757	6757	6757	6757	6757	20600
1997	6369	6335	6044	6221	6221	6221	6221	6221	6221	6221	6221	21000
1996	6024	5909	5567	5728	5728	5728	5728	5728	5728	5728	5728	20500
1995	5698	5512	5127	5273	5273	5273	5273	5273	5273	5273	5273	20600
1994	5390	5142	4723	4855	4855	4855	4855	4855	4855	4855	4855	14800
1993	5099	4797	4350	4470	4470	4470	4470	4470	4470	4470	4470	17000
1992	4823	4475	4006	4116	4116	4116	4116	4116	4116	4116	4116	15500
1991	4562	4174	3690	3789	3789	3789	3789	3789	3789	3789	3789	13000

Diesel Annual Vehicle Miles Travelled

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	17825	20140	20140	17608	23611	23611	23611	43946	43946	43946	86375	58500
2009	16478	17572	17572	16217	20947	20947	20947	40504	40504	40504	79434	55400
2008	15233	15432	15432	14937	18583	18583	18583	37332	37332	37332	73051	54200
2007	14081	13639	13639	13758	16486	16486	16486	34408	34408	34408	67181	53400
2006	13017	12133	12133	12671	14625	14625	14625	31713	31713	31713	61782	53000
2005	12033	10863	10863	11671	12975	12975	12975	29229	29229	29229	56817	47600
2004	11124	9788	9788	10749	11511	11511	11511	26939	26939	26939	52252	37800
2003	10283	8877	8877	9901	10212	10212	10212	24929	24929	24929	48053	36400
2002	9506	8103	8103	9119	9059	9059	9059	22885	22885	22885	44191	32500
2001	8788	7444	7444	8399	8037	8037	8037	21092	21092	21092	40640	31500
2000	8123	6883	6883	7736	7130	7130	7130	19440	19440	19440	37374	26000
1999	7509	6405	6405	7125	6325	6325	6325	17918	17918	17918	34371	22750
1998	7509	5999	5999	6562	5612	5612	5612	16514	16514	16514	31609	20600
1997	6842	5655	5655	6044	4978	4978	4978	15221	15221	15221	29069	21000
1996	5932	5365	5365	5567	4416	4416	4416	14029	14029	14029	26733	20500
1995	5484	5123	5123	5127	3918	3918	3918	12930	12930	12930	24585	20600
1994	5069	4923	4923	4723	3476	3476	3476	11917	11917	11917	22609	14800
1993	4686	4763	4763	4350	3084	3084	3084	10984	10984	10984	20792	17000
1992	4332	4637	4637	4006	2736	2736	2736	10123	10123	10123	19121	15500
1991	4005	4543	4543	3690	2427	2427	2427	9931	9931	9931	17585	13000

Ratio of Gasoline Annual VMT to Diesel Annual VMT

Year	LDV	I	IIA	IIB	III-V	VI	VII-VIII	VIIIA	VIIIB	BUSES
2010	0.7359	0.7766	0.8743	1.0342	0.77129	0.4144	0.4144	0.4144	0.2108	1
2009	0.753	0.8303	0.9229	1.0339	0.80045	0.414	0.414	0.414	0.2111	1
2008	0.7705	0.8819	0.9679	1.0335	0.83071	0.4135	0.4135	0.4135	0.2113	1
2007	0.7885	0.9309	1.0087	1.0331	0.86213	0.4131	0.4131	0.4131	0.2116	1
2006	0.8069	0.9761	1.0443	1.0328	0.89477	0.4126	0.4126	0.4126	0.2118	1
2005	0.8256	1.017	1.0744	1.0323	0.92855	0.4122	0.4122	0.4122	0.212	1
2004	0.844	1.0529	1.0982	1.032	0.96369	0.4118	0.4118	0.4118	0.2123	1
2003	0.8644	1.083	1.1154	1.0315	1.0001	0.4097	0.4097	0.4097	0.2125	1
2002	0.8846	1.1068	1.1254	1.0311	1.03797	0.4109	0.4109	0.4109	0.2128	1
2001	0.9051	1.1239	1.1283	1.0307	1.07714	0.4104	0.4104	0.4104	0.213	1
2000	0.9263	1.1338	1.1239	1.0304	1.11795	0.41	0.41	0.41	0.2133	1
1999	0.9478	1.1366	1.1124	1.03	1.16032	0.4096	0.4096	0.4096	0.2135	1
1998	0.8967	1.132	1.0938	1.0297	1.20403	0.4092	0.4092	0.4092	0.2138	1
1997	0.9309	1.1202	1.0688	1.0293	1.2497	0.4087	0.4087	0.4087	0.214	1
1996	1.0155	1.1014	1.0377	1.0289	1.2971	0.4083	0.4083	0.4083	0.2143	1
1995	1.039	1.0759	1.0008	1.0285	1.34584	0.4078	0.4078	0.4078	0.2145	1
1994	1.0633	1.0445	0.9594	1.0279	1.39672	0.4074	0.4074	0.4074	0.2147	1
1993	1.0881	1.0071	0.9133	1.0276	1.44942	0.407	0.407	0.407	0.215	1
1992	1.1133	0.9651	0.8639	1.0275	1.50439	0.4066	0.4066	0.4066	0.2153	1
1991	1.1391	0.9188	0.8122	1.0268	1.56119	0.3815	0.3815	0.3815	0.2155	1

Diesel Mile Fractions

Year	LDV	LDT I	LDT IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	0.0041	0.0192	0.0703	0.4616	0.56456	0.7955	0.2448	0.9061	0.956	0.9996	1	1
1994	0.004	0.0168	0.0647	0.4617	0.55542	0.7894	0.238	0.9062	0.956	0.9996	1	1
1993	0.0039	0.0147	0.0577	0.4618	0.54624	0.7831	0.2313	0.9063	0.9561	0.9996	1	1
1992	0.0038	0.0129	0.0555	0.4519	0.53702	0.7768	0.2248	0.9064	0.9561	0.9996	1	1
1991	0.0037	0.0113	0.0518	0.452	0.52777	0.7608	0.2184	0.9065	0.9562	0.9996	1	1
1990	0.0036	0.0108	0.0486	0.4421	0.51852	0.7444	0.2121	0.9066	0.9562	0.9996	1	1
1989	0.0036	0.0095	0.0457	0.4323	0.50925	0.7274	0.206	0.9067	0.9562	0.9996	1	1
1988	0.0035	0.0083	0.0433	0.4224	0.49998	0.71	0.2	0.9071	0.9565	0.9996	1	1
1987	0.0034	0.0072	0.0411	0.4125	0.49068	0.6921	0.1941	0.9068	0.9563	0.9996	1	1
1986	0.0033	0.0062	0.0392	0.4027	0.48143	0.6739	0.1884	0.9069	0.9564	0.9996	1	1
1985	0.0022	0.0053	0.0375	0.3928	0.47215	0.6553	0.1828	0.907	0.9564	0.9996	1	1
1984	0.0021	0.0044	0.0361	0.383	0.4629	0.6363	0.1773	0.8964	0.9565	0.9996	1	1
1983	0.0022	0.0035	0.0321	0.3731	0.45371	0.6172	0.1719	0.8856	0.9565	0.9996	1	1
1982	0.0021	0.0027	0.0281	0.3633	0.4445	0.5978	0.1667	0.8744	0.9566	0.9996	1	1
1981	0.0014	0.0018	0.0241	0.3535	0.43533	0.5782	0.1616	0.863	0.9566	0.9996	1	1
1980	0.0013	0.0009	0.02	0.3436	0.42629	0.5585	0.1567	0.8512	0.952	0.9996	1	1
1979	0.0012	0.0005	0.0167	0.3338	0.41724	0.5388	0.1518	0.8391	0.9474	0.9996	1	1
1978	0.001	0.0001	0.0153	0.324	0.39863	0.519	0.1471	0.8267	0.9427	0.9996	1	1
1977	0.0008	0.0001	0.0139	0.3141	0.38026	0.4993	0.1425	0.8139	0.9379	0.9996	1	1
1976	0.0008	0.0001	0.0123	0.2945	0.36225	0.4391	0.138	0.8105	0.9369	0.9996	1	1

4/28/1992

Vehicle Emission Rates (g/mi - light duty, g/BHP-hr - heavy duty)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	0.200	0.260	0.450	0.480	0.440	0.440	0.440	0.440	0.440	0.440	0.420	0.420
1989	0.200	0.260	0.450	0.510	0.450	0.450	0.450	0.450	0.450	0.450	0.440	0.440
1988	0.200	0.260	0.450	0.540	0.450	0.450	0.450	0.450	0.450	0.450	0.410	0.410
1987	0.200	0.260	0.260	0.690	0.690	0.690	0.690	0.623	0.623	0.431	0.431	0.431
1986	0.240	0.240	0.240	0.690	0.690	0.690	0.690	0.623	0.623	0.431	0.431	0.431
1985	0.270	0.320	0.320	0.690	0.690	0.690	0.690	0.623	0.623	0.431	0.431	0.431
1984	0.300	0.320	0.320	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1983	0.340	0.280	0.280	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1982	0.310	0.290	0.290	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1981	0.350	0.350	0.350	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1980	0.400	0.400	0.400	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1979	0.450	0.450	0.450	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1978	0.500	0.500	0.500	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1977	0.550	0.550	0.550	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1976	0.600	0.600	0.600	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1975	0.600	0.600	0.600	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1974	0.600	0.600	0.600	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1973	0.600	0.600	0.600	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1972	0.600	0.600	0.600	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1971	0.600	0.600	0.600	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751

Heavy Duty Conversion Factors

Year	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	0.856	1.650	1.650	1.650	1.825	2.077	2.926	2.912	3.241
1989	0.873	1.682	1.682	1.682	1.840	2.093	2.949	2.946	3.241
1988	0.890	1.714	1.714	1.714	1.853	2.109	2.971	2.974	3.241
1987	0.906	1.747	1.747	1.747	1.868	2.126	2.995	3.010	3.241
1986	0.909	1.752	1.752	1.752	1.882	2.141	3.016	3.046	3.241
1985	0.912	1.758	1.758	1.758	1.894	2.155	3.036	3.076	3.241
1984	0.916	1.766	1.766	1.766	1.904	2.167	3.052	3.103	3.241
1983	0.919	1.772	1.772	1.772	1.916	2.181	3.072	3.120	3.241
1982	0.923	1.779	1.779	1.779	1.927	2.193	3.089	3.152	3.241
1981	0.942	1.776	1.776	1.776	1.992	2.229	3.106	3.255	3.172
1980	0.000	1.773	1.773	1.773	2.060	2.254	3.062	3.332	3.069
1979	0.000	1.770	1.770	1.770	2.135	2.412	3.085	3.307	3.027
1978	0.000	1.768	1.768	1.768	2.216	2.616	3.126	3.361	3.025
1977	0.000	1.765	1.765	1.765	2.303	2.753	3.180	3.402	2.990
1976	0.000	1.762	1.762	1.762	2.342	2.760	3.156	3.353	2.935
1975	0.000	1.879	1.879	1.879	2.442	2.780	3.098	3.299	3.076
1974	0.000	2.009	2.009	2.009	2.551	2.775	3.104	3.326	2.962
1973	0.000	2.161	2.161	2.161	2.601	2.760	3.031	3.275	3.136
1972	0.000	2.161	2.161	2.161	2.608	2.701	3.031	3.275	3.342
1971	0.000	2.161	2.161	2.161	2.554	2.701	3.031	3.275	3.144

Vehicle Emission Rates (g/mi - all vehicle classes)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	0.200	0.260	0.450	0.411	0.726	0.726	0.726	0.803	0.914	1.287	1.223	1.361
1989	0.200	0.260	0.450	0.445	0.757	0.757	0.757	0.828	0.942	1.327	1.296	1.426
1988	0.200	0.260	0.450	0.480	0.772	0.772	0.772	0.834	0.949	1.337	1.219	1.329
1987	0.200	0.260	0.260	0.625	1.205	1.205	1.205	1.164	1.325	1.291	1.297	1.397
1986	0.240	0.240	0.240	0.627	1.209	1.209	1.209	1.172	1.334	1.300	1.313	1.397
1985	0.270	0.320	0.320	0.629	1.213	1.213	1.213	1.180	1.343	1.308	1.326	1.397
1984	0.300	0.320	0.320	0.614	1.183	1.183	1.183	1.339	1.523	2.292	2.331	2.434
1983	0.340	0.280	0.280	0.616	1.187	1.187	1.187	1.347	1.533	2.307	2.343	2.434

Year												
1982	0.310	0.290	0.290	0.618	1.192	1.192	1.192	1.355	1.542	2.320	2.367	2.434
1981	0.350	0.350	0.350	0.631	1.190	1.190	1.190	1.400	1.567	2.333	2.445	2.382
1980	0.400	0.400	0.400	0.000	1.188	1.188	1.188	1.448	1.585	2.300	2.502	2.305
1979	0.450	0.450	0.450	0.000	1.186	1.186	1.186	1.501	1.696	2.317	2.484	2.273
1978	0.500	0.500	0.500	0.000	1.185	1.185	1.185	1.558	1.839	2.348	2.524	2.272
1977	0.550	0.550	0.550	0.000	1.183	1.183	1.183	1.619	1.935	2.388	2.555	2.245
1976	0.600	0.600	0.600	0.000	1.181	1.181	1.181	1.646	1.940	2.370	2.518	2.204
1975	0.600	0.600	0.600	0.000	1.259	1.259	1.259	1.717	1.954	2.327	2.478	2.310
1974	0.600	0.600	0.600	0.000	1.346	1.346	1.346	1.793	1.951	2.331	2.498	2.224
1973	0.600	0.600	0.600	0.000	1.448	1.448	1.448	1.828	1.940	2.276	2.460	2.355
1972	0.600	0.600	0.600	0.000	1.448	1.448	1.448	1.833	1.899	2.276	2.460	2.510
1971	0.600	0.600	0.600	0.000	1.448	1.448	1.448	1.795	1.899	2.276	2.460	2.361

Urban Diesel Particulate Emissions (billions of grams)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	0.006	0.001	0.034	0.544	0.074	0.108	0.008	0.429	1.468	0.691	2.533	0.318
1989	0.008	0.001	0.052	0.594	0.000	0.096	0.039	0.388	1.341	0.783	2.943	0.331
1988	0.000	0.008	0.046	0.469	0.000	0.075	0.015	0.289	1.290	0.724	2.587	0.217
1987	0.017	0.023	0.027	0.520	0.000	0.010	0.005	0.201	1.463	0.549	2.200	0.313
1986	0.202	0.017	0.023	0.648	0.000	0.000	0.000	0.184	1.150	0.426	1.709	0.248
1985	0.329	0.030	0.035	0.681	0.021	0.000	0.000	0.247	1.015	0.446	1.794	0.221
1984	0.366	0.028	0.048	0.547	0.009	0.000	0.007	0.236	0.760	0.831	2.663	0.349
1983	0.315	0.027	0.077	0.257	0.000	0.000	0.001	0.150	0.493	0.043	1.818	0.345
1982	0.400	0.053	0.030	0.213	0.001	0.000	0.001	0.108	0.387	0.226	1.173	0.219
1981	0.486	0.071	0.021	0.000	0.000	0.000	0.000	0.115	0.328	0.297	1.251	0.276
1980	0.294	0.118	0.006	0.000	0.000	0.000	0.000	0.084	0.309	0.330	1.160	0.239
1979	0.126	0.038	0.000	0.000	0.000	0.000	0.000	0.083	0.209	0.381	1.383	0.148
1978	0.066	0.034	0.000	0.000	0.000	0.000	0.000	0.065	0.166	0.313	1.011	0.140
1977	0.016	0.002	0.001	0.000	0.000	0.000	0.000	0.016	0.091	0.169	0.798	0.085
1976	0.007	0.000	0.001	0.000	0.000	0.000	0.000	0.019	0.045	0.101	0.409	0.148
1975	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.005	0.040	0.093	0.211	0.160
1974	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.012	0.032	0.098	0.370	0.094
1973	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.011	0.034	0.100	0.277	0.069
1972	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.007	0.026	0.073	0.166	0.057
1971	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.004	0.021	0.040	0.115	0.036
Total DP	2.637	0.452	0.399	4.474	0.105	0.289	0.076	2.651	10.669	6.715	26.572	4.011

Adjustment of Particulate Mass For Freeway Road Use

$$DPAdj = DP *[(1-FMF) * (4/3 - 1/3*FFR) + FMF * FFR]$$

FMF	0.25	0.25	0.25	0.25	0.22	0.22	0.22	0.39	0.45	0.39	0.73	0.1
FFR	0.69	0.69	0.69	0.69	0.63	0.63	0.63	0.63	0.63	0.63	0.58	0.58
DPAdj=	2.637	0.452	0.399	4.474	0.107	0.293	0.077	2.468	9.617	6.251	19.430	4.348

Grand total billions of grams of particulates in urban areas = 50.55154

Total Urban Fleet Vehicle Miles Travelled (billions of miles) => 882.027

National fleet average emission factor (g/mi)= 0.0573

03/27/1992

Vehicle Emission Rates (g/mi - light duty, g/BHP-hr - heavy duty)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	0.100	0.100	0.130	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1994	0.200	0.260	0.130	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1993	0.200	0.260	0.130	0.230	0.220	0.220	0.220	0.220	0.220	0.220	0.270	0.084
1992	0.200	0.260	0.130	0.230	0.220	0.220	0.220	0.220	0.220	0.220	0.270	0.270
1991	0.200	0.260	0.130	0.230	0.220	0.220	0.220	0.220	0.220	0.220	0.270	0.270
1990	0.200	0.260	0.450	0.480	0.440	0.440	0.440	0.440	0.440	0.440	0.420	0.420
1989	0.200	0.260	0.450	0.510	0.450	0.450	0.450	0.450	0.450	0.450	0.440	0.440
1988	0.200	0.260	0.450	0.540	0.450	0.450	0.450	0.450	0.450	0.450	0.410	0.410
1987	0.200	0.260	0.260	0.690	0.690	0.690	0.690	0.623	0.623	0.431	0.431	0.431
1986	0.240	0.240	0.240	0.690	0.690	0.690	0.690	0.623	0.623	0.431	0.431	0.431
1985	0.270	0.320	0.320	0.690	0.690	0.690	0.690	0.623	0.623	0.431	0.431	0.431
1984	0.300	0.320	0.320	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1983	0.340	0.280	0.280	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1982	0.310	0.290	0.290	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1981	0.350	0.350	0.350	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1980	0.400	0.400	0.400	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1979	0.450	0.450	0.450	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1978	0.500	0.500	0.500	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1977	0.550	0.550	0.550	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1976	0.600	0.600	0.600	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751

Heavy Duty Conversion Factors

Year	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	0.8046	1.5509	1.5509	1.5509	1.7951	2.043	2.878	2.7879	3.241
1994	0.8121	1.5653	1.5653	1.5653	1.8025	2.051	2.889	2.81425	3.241
1993	0.8171	1.5748	1.5748	1.5748	1.8079	2.057	2.898	2.83958	3.241
1992	0.8228	1.5859	1.5859	1.5859	1.8118	2.062	2.904	2.86442	3.241
1991	0.8398	1.6186	1.6186	1.6186	1.821	2.072	2.919	2.89307	3.241
1990	0.8563	1.6505	1.6505	1.6505	1.825	2.077	2.926	2.91216	3.241
1989	0.8728	1.6823	1.6823	1.6823	1.8396	2.093	2.949	2.94551	3.241
1988	0.8895	1.7145	1.7145	1.7145	1.8534	2.109	2.971	2.97422	3.241
1987	0.9064	1.7471	1.7471	1.7471	1.8683	2.126	2.995	3.00992	3.241
1986	0.9087	1.7515	1.7515	1.7515	1.8816	2.141	3.016	3.04573	3.241
1985	0.9121	1.758	1.758	1.758	1.8937	2.155	3.036	3.07604	3.241
1984	0.9161	1.7656	1.7656	1.7656	1.9042	2.167	3.052	3.10326	3.241

1983	0.9195	1.7722	1.7722	1.7722	1.9164	2.181	3.072	3.12017	3.241
1982	0.923	1.779	1.779	1.779	1.927	2.193	3.089	3.152	3.241
1981	0.942	1.776	1.776	1.776	1.992	2.229	3.106	3.255	3.172
1980	0	1.773	1.773	1.773	2.06	2.254	3.062	3.332	3.069
1979	0	1.77	1.77	1.77	2.135	2.412	3.085	3.307	3.027
1978	0	1.768	1.768	1.768	2.216	2.616	3.126	3.361	3.025
1977	0	1.765	1.765	1.765	2.303	2.753	3.18	3.402	2.99
1976	0	1.762	1.762	1.762	2.342	2.76	3.156	3.353	2.935

Vehicle Emission Rates (g/mi - all vehicle classes, includes fuel sulfur adjustment)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	0.100	0.100	0.114	0.068	0.130	0.130	0.130	0.151	0.172	0.242	0.234	0.194
1994	0.137	0.197	0.114	0.068	0.131	0.131	0.131	0.151	0.172	0.243	0.236	0.194
1993	0.137	0.197	0.114	0.177	0.326	0.326	0.326	0.374	0.426	0.600	0.730	0.272
1992	0.137	0.197	0.114	0.179	0.328	0.328	0.328	0.375	0.427	0.601	0.736	0.752
1991	0.137	0.197	0.114	0.182	0.335	0.335	0.335	0.377	0.429	0.604	0.744	0.752
1990	0.137	0.197	0.387	0.368	0.644	0.644	0.644	0.712	0.810	1.141	1.077	1.199
1989	0.137	0.197	0.387	0.401	0.673	0.673	0.673	0.736	0.837	1.180	1.149	1.264
1988	0.137	0.197	0.387	0.436	0.686	0.686	0.686	0.741	0.844	1.188	1.071	1.167
1987	0.137	0.197	0.197	0.580	1.118	1.118	1.118	1.071	1.218	1.141	1.147	1.235
1986	0.177	0.177	0.177	0.582	1.121	1.121	1.121	1.078	1.227	1.149	1.160	1.235
1985	0.207	0.257	0.257	0.584	1.125	1.125	1.125	1.085	1.235	1.157	1.172	1.235
1984	0.237	0.257	0.257	0.568	1.095	1.095	1.095	1.243	1.415	2.140	2.175	2.272
1983	0.277	0.217	0.217	0.570	1.099	1.099	1.099	1.251	1.424	2.153	2.187	2.272
1982	0.247	0.227	0.227	0.572	1.103	1.103	1.103	1.258	1.432	2.165	2.210	2.272
1981	0.287	0.287	0.287	0.584	1.101	1.101	1.101	1.301	1.456	2.177	2.282	2.224
1980	0.337	0.337	0.337	0.000	1.099	1.099	1.099	1.345	1.472	2.146	2.336	2.151
1979	0.387	0.387	0.387	0.000	1.097	1.097	1.097	1.394	1.575	2.163	2.318	2.122
1978	0.437	0.437	0.437	0.000	1.096	1.096	1.096	1.447	1.708	2.191	2.356	2.121
1977	0.487	0.487	0.487	0.000	1.094	1.094	1.094	1.504	1.798	2.229	2.385	2.096
1976	0.537	0.537	0.537	0.000	1.092	1.092	1.092	1.529	1.802	2.212	2.350	2.057

Urban Diesel Particulate Emissions (billions of grams)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	0.016	0.002	0.022	0.139	0.011	0.009	0.002	0.114	0.256	0.147	0.551	0.050
1994	0.020	0.002	0.015	0.123	0.010	0.007	0.002	0.106	0.230	0.135	0.512	0.046

Year												
1993	0.015	0.000	0.011	0.280	0.023	0.015	0.004	0.241	0.505	0.307	1.425	0.060
1992	0.011	0.000	0.008	0.245	0.020	0.013	0.003	0.216	0.442	0.275	1.276	0.164
1991	0.010	0.000	0.006	0.209	0.016	0.011	0.003	0.192	0.382	0.242	1.133	0.160
1990	0.002	0.000	0.014	0.299	0.033	0.049	0.003	0.228	0.781	0.367	1.324	0.225
1989	0.003	0.000	0.022	0.321	0.000	0.042	0.016	0.195	0.673	0.393	1.456	0.195
1988	0.000	0.003	0.020	0.247	0.000	0.032	0.006	0.135	0.604	0.339	1.179	0.122
1987	0.005	0.009	0.010	0.269	0.000	0.004	0.002	0.090	0.652	0.235	0.932	0.158
1986	0.054	0.006	0.008	0.319	0.000	0.000	0.000	0.076	0.474	0.169	0.669	0.119
1985	0.074	0.012	0.013	0.317	0.008	0.000	0.000	0.095	0.390	0.164	0.654	0.094
1984	0.068	0.011	0.018	0.241	0.003	0.000	0.002	0.086	0.276	0.304	0.963	0.167
1983	0.054	0.010	0.027	0.107	0.000	0.000	0.000	0.052	0.170	0.015	0.623	0.151
1982	0.057	0.018	0.010	0.084	0.000	0.000	0.000	0.036	0.129	0.075	0.386	0.105
1981	0.061	0.026	0.007	0.000	0.000	0.000	0.000	0.037	0.106	0.095	0.399	0.127
1980	0.036	0.043	0.002	0.000	0.000	0.000	0.000	0.026	0.096	0.104	0.361	0.130
1979	0.015	0.014	0.000	0.000	0.000	0.000	0.000	0.025	0.063	0.118	0.421	0.064
1978	0.007	0.013	0.000	0.000	0.000	0.000	0.000	0.020	0.050	0.095	0.304	0.074
1977	0.002	0.001	0.000	0.000	0.000	0.000	0.000	0.005	0.027	0.051	0.239	0.040
1976	0.001	0.000	0.001	0.000	0.000	0.000	0.000	0.006	0.014	0.032	0.120	0.059
Total DP	0.511	0.170	0.216	3.199	0.125	0.183	0.043	1.979	6.318	3.663	14.927	2.310	33.644

Adjustment of Particulate Mass For Freeway Road Use

$$DPAdj = DP *[(1-FMF) * (4/3 - 1/3*FFR) + FMF * FFR]$$

FMF	0.25	0.25	0.25	0.25	0.22	0.22	0.22	0.39	0.45	0.39	0.73	0.1
FFR	0.69	0.69	0.69	0.69	0.63	0.63	0.63	0.63	0.63	0.63	0.58	0.58
DPAdj=	0.511	0.170	0.216	3.199	0.127	0.186	0.044	1.842	5.694	3.410	10.914	2.504

Grand total billions of grams of particulates in urban areas =   28.8180

Total Urban Fleet Vehicle Miles Travelled (billions of miles) =>   944.8052

National fleet average emission factor (g/mi)=   0.0305

03/27/1992

ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR

Vehicle Emission Rates (g/mi - light duty, g/BHP-hr - heavy duty)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1999	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1998	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1997	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1996	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1995	0.100	0.100	0.130	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1994	0.200	0.260	0.130	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1993	0.200	0.260	0.130	0.230	0.220	0.220	0.220	0.220	0.220	0.220	0.270	0.084
1992	0.200	0.260	0.130	0.230	0.220	0.220	0.220	0.220	0.220	0.220	0.270	0.270
1991	0.200	0.260	0.130	0.230	0.220	0.220	0.220	0.220	0.220	0.220	0.270	0.270
1990	0.200	0.260	0.450	0.480	0.440	0.440	0.440	0.440	0.440	0.440	0.420	0.420
1989	0.200	0.260	0.450	0.510	0.450	0.450	0.450	0.450	0.450	0.450	0.440	0.440
1988	0.200	0.260	0.450	0.540	0.450	0.450	0.450	0.450	0.450	0.450	0.410	0.410
1987	0.200	0.260	0.260	0.690	0.690	0.690	0.690	0.623	0.623	0.431	0.431	0.431
1986	0.240	0.240	0.240	0.690	0.690	0.690	0.690	0.623	0.623	0.431	0.431	0.431
1985	0.270	0.320	0.320	0.690	0.690	0.690	0.690	0.623	0.623	0.431	0.431	0.431
1984	0.300	0.320	0.320	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1983	0.340	0.280	0.280	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1982	0.310	0.290	0.290	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751
1981	0.350	0.350	0.350	0.670	0.670	0.670	0.670	0.703	0.703	0.751	0.751	0.751

Heavy Duty Conversion Factors

Year	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	0.785	1.513	1.513	1.513	1.763	2.006	2.825	2.644	3.241
1999	0.787	1.517	1.517	1.517	1.768	2.012	2.834	2.671	3.241
1998	0.790	1.522	1.522	1.522	1.776	2.021	2.847	2.700	3.241
1997	0.793	1.529	1.529	1.529	1.782	2.028	2.856	2.728	3.241
1996	0.799	1.540	1.540	1.540	1.789	2.035	2.867	2.759	3.241
1995	0.805	1.551	1.551	1.551	1.795	2.043	2.878	2.788	3.241
1994	0.812	1.565	1.565	1.565	1.803	2.051	2.889	2.814	3.241
1993	0.817	1.575	1.575	1.575	1.808	2.057	2.898	2.840	3.241
1992	0.823	1.586	1.586	1.586	1.812	2.062	2.904	2.864	3.241
1991	0.840	1.619	1.619	1.619	1.821	2.072	2.919	2.893	3.241
1990	0.856	1.650	1.650	1.650	1.825	2.077	2.926	2.912	3.241
1989	0.873	1.682	1.682	1.682	1.840	2.093	2.949	2.946	3.241

1988	0.890	1.714	1.714	1.714	1.853	2.109	2.971	2.974	3.241
1987	0.906	1.747	1.747	1.747	1.868	2.126	2.995	3.010	3.241
1986	0.909	1.752	1.752	1.752	1.882	2.141	3.016	3.046	3.241
1985	0.912	1.758	1.758	1.758	1.894	2.155	3.036	3.076	3.241
1984	0.916	1.766	1.766	1.766	1.904	2.167	3.052	3.103	3.241
1983	0.919	1.772	1.772	1.772	1.916	2.181	3.072	3.120	3.241
1982	0.923	1.779	1.779	1.779	1.927	2.193	3.089	3.152	3.241
1981	0.942	1.776	1.776	1.776	1.992	2.229	3.106	3.255	3.172

Vehicle Emission Rates (g/mi - all vehicle classes, includes fuel sulfur adjustment)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.148	0.168	0.237	0.222	0.194
1999	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.148	0.169	0.238	0.224	0.194
1998	0.100	0.100	0.120	0.066	0.128	0.128	0.128	0.149	0.170	0.239	0.227	0.194
1997	0.100	0.100	0.120	0.067	0.128	0.128	0.128	0.150	0.170	0.240	0.229	0.194
1996	0.100	0.100	0.120	0.067	0.129	0.129	0.129	0.150	0.171	0.241	0.232	0.194
1995	0.100	0.100	0.114	0.068	0.130	0.130	0.130	0.151	0.172	0.242	0.234	0.194
1994	0.137	0.197	0.114	0.068	0.131	0.131	0.131	0.151	0.172	0.243	0.236	0.194
1993	0.137	0.197	0.114	0.177	0.326	0.326	0.326	0.374	0.426	0.600	0.730	0.272
1992	0.137	0.197	0.114	0.179	0.328	0.328	0.328	0.375	0.427	0.601	0.736	0.833
1991	0.137	0.197	0.114	0.182	0.335	0.335	0.335	0.377	0.429	0.604	0.744	0.833
1990	0.137	0.197	0.387	0.368	0.644	0.644	0.644	0.712	0.810	1.141	1.077	1.199
1989	0.137	0.197	0.387	0.401	0.673	0.673	0.673	0.736	0.837	1.180	1.149	1.264
1988	0.137	0.197	0.387	0.436	0.686	0.686	0.686	0.741	0.844	1.188	1.071	1.167
1987	0.137	0.197	0.197	0.580	1.118	1.118	1.118	1.071	1.218	1.141	1.147	1.235
1986	0.177	0.177	0.177	0.582	1.121	1.121	1.121	1.078	1.227	1.149	1.160	1.235
1985	0.207	0.257	0.257	0.584	1.125	1.125	1.125	1.085	1.235	1.157	1.172	1.235
1984	0.237	0.257	0.257	0.568	1.095	1.095	1.095	1.243	1.415	2.140	2.175	2.272
1983	0.277	0.217	0.217	0.570	1.099	1.099	1.099	1.251	1.424	2.153	2.187	2.272
1982	0.247	0.227	0.227	0.572	1.103	1.103	1.103	1.258	1.432	2.165	2.210	2.272
1981	0.287	0.287	0.287	0.584	1.101	1.101	1.101	1.301	1.456	2.177	2.282	2.224

Urban Diesel Particulate Emissions (billions of grams)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	0.026	0.015	0.051	0.162	0.012	0.010	0.002	0.123	0.272	0.153	0.556	0.052
1999	0.023	0.011	0.042	0.146	0.010	0.009	0.002	0.113	0.247	0.141	0.515	0.048

Year												
1998	0.021	0.008	0.032	0.131	0.010	0.008	0.001	0.102	0.225	0.129	0.469	0.045
1997	0.019	0.005	0.024	0.114	0.008	0.006	0.001	0.090	0.201	0.114	0.425	0.044
1996	0.012	0.003	0.017	0.099	0.007	0.005	0.001	0.079	0.178	0.101	0.376	0.044
1995	0.010	0.001	0.011	0.085	0.006	0.004	0.001	0.069	0.153	0.088	0.327	0.040
1994	0.011	0.001	0.007	0.073	0.005	0.004	0.001	0.060	0.130	0.076	0.286	0.031
1993	0.008	0.000	0.006	0.162	0.011	0.007	0.002	0.127	0.266	0.162	0.739	0.039
1992	0.005	0.000	0.004	0.136	0.009	0.006	0.001	0.104	0.214	0.133	0.611	0.104
1991	0.004	0.000	0.003	0.111	0.007	0.005	0.001	0.086	0.171	0.108	0.503	0.096
1990	0.001	0.000	0.007	0.151	0.014	0.020	0.001	0.095	0.326	0.153	0.547	0.108
1989	0.001	0.000	0.010	0.153	0.000	0.017	0.005	0.076	0.263	0.154	0.563	0.100
1988	0.000	0.001	0.009	0.111	0.000	0.012	0.002	0.050	0.223	0.125	0.433	0.057
1987	0.001	0.004	0.005	0.114	0.000	0.002	0.001	0.032	0.233	0.084	0.328	0.081
1986	0.008	0.003	0.004	0.129	0.000	0.000	0.000	0.026	0.164	0.058	0.228	0.059
1985	0.010	0.005	0.006	0.122	0.003	0.000	0.000	0.032	0.131	0.055	0.218	0.055
1984	0.009	0.005	0.008	0.089	0.001	0.000	0.001	0.028	0.092	0.101	0.315	0.078
1983	0.007	0.004	0.012	0.038	0.000	0.000	0.000	0.017	0.055	0.005	0.200	0.085
1982	0.008	0.008	0.004	0.029	0.000	0.000	0.000	0.012	0.042	0.025	0.123	0.052
1981	0.009	0.011	0.003	0.000	0.000	0.000	0.000	0.012	0.035	0.033	0.127	0.054
Total	0.192	0.085	0.264	2.155	0.102	0.116	0.022	1.333	3.621	1.999	7.888	1.272

Total: 19.050

Adjustment of Particulate Mass For Freeway Road Use

$$DPAdj = DP * [(1-FMF) * (4/3 - 1/3*FFR) + FMF * FFR]$$

FMF	0.25	0.25	0.25	0.25	0.22	0.22	0.22	0.39	0.45	0.39	0.73	0.1
FFR	0.69	0.69	0.69	0.69	0.63	0.63	0.63	0.63	0.63	0.63	0.58	0.58
DPAdj=	0.192	0.085	0.264	2.155	0.104	0.117	0.023	1.241	3.264	1.861	5.768	1.379

Grand total billions of grams of particulates in urban areas =   16.4530

Total Urban Fleet Vehicle Miles Travelled (billions of miles) =>   1012

National fleet average emission factor (g/mi)=   0.0160

04/28/1992

ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR
ERR

Vehicle Emission Rates (g/mi - light duty, g/BHP-hr - heavy duty)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
2009	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
2008	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
2007	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
2006	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
2005	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
2004	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
2003	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
2002	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
2001	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
2000	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1999	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1998	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1997	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1996	0.100	0.100	0.120	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1995	0.100	0.100	0.130	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1994	0.200	0.260	0.130	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.084	0.060
1993	0.200	0.260	0.130	0.230	0.220	0.220	0.220	0.220	0.220	0.220	0.270	0.084
1992	0.200	0.260	0.130	0.230	0.220	0.220	0.220	0.220	0.220	0.220	0.270	0.270
1991	0.200	0.260	0.130	0.230	0.220	0.220	0.220	0.220	0.220	0.220	0.270	0.270

Heavy Duty Conversion Factors

Year	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	0.785	1.513	1.513	1.513	1.740	1.980	2.789	2.588	3.241
2009	0.785	1.513	1.513	1.513	1.742	1.982	2.792	2.590	3.241
2008	0.785	1.513	1.513	1.513	1.744	1.985	2.796	2.599	3.241
2007	0.785	1.513	1.513	1.513	1.747	1.988	2.800	2.603	3.241
2006	0.785	1.513	1.513	1.513	1.749	1.991	2.804	2.610	3.241
2005	0.785	1.513	1.513	1.513	1.752	1.994	2.809	2.616	3.241
2004	0.785	1.513	1.513	1.513	1.754	1.996	2.811	2.625	3.241
2003	0.785	1.513	1.513	1.513	1.756	1.998	2.814	2.630	3.241
2002	0.785	1.513	1.513	1.513	1.758	2.001	2.818	2.633	3.241
2001	0.785	1.513	1.513	1.513	1.761	2.004	2.823	2.643	3.241
2000	0.785	1.513	1.513	1.513	1.763	2.006	2.825	2.644	3.241
1999	0.787	1.517	1.517	1.517	1.768	2.012	2.834	2.671	3.241

1998	0.790	1.522	1.522	1.522	1.776	2.021	2.847	2.700	3.241
1997	0.793	1.529	1.529	1.529	1.782	2.028	2.856	2.728	3.241
1996	0.799	1.540	1.540	1.540	1.789	2.035	2.867	2.759	3.241
1995	0.805	1.551	1.551	1.551	1.795	2.043	2.878	2.788	3.241
1994	0.812	1.565	1.565	1.565	1.803	2.051	2.889	2.814	3.241
1993	0.817	1.575	1.575	1.575	1.808	2.057	2.898	2.840	3.241
1992	0.823	1.586	1.586	1.586	1.812	2.062	2.904	2.864	3.241
1991	0.840	1.619	1.619	1.619	1.821	2.072	2.919	2.893	3.241

Vehicle Emission Rates (g/mi - all vehicle classes, includes fuel sulfur adjustment)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.146	0.166	0.234	0.217	0.194
2009	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.146	0.166	0.235	0.218	0.194
2008	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.147	0.167	0.235	0.218	0.194
2007	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.147	0.167	0.235	0.219	0.194
2006	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.147	0.167	0.236	0.219	0.194
2005	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.147	0.167	0.236	0.220	0.194
2004	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.147	0.168	0.236	0.220	0.194
2003	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.147	0.168	0.236	0.221	0.194
2002	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.148	0.168	0.237	0.221	0.194
2001	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.148	0.168	0.237	0.222	0.194
2000	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.148	0.168	0.237	0.222	0.194
1999	0.100	0.100	0.120	0.066	0.127	0.127	0.127	0.148	0.169	0.238	0.224	0.194
1998	0.100	0.100	0.120	0.066	0.128	0.128	0.128	0.149	0.170	0.239	0.227	0.194
1997	0.100	0.100	0.120	0.067	0.128	0.128	0.128	0.150	0.170	0.240	0.229	0.194
1996	0.100	0.100	0.120	0.067	0.129	0.129	0.129	0.150	0.171	0.241	0.232	0.194
1995	0.100	0.100	0.114	0.068	0.130	0.130	0.130	0.151	0.172	0.242	0.234	0.194
1994	0.137	0.197	0.114	0.068	0.131	0.131	0.131	0.151	0.172	0.243	0.236	0.194
1993	0.137	0.197	0.114	0.177	0.326	0.326	0.326	0.374	0.426	0.600	0.730	0.272
1992	0.137	0.197	0.114	0.179	0.328	0.328	0.328	0.375	0.427	0.601	0.736	0.833
1991	0.137	0.197	0.114	0.182	0.335	0.335	0.335	0.377	0.429	0.604	0.744	0.833

Urban Diesel Particulate Emissions (billions of grams)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	0.04447	0.0452	0.087	0.2126	0.0135	0.0135	0.002	0.135	0.29874	0.1675	0.6049158	0.05631
2009	0.04038	0.0361	0.0723	0.1934	0.0118	0.0118	0.002	0.123	0.27218	0.15326	0.5517786	0.05284

Year													
2008	0.03658	0.0288	0.0578	0.1746	0.0102	0.0101	0.002	0.112	0.24743	0.13908	0.500769	0.04999	
2007	0.03289	0.0228	0.0495	0.1529	0.0088	0.0084	0.001	0.1	0.22213	0.1247	0.4480652	0.04973	
2006	0.02936	0.018	0.0412	0.1368	0.0076	0.0071	0.001	0.089	0.19675	0.11016	0.3956757	0.0482	
2005	0.02562	0.0155	0.0342	0.1191	0.0064	0.006	0.001	0.078	0.17176	0.09646	0.3454839	0.04283	
2004	0.02175	0.0121	0.0282	0.1024	0.0054	0.0051	0.001	0.067	0.14639	0.08314	0.2962889	0.03277	
2003	0.0176	0.0092	0.0232	0.0874	0.0045	0.0042	0.001	0.056	0.12337	0.06822	0.2481084	0.03134	
2002	0.01355	0.0069	0.0188	0.0732	0.0037	0.0034	0.001	0.046	0.10203	0.05695	0.2040008	0.02639	
2001	0.00992	0.0051	0.0152	0.0607	0.003	0.0028	0.000	0.038	0.08372	0.047	0.1681626	0.02518	
2000	0.00466	0.0037	0.0122	0.0498	0.0024	0.0022	0.000	0.031	0.06813	0.03835	0.1359968	0.02002	
1999	0.00317	0.0026	0.0099	0.0414	0.002	0.0018	0.000	0.025	0.05458	0.03121	0.1114964	0.01628	
1998	0.00231	0.0017	0.0074	0.034	0.0018	0.0014	0.000	0.02	0.04387	0.02506	0.0892853	0.01349	
1997	0.00153	0.0011	0.0053	0.027	0.0013	0.001	0.000	0.016	0.03478	0.01969	0.0715085	0.01275	
1996	0.00066	0.0006	0.0036	0.0212	0.001	0.0008	0.000	0.012	0.02739	0.01554	0.0566612	0.01196	
1995	0.00041	0.0002	0.0023	0.0165	0.0008	0.0006	0.000	0.01	0.02163	0.01235	0.0446922	0.01138	
1994	0.00037	0.0002	0.0015	0.0129	0.0006	0.0004	0.000	0.008	0.01676	0.0099	0.0361856	0.00732	
1993	0.00021	0.0000	0.0011	0.026	0.0012	0.0008	0.000	0.015	0.032	0.01951	0.0870445	0.01018	
1992	0.00011	0.0000	0.0008	0.02	0.0009	0.0006	0.000	0.012	0.02433	0.01521	0.0687815	0.0264	
1991	0.00008	0.0000	0.0006	0.0153	0.0007	0.0005	0.000	0.01	0.02009	0.01279	0.0532105	0.02031	
Total	0.286	0.210	0.472	1.577	0.088	0.083	0.013	1.002	2.208	1.246	4.518	0.566	12.268

Adjustment of Particulate Mass For Freeway Road Use

$$DPAdj = DP *[(1-FMF) * (4/3 - 1/3*FFR) + FMF * FFR]$$

FMF	0.25	0.25	0.25	0.25	0.22	0.22	0.22	0.39	0.45	0.39	0.73	0.1
FFR	0.69	0.69	0.69	0.69	0.63	0.63	0.63	0.63	0.63	0.63	0.58	0.58
DPAdj=	0.28563	0.21	0.472	1.5772	0.0889	0.0838	0.013	0.933	1.99018	1.16002	3.303643	0.61318

Grand total billions of grams of particulates in urban areas =   10.73051

Total Urban Fleet Vehicle Miles Travelled (billions of miles) =>   1160.5671

National fleet average emission factor (g/mi)=   0.0092459

04/28/1992

Urban Diesel Vehicle Miles Travelled (billions of miles)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses	Totals	%ages
1990	0.029	0.003	0.075	1.325	0.102	0.149	0.011	0.534	1.606	0.537	2.071	0.234	6.675	12.488
1989	0.038	0.002	0.115	1.335	0.000	0.126	0.052	0.469	1.423	0.590	2.271	0.232	6.654	12.448
1988	0.000	0.033	0.102	0.977	0.000	0.097	0.019	0.346	1.359	0.542	2.121	0.163	5.760	10.777
1987	0.083	0.087	0.105	0.832	0.000	0.008	0.004	0.173	1.104	0.425	1.696	0.224	4.743	8.873
1986	0.840	0.072	0.095	1.034	0.000	0.000	0.000	0.157	0.862	0.328	1.302	0.177	4.866	9.103
1985	1.219	0.095	0.109	1.081	0.018	0.000	0.000	0.210	0.756	0.341	1.353	0.158	5.339	9.989
1984	1.219	0.087	0.149	0.892	0.007	0.000	0.006	0.176	0.499	0.362	1.143	0.143	4.683	8.761
1983	0.926	0.098	0.274	0.417	0.000	0.000	0.001	0.112	0.322	0.019	0.776	0.142	3.085	5.772
1982	1.290	0.182	0.103	0.344	0.001	0.000	0.001	0.080	0.251	0.097	0.496	0.090	2.933	5.488
1981	1.389	0.204	0.059	0.000	0.000	0.000	0.000	0.082	0.209	0.127	0.512	0.116	2.697	5.046
1980	0.735	0.295	0.014	0.000	0.000	0.000	0.000	0.058	0.195	0.144	0.463	0.104	2.008	3.757
1979	0.281	0.085	0.000	0.000	0.000	0.000	0.000	0.055	0.123	0.164	0.557	0.065	1.330	2.488
1978	0.132	0.068	0.000	0.000	0.000	0.000	0.000	0.042	0.090	0.133	0.401	0.062	0.928	1.736
1977	0.029	0.004	0.002	0.000	0.000	0.000	0.000	0.010	0.047	0.071	0.312	0.038	0.512	0.958
1976	0.012	0.000	0.002	0.000	0.000	0.000	0.000	0.011	0.023	0.043	0.163	0.067	0.322	0.602
1975	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.003	0.020	0.040	0.085	0.069	0.218	0.408
1974	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.006	0.016	0.042	0.148	0.042	0.255	0.477
1973	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.006	0.018	0.044	0.113	0.029	0.209	0.392
1972	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.004	0.014	0.032	0.068	0.023	0.140	0.261
1971	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.002	0.011	0.018	0.047	0.015	0.093	0.174
Totals	8.222	1.315	1.203	8.237	0.128	0.381	0.093	2.535	8.951	4.099	16.097	2.193		

Urban Gasoline Vehicle Miles Travelled (billions of miles)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses	Totals	%ages
1990	71.972	21.958	6.466	3.523	0.090	0.108	0.032	0.086	0.114	0.000	0.000	0.000	104.350	12.594
1989	71.247	20.253	6.960	3.909	0.141	0.099	0.012	0.089	0.187	0.000	0.000	0.000	102.896	12.418
1988	72.121	19.128	6.515	3.940	0.095	0.062	0.040	0.170	0.217	0.002	0.000	0.000	102.291	12.345
1987	65.644	16.160	5.186	3.100	0.085	0.006	0.046	0.148	0.242	0.004	0.000	0.000	90.620	10.937
1986	67.098	13.943	4.839	2.627	0.063	0.000	0.032	0.134	0.252	0.003	0.000	0.000	88.992	10.740
1985	58.175	11.973	4.561	2.383	0.037	0.000	0.024	0.112	0.204	0.003	0.000	0.000	77.473	9.350
1984	47.957	9.095	3.823	2.058	0.019	0.000	0.015	0.121	0.152	0.002	0.000	0.000	63.241	7.633
1983	35.583	5.204	3.164	2.162	0.000	0.000	0.003	0.090	0.097	0.000	0.000	0.000	46.302	5.588
1982	24.224	3.741	2.291	1.445	0.003	0.000	0.002	0.072	0.097	0.001	0.000	0.000	31.876	3.847
1981	19.346	2.568	1.772	1.370	0.003	0.000	0.005	0.103	0.052	0.002	0.000	0.000	25.219	3.044
1980	15.159	2.320	1.775	1.340	0.010	0.000	0.004	0.112	0.048	0.002	0.000	0.000	20.769	2.507
1979	13.044	2.815	2.440	1.819	0.031	0.006	0.005	0.152	0.039	0.004	0.000	0.000	20.356	2.457
1978	9.727	2.504	2.757	2.059	0.126	0.010	0.004	0.132	0.022	0.004	0.000	0.000	17.347	2.094
1977	6.723	2.100	1.916	1.427	0.051	0.004	0.005	0.117	0.013	0.002	0.000	0.000	12.359	1.492
1976	4.170	1.763	1.218	0.908	0.050	0.000	0.008	0.087	0.011	0.002	0.000	0.000	8.215	0.991

Year														
1975	2.450	1.208	0.675	0.502	0.022	0.001	0.006	0.074	0.008	0.003	0.000	0.000	4.948	0.597
1974	1.733	1.322	0.399	0.297	0.016	0.002	0.007	0.076	0.011	0.003	0.000	0.000	3.865	0.467
1973	1.494	1.293	0.346	0.262	0.031	0.002	0.007	0.068	0.010	0.003	0.000	0.000	3.515	0.424
1972	1.016	0.895	0.223	0.166	0.027	0.005	0.010	0.042	0.007	0.002	0.000	0.000	2.394	0.289
1971	0.657	0.583	0.147	0.109	0.002	0.006	0.012	0.026	0.006	0.001	0.000	0.000	1.548	0.187
Totals	589.539	140.827	57.471	35.405	0.903	0.312	0.280	2.011	1.788	0.041	0.000	0.000		
Gas	589.539	140.827	57.471	35.405	0.903	0.312	0.280	2.011	1.788	0.041	0.000	0.000	828.576	
Fract.	0.712	0.170	0.069	0.043	0.001	0.000	0.000	0.002	0.002	0.000	0.000	0.000		
Diesel	8.222	1.315	1.203	8.237	0.128	0.381	0.093	2.535	8.951	4.099	16.097	2.193	53.452	
Fract.	0.154	0.025	0.022	0.154	0.002	0.007	0.002	0.047	0.167	0.077	0.301	0.041		
UVMT	597.761	142.141	58.673	43.642	1.031	0.693	0.373	4.546	10.738	4.140	16.097	2.193		

Total Urban Miles ---- =   882.028   billion

03/27/1991

Urban Diesel Vehicle Miles Travelled (billions of miles)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses	Totals	%ages
1990	0.158	0.023	0.191	2.060	0.088	0.066	0.014	0.759	1.490	0.610	2.355	0.259	8.075	14.445
1989	0.144	0.010	0.130	1.799	0.076	0.056	0.013	0.700	1.335	0.558	2.165	0.238	7.224	12.923
1988	0.110	0.002	0.098	1.580	0.070	0.047	0.011	0.644	1.186	0.511	1.953	0.220	6.431	11.504
1987	0.081	0.001	0.072	1.371	0.061	0.041	0.010	0.575	1.036	0.458	1.734	0.218	5.658	10.121
1986	0.072	0.001	0.052	1.145	0.047	0.033	0.009	0.510	0.890	0.400	1.524	0.213	4.896	8.758
1985	0.018	0.001	0.037	0.813	0.052	0.076	0.005	0.321	0.964	0.322	1.228	0.187	4.025	7.201
1984	0.022	0.001	0.058	0.799	0.000	0.063	0.024	0.265	0.804	0.333	1.267	0.154	3.789	6.779
1983	0.000	0.016	0.052	0.566	0.000	0.047	0.008	0.183	0.716	0.285	1.101	0.105	3.079	5.507
1982	0.037	0.044	0.052	0.463	0.000	0.004	0.002	0.084	0.535	0.206	0.813	0.128	2.367	4.234
1981	0.305	0.035	0.046	0.549	0.000	0.000	0.000	0.070	0.386	0.147	0.577	0.096	2.213	3.959
1980	0.357	0.046	0.052	0.543	0.007	0.000	0.000	0.088	0.316	0.142	0.558	0.076	2.185	3.909
1979	0.286	0.041	0.070	0.425	0.003	0.000	0.002	0.069	0.195	0.142	0.443	0.074	1.749	3.129
1978	0.196	0.045	0.125	0.187	0.000	0.000	0.000	0.041	0.119	0.007	0.285	0.066	1.072	1.917
1977	0.232	0.081	0.046	0.146	0.000	0.000	0.000	0.028	0.090	0.035	0.175	0.046	0.878	1.572
1976	0.212	0.089	0.026	0.000	0.000	0.000	0.000	0.028	0.072	0.044	0.175	0.057	0.703	1.258
1975	0.106	0.128	0.006	0.000	0.000	0.000	0.000	0.019	0.065	0.048	0.154	0.060	0.588	1.052
1974	0.039	0.036	0.000	0.000	0.000	0.000	0.000	0.018	0.040	0.054	0.182	0.030	0.399	0.714
1973	0.016	0.029	0.000	0.000	0.000	0.000	0.000	0.014	0.029	0.043	0.129	0.035	0.295	0.528
1972	0.004	0.002	0.001	0.000	0.000	0.000	0.000	0.003	0.015	0.023	0.100	0.019	0.166	0.297
1971	0.002	0.000	0.001	0.000	0.000	0.000	0.000	0.004	0.008	0.014	0.051	0.028	0.108	0.193
Totals	2.397	0.632	1.115	12.445	0.405	0.432	0.099	4.422	10.290	4.384	16.968	2.312		

Urban Gasoline Vehicle Miles Travelled (billions of miles)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses	Totals	%ages
1990	89.351	18.024	8.194	3.957	0.066	0.029	0.043	0.079	0.069	0.000	0.000	0.000	119.812	13.479
1989	83.214	16.437	7.387	3.611	0.060	0.027	0.039	0.080	0.068	0.000	0.000	0.000	110.923	12.479
1988	77.225	14.906	6.677	3.315	0.059	0.024	0.036	0.080	0.066	0.000	0.000	0.000	102.388	11.518
1987	71.039	13.305	5.980	3.009	0.056	0.023	0.034	0.078	0.063	0.000	0.000	0.000	93.586	10.528
1986	64.833	11.812	5.333	2.760	0.046	0.023	0.032	0.075	0.058	0.000	0.000	0.000	84.973	9.559
1985	49.830	14.372	3.970	2.158	0.055	0.066	0.019	0.052	0.068	0.000	0.000	0.000	70.591	7.941
1984	45.982	12.909	4.163	2.334	0.084	0.059	0.007	0.050	0.105	0.000	0.000	0.000	65.693	7.390
1983	41.775	11.812	3.773	2.277	0.055	0.036	0.021	0.089	0.113	0.001	0.000	0.000	59.952	6.744
1982	32.576	9.590	2.885	1.722	0.047	0.003	0.022	0.071	0.116	0.002	0.000	0.000	47.035	5.291
1981	27.374	7.891	2.570	1.392	0.034	0.000	0.014	0.060	0.112	0.001	0.000	0.000	39.448	4.438
1980	19.121	6.417	2.294	1.195	0.019	0.000	0.010	0.047	0.085	0.001	0.000	0.000	29.189	3.284
1979	12.656	4.608	1.817	0.978	0.009	0.000	0.006	0.047	0.059	0.001	0.000	0.000	20.181	2.270
1978	7.813	2.485	1.416	0.968	0.000	0.000	0.001	0.033	0.036	0.000	0.000	0.000	12.752	1.435
1977	4.574	1.687	0.973	0.613	0.001	0.000	0.001	0.025	0.034	0.000	0.000	0.000	7.910	0.890
1976	3.312	1.100	0.710	0.552	0.001	0.000	0.002	0.035	0.018	0.001	0.000	0.000	5.731	0.645
1975	2.457	0.954	0.686	0.515	0.004	0.000	0.001	0.037	0.016	0.001	0.000	0.000	4.671	0.525

1974	2.012	1.107	0.900	0.672	0.011	0.002	0.002	0.050	0.013	0.001	0.000	0.000	4.769	0.537
1973	1.457	0.946	0.988	0.731	0.045	0.004	0.001	0.043	0.007	0.001	0.000	0.000	4.224	0.475
1972	1.021	0.776	0.666	0.497	0.018	0.002	0.002	0.038	0.004	0.001	0.000	0.000	3.023	0.340
1971	0.639	0.640	0.414	0.308	0.017	0.000	0.002	0.028	0.003	0.001	0.000	0.000	2.052	0.231
Totals	638.260	151.780	61.798	33.563	0.688	0.299	0.295	1.097	1.114	0.012	0.000	0.000		
Gas	638.260	151.780	61.798	33.563	0.688	0.299	0.295	1.097	1.114	0.012	0.000	0.000	888.905	
Fract.	0.718	0.171	0.070	0.038	0.001	0.000	0.000	0.001	0.001	0.000	0.000	0.000		
Diesel	2.397	0.632	1.115	12.445	0.405	0.432	0.099	4.422	10.290	4.384	16.968	2.312	55.900	
Fract.	0.043	0.011	0.020	0.223	0.007	0.008	0.002	0.079	0.184	0.078	0.304	0.041		
UVMT	640.657	152.411	62.913	46.009	1.092	0.731	0.394	5.519	11.405	4.395	16.968	2.312		

Total Urban Miles ---- =   944.805 billion

03/27/1991

Urban Diesel Vehicle Miles Travelled (billions of miles)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses	Totals	%ages
2000	0.257	0.154	0.424	2.450	0.091	0.082	0.015	0.832	1.614	0.645	2.504	0.266	9.334	14.707
1999	0.233	0.110	0.351	2.201	0.080	0.070	0.013	0.760	1.464	0.594	2.295	0.245	8.417	13.261
1998	0.212	0.075	0.268	1.971	0.078	0.060	0.011	0.681	1.325	0.538	2.069	0.231	7.520	11.848
1997	0.191	0.048	0.197	1.714	0.063	0.050	0.010	0.603	1.182	0.477	1.853	0.224	6.610	10.415
1996	0.120	0.027	0.140	1.479	0.055	0.041	0.008	0.527	1.040	0.418	1.622	0.228	5.707	8.991
1995	0.097	0.012	0.096	1.263	0.045	0.034	0.007	0.455	0.894	0.366	1.397	0.208	4.874	7.679
1994	0.083	0.005	0.065	1.075	0.038	0.028	0.006	0.395	0.754	0.315	1.209	0.158	4.132	6.510
1993	0.057	0.001	0.049	0.915	0.034	0.023	0.005	0.339	0.625	0.269	1.012	0.142	3.471	5.469
1992	0.036	0.001	0.036	0.763	0.028	0.019	0.004	0.278	0.501	0.222	0.830	0.125	2.843	4.479
1991	0.026	0.001	0.025	0.608	0.021	0.014	0.003	0.229	0.399	0.179	0.676	0.116	2.298	3.620
1990	0.005	0.001	0.018	0.409	0.022	0.032	0.002	0.133	0.402	0.134	0.508	0.090	1.757	2.768
1989	0.005	0.001	0.027	0.380	0.000	0.025	0.008	0.103	0.314	0.131	0.490	0.079	1.563	2.463
1988	0.000	0.007	0.024	0.254	0.000	0.017	0.003	0.067	0.265	0.106	0.404	0.049	1.196	1.885
1987	0.007	0.019	0.023	0.196	0.000	0.001	0.000	0.030	0.191	0.074	0.286	0.066	0.894	1.409
1986	0.047	0.015	0.020	0.221	0.000	0.000	0.000	0.024	0.133	0.051	0.197	0.048	0.756	1.192
1985	0.051	0.020	0.023	0.209	0.002	0.000	0.000	0.030	0.106	0.048	0.186	0.044	0.719	1.132
1984	0.039	0.018	0.030	0.157	0.001	0.000	0.001	0.023	0.065	0.047	0.145	0.034	0.559	0.881
1983	0.025	0.019	0.054	0.067	0.000	0.000	0.000	0.013	0.038	0.002	0.091	0.038	0.347	0.547
1982	0.031	0.034	0.020	0.050	0.000	0.000	0.000	0.009	0.029	0.011	0.055	0.023	0.263	0.415
1981	0.030	0.039	0.011	0.000	0.000	0.000	0.000	0.010	0.024	0.015	0.055	0.024	0.209	0.329
Totals	1.551	0.607	1.901	16.386	0.556	0.496	0.096	5.543	11.366	4.641	17.886	2.438		

Urban Gasoline Vehicle Miles Travelled (billions of miles)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses	Totals	%ages
2000	94.360	19.836	8.460	3.801	0.068	0.029	0.045	0.050	0.067	0.000	0.000	0.000	126.717	13.359
1999	87.650	18.197	7.778	3.560	0.063	0.027	0.041	0.052	0.061	0.000	0.000	0.000	117.427	12.380
1998	81.350	16.516	7.159	3.324	0.063	0.025	0.037	0.052	0.055	0.000	0.000	0.000	108.580	11.447
1997	75.118	14.892	6.414	3.014	0.053	0.023	0.033	0.051	0.049	0.000	0.000	0.000	99.647	10.505
1996	68.936	13.343	5.702	2.716	0.048	0.020	0.030	0.050	0.043	0.000	0.000	0.000	90.887	9.582
1995	61.812	11.790	5.031	2.422	0.041	0.018	0.026	0.047	0.041	0.000	0.000	0.000	81.228	8.563
1994	53.787	10.473	4.414	2.154	0.036	0.016	0.022	0.045	0.038	0.000	0.000	0.000	70.985	7.484
1993	44.743	9.202	3.870	1.917	0.034	0.014	0.019	0.042	0.035	0.000	0.000	0.000	59.875	6.312
1992	35.282	7.894	3.330	1.672	0.031	0.013	0.016	0.038	0.030	0.000	0.000	0.000	48.306	5.093
1991	26.428	6.685	2.835	1.463	0.025	0.012	0.014	0.034	0.026	0.000	0.000	0.000	37.521	3.956
1990	16.417	7.698	1.999	1.084	0.028	0.033	0.008	0.021	0.028	0.000	0.000	0.000	27.317	2.880
1989	12.157	6.538	1.976	1.109	0.040	0.028	0.003	0.019	0.041	0.000	0.000	0.000	21.911	2.310
1988	9.139	5.648	1.690	1.021	0.025	0.016	0.008	0.033	0.042	0.000	0.000	0.000	17.621	1.858
1987	6.177	4.329	1.225	0.730	0.020	0.001	0.008	0.025	0.041	0.001	0.000	0.000	12.557	1.324
1986	4.694	3.384	1.032	0.560	0.013	0.000	0.005	0.020	0.039	0.000	0.000	0.000	9.748	1.028

1985	3.041	2.628	0.881	0.460	0.007	0.000	0.003	0.016	0.028	0.000	0.000	0.000	7.066	0.745
1984	1.953	1.813	0.672	0.362	0.003	0.000	0.002	0.015	0.019	0.000	0.000	0.000	4.840	0.510
1983	1.212	0.945	0.506	0.345	0.000	0.000	0.000	0.011	0.012	0.000	0.000	0.000	3.031	0.320
1982	0.725	0.619	0.338	0.211	0.000	0.000	0.000	0.008	0.011	0.000	0.000	0.000	1.914	0.202
1981	0.520	0.402	0.245	0.189	0.000	0.000	0.001	0.011	0.006	0.000	0.000	0.000	1.374	0.145
Totals	685.501	162.832	65.557	32.113	0.598	0.276	0.319	0.641	0.712	0.003	0.000	0.000		
Gas	685.501	162.832	65.557	32.113	0.598	0.276	0.319	0.641	0.712	0.003	0.000	0.000	948.552	
Fract.	0.723	0.172	0.069	0.034	0.001	0.000	0.000	0.001	0.001	0.000	0.000	0.000		
Diesel	1.551	0.607	1.901	16.386	0.556	0.496	0.096	5.543	11.366	4.641	17.886	2.438	63.467	
Fract.	0.024	0.010	0.030	0.258	0.009	0.008	0.002	0.087	0.179	0.073	0.282	0.038		
UVMT	687.052	163.439	67.458	48.499	1.154	0.772	0.415	6.184	12.077	4.644	17.886	2.438		

Total Urban Miles ---- =   1012   billion

04/28/1991

Urban Diesel Vehicle Miles Travelled (billions of miles)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses	Totals	%ages
2010	0.445	0.452	0.725	3.224	0.106	0.106	0.017	0.926	1.796	0.715	2.783	0.290	11.584	14.314
2009	0.404	0.361	0.602	2.934	0.093	0.093	0.015	0.842	1.635	0.654	2.537	0.272	10.440	12.900
2008	0.366	0.288	0.482	2.648	0.080	0.080	0.013	0.765	1.484	0.592	2.294	0.257	9.349	11.552
2007	0.329	0.228	0.413	2.319	0.069	0.066	0.011	0.683	1.330	0.530	2.050	0.256	8.284	10.236
2006	0.294	0.180	0.343	2.076	0.059	0.056	0.009	0.604	1.177	0.468	1.805	0.248	7.319	9.043
2005	0.256	0.155	0.285	1.806	0.051	0.048	0.008	0.527	1.026	0.409	1.572	0.220	6.363	7.862
2004	0.218	0.121	0.235	1.554	0.043	0.040	0.006	0.452	0.873	0.352	1.344	0.169	5.406	6.679
2003	0.176	0.092	0.193	1.325	0.035	0.033	0.005	0.379	0.735	0.289	1.123	0.161	4.547	5.619
2002	0.136	0.069	0.157	1.110	0.029	0.027	0.004	0.312	0.607	0.241	0.922	0.136	3.749	4.633
2001	0.099	0.051	0.126	0.920	0.024	0.022	0.003	0.257	0.497	0.198	0.757	0.129	3.086	3.813
2000	0.047	0.037	0.102	0.756	0.019	0.017	0.003	0.209	0.404	0.162	0.612	0.103	2.471	3.053
1999	0.032	0.026	0.083	0.625	0.016	0.014	0.002	0.168	0.323	0.131	0.497	0.084	2.000	2.472
1998	0.023	0.017	0.062	0.513	0.014	0.011	0.002	0.133	0.258	0.105	0.394	0.069	1.600	1.977
1997	0.015	0.011	0.044	0.405	0.010	0.008	0.001	0.104	0.204	0.082	0.312	0.066	1.262	1.560
1996	0.007	0.006	0.030	0.316	0.008	0.006	0.001	0.081	0.160	0.065	0.245	0.061	0.986	1.218
1995	0.004	0.002	0.020	0.244	0.006	0.004	0.001	0.064	0.126	0.051	0.191	0.059	0.772	0.955
1994	0.003	0.001	0.013	0.189	0.005	0.003	0.001	0.051	0.097	0.041	0.153	0.038	0.594	0.734
1993	0.002	0.000	0.010	0.147	0.004	0.002	0.000	0.041	0.075	0.033	0.119	0.037	0.470	0.580
1992	0.001	0.000	0.007	0.112	0.003	0.002	0.000	0.032	0.057	0.025	0.093	0.032	0.364	0.450
1991	0.001	0.000	0.005	0.084	0.002	0.001	0.000	0.027	0.047	0.021	0.072	0.024	0.284	0.351
Totals	2.854	2.099	3.936	23.308	0.675	0.640	0.101	6.656	12.912	5.162	19.875	2.710		

Urban Gasoline Vehicle Miles Travelled (billions of miles)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses	Totals	%ages
2010	108.755	23.044	9.585	3.761	0.080	0.027	0.050	0.056	0.075	0.000	0.000	0.000	145.432	13.470
2009	101.052	21.105	8.707	3.421	0.073	0.024	0.045	0.051	0.068	0.000	0.000	0.000	134.546	12.462
2008	93.663	19.298	7.865	3.086	0.065	0.022	0.041	0.046	0.061	0.000	0.000	0.000	124.148	11.499
2007	86.193	17.497	7.019	2.812	0.058	0.019	0.037	0.041	0.055	0.000	0.000	0.000	113.732	10.534
2006	78.722	15.816	6.274	2.516	0.052	0.017	0.033	0.036	0.049	0.000	0.000	0.000	103.516	9.588
2005	70.305	14.187	5.580	2.279	0.046	0.016	0.028	0.032	0.042	0.000	0.000	0.000	92.515	8.569
2004	61.020	12.586	4.908	2.041	0.040	0.015	0.024	0.027	0.036	0.000	0.000	0.000	80.697	7.474
2003	50.552	10.960	4.275	1.812	0.035	0.013	0.020	0.023	0.030	0.000	0.000	0.000	67.720	6.273
2002	39.846	9.536	3.660	1.580	0.030	0.012	0.017	0.019	0.025	0.000	0.000	0.000	54.724	5.069
2001	29.848	8.182	3.101	1.365	0.025	0.010	0.014	0.015	0.020	0.000	0.000	0.000	42.580	3.944
2000	21.525	6.955	2.613	1.168	0.021	0.009	0.011	0.013	0.017	0.000	0.000	0.000	32.332	2.995
1999	14.981	5.876	2.213	1.008	0.018	0.008	0.009	0.011	0.013	0.000	0.000	0.000	24.137	2.236
1998	10.328	4.870	1.862	0.862	0.016	0.007	0.007	0.010	0.011	0.000	0.000	0.000	17.972	1.665
1997	7.096	3.993	1.515	0.710	0.012	0.005	0.006	0.009	0.008	0.000	0.000	0.000	13.355	1.237
1996	4.813	3.246	1.219	0.578	0.010	0.004	0.004	0.008	0.007	0.000	0.000	0.000	9.890	0.916

1995	3.236	2.592	0.973	0.467	0.008	0.003	0.004	0.007	0.006	0.000	0.000	0.000	7.294	0.676
1994	2.210	2.085	0.778	0.377	0.006	0.003	0.003	0.006	0.005	0.000	0.000	0.000	5.473	0.507
1993	1.500	1.673	0.619	0.306	0.005	0.002	0.002	0.005	0.004	0.000	0.000	0.000	4.116	0.381
1992	1.026	1.316	0.490	0.244	0.005	0.002	0.002	0.004	0.003	0.000	0.000	0.000	3.092	0.286
1991	0.711	1.052	0.391	0.202	0.003	0.002	0.002	0.004	0.003	0.000	0.000	0.000	2.369	0.219
Totals	787.381	185.867	73.645	30.595	0.609	0.219	0.360	0.422	0.538	0.001	0.000	0.000		
Gas	787.381	185.867	73.645	30.595	0.609	0.219	0.360	0.422	0.538	0.001	0.000	0.000	1079.64	
Fract.	0.729	0.172	0.068	0.028	0.001	0.000	0.000	0.000	0.000	0.000	0.000	0.000		
Diesel	2.854	2.099	3.936	23.308	0.675	0.640	0.101	6.656	12.912	5.162	19.875	2.710	80.930	
Fract.	0.035	0.026	0.049	0.288	0.008	0.008	0.001	0.082	0.160	0.064	0.246	0.033		
UVMT	790.235	187.966	77.581	53.903	1.284	0.859	0.461	7.078	13.450	5.163	19.875	2.710		

Total Urban Miles ---- =   1161   billion

04/28/1991

Vehicle Sales (Thousands)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	9301.00	2866.00	756.91	340.06	20.87	27.45	5.06	38.21	85.35	29.12	92.21	3.99
1989	9772.00	2854.00	895.21	402.19	19.20	27.00	7.20	39.10	93.40	34.82	110.28	4.19
1988	10530.00	2926.00	919.84	413.26	14.20	21.20	8.30	53.60	103.00	35.62	112.78	3.02
1987	10277.00	2696.00	810.82	364.28	14.00	2.10	8.20	44.30	102.60	31.49	99.71	4.22
1986	11460.00	2541.00	837.38	376.22	11.60	0.00	5.90	44.80	100.70	27.10	85.80	3.38
1985	11042.00	2408.00	883.34	396.86	10.90	0.00	5.10	48.40	97.00	32.06	101.54	3.37
1984	10391.00	2031.00	844.56	379.44	6.00	0.00	5.00	56.00	78.00	38.64	99.36	3.89
1983	9182.00	1314.00	832.83	374.17	0.00	0.00	1.00	47.00	59.00	2.46	79.54	4.08
1982	7982.00	1102.00	663.09	297.91	1.00	0.00	1.00	44.00	62.00	15.20	60.80	2.96
1981	8536.00	896.00	586.50	263.50	1.00	0.00	2.00	72.00	51.00	24.00	76.00	4.06
1980	8979.00	985.00	672.75	302.25	4.00	0.00	2.00	90.00	58.00	32.76	84.24	4.57
1979	10673.00	1271.00	1086.06	487.94	15.00	3.00	3.00	146.00	50.00	48.72	125.28	3.44
1978	11314.00	1334.00	1476.60	663.40	73.00	6.00	3.00	156.00	41.00	50.22	111.78	3.81
1977	11183.00	1306.00	1244.07	558.93	36.00	3.00	5.00	163.00	28.00	32.43	108.57	2.44
1976	10110.00	1318.00	966.69	434.31	43.00	0.00	9.00	153.00	22.00	26.19	70.81	4.75
1975	8624.00	1101.00	656.88	295.12	23.00	1.00	9.00	159.00	23.00	36.52	46.48	5.26
1974	8853.00	1467.00	480.24	215.76	21.00	3.00	14.00	207.00	31.00	45.88	102.12	4.82
1973	11424.00	1754.00	523.02	234.98	50.00	3.00	16.00	236.00	37.00	57.35	97.65	3.20
1972	10940.00	1498.00	413.31	185.69	55.00	11.00	29.00	182.00	35.00	52.92	73.08	2.90
1971	10242.00	1185.00	336.72	151.28	6.00	15.00	46.00	140.00	34.00	34.51	64.09	2.51

Annual Vehicle Miles Travelled for Gas and Diesel Vehicles (Billions)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	122.024	44.826	13.347	6.135	0.432	0.576	0.098	1.286	3.399	1.279	7.964	0.234
1989	120.782	41.349	14.434	6.639	0.320	0.506	0.143	1.172	3.197	1.405	8.733	0.232
1988	122.231	39.114	13.499	6.223	0.215	0.358	0.134	1.203	3.137	1.301	8.157	0.163
1987	111.397	33.145	10.796	4.977	0.193	0.031	0.115	0.805	2.692	1.030	6.525	0.224
1986	115.103	28.592	10.071	4.634	0.144	0.000	0.073	0.727	2.238	0.793	5.010	0.177
1985	100.628	24.632	9.530	4.383	0.124	0.000	0.055	0.758	1.926	0.825	5.204	0.158
1984	83.327	18.745	8.104	3.735	0.060	0.000	0.047	0.721	1.307	0.870	4.392	0.144
1983	61.917	10.822	7.011	3.262	0.000	0.000	0.008	0.499	0.844	0.046	2.985	0.142
1982	43.209	7.995	4.890	2.264	0.008	0.000	0.007	0.382	0.704	0.238	1.905	0.090

Year												
1981	35.121	5.653	3.733	1.736	0.007	0.000	0.011	0.488	0.524	0.311	1.967	0.116
1980	26.912	5.324	3.655	1.694	0.022	0.000	0.009	0.470	0.488	0.350	1.782	0.103
1979	22.585	5.926	4.976	2.303	0.071	0.014	0.011	0.595	0.325	0.409	2.144	0.065
1978	16.689	5.245	5.630	2.604	0.287	0.024	0.009	0.506	0.225	0.336	1.540	0.062
1977	11.396	4.301	3.910	1.808	0.116	0.010	0.012	0.396	0.121	0.176	1.199	0.038
1976	7.064	3.598	2.486	1.149	0.114	0.000	0.017	0.302	0.069	0.110	0.625	0.067
1975	4.128	2.476	1.374	0.635	0.050	0.002	0.014	0.243	0.059	0.111	0.327	0.069
1974	2.911	2.701	0.812	0.375	0.037	0.005	0.017	0.255	0.057	0.114	0.570	0.042
1973	2.563	2.634	0.712	0.329	0.070	0.004	0.015	0.231	0.056	0.117	0.432	0.029
1972	1.688	1.830	0.452	0.209	0.062	0.012	0.022	0.142	0.042	0.086	0.257	0.023
1971	1.075	1.177	0.296	0.136	0.005	0.014	0.028	0.087	0.034	0.047	0.178	0.015
Total	1012.749	290.084	119.718	55.231	2.336	1.555	0.844	11.269	21.441	9.955	61.897	2.193

Survival Rates (%)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
1989	99.6	99.3	99.3	99.3	99.3	99.3	99.7	99.7	99.7	99.7	99.7	99.9
1988	98.9	98.2	98.2	98.2	98.2	98.2	99.0	99.0	99.0	99.0	99.0	99.8
1987	97.6	96.8	96.8	96.8	96.8	96.8	97.4	97.4	97.4	97.4	97.4	99.5
1986	95.4	95.0	95.0	95.0	95.0	95.0	94.5	94.5	94.5	94.5	94.5	99.1
1985	91.4	92.6	92.6	92.6	92.6	92.6	90.2	90.2	90.2	90.2	90.2	98.5
1984	85.0	89.6	89.6	89.6	89.6	89.6	84.6	84.6	84.6	84.6	84.6	97.5
1983	75.6	85.8	85.8	85.8	85.8	85.8	78.1	78.1	78.1	78.1	78.1	95.5
1982	64.0	81.3	81.3	81.3	81.3	81.3	70.9	70.9	70.9	70.9	70.9	93.5
1981	51.4	76.1	76.1	76.1	76.1	76.1	63.7	63.7	63.7	63.7	63.7	90.5
1980	39.7	70.3	70.3	70.3	70.3	70.3	56.6	56.6	56.6	56.6	56.6	87.0
1979	29.7	64.3	64.3	64.3	64.3	64.3	49.8	49.8	49.8	49.8	49.8	83.0
1978	21.9	58.1	58.1	58.1	58.1	58.1	43.6	43.6	43.6	43.6	43.6	79.0
1977	16.0	52.0	52.0	52.0	52.0	52.0	38.0	38.0	38.0	38.0	38.0	74.0
1976	11.6	46.2	46.2	46.2	46.2	46.2	33.0	33.0	33.0	33.0	33.0	69.0
1975	8.4	40.8	40.8	40.8	40.8	40.8	28.6	28.6	28.6	28.6	28.6	64.0
1974	6.1	35.8	35.8	35.8	35.8	35.8	24.7	24.7	24.7	24.7	24.7	59.0
1973	4.4	31.3	31.3	31.3	31.3	31.3	21.3	21.3	21.3	21.3	21.3	54.0
1972	3.2	27.3	27.3	27.3	27.3	27.3	18.4	18.4	18.4	18.4	18.4	50.0
1971	2.3	23.8	23.8	23.8	23.8	23.8	15.8	15.8	15.8	15.8	15.8	46.0

VMT Fractions

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1990	0.121	0.155	0.112	0.111	0.185	0.370	0.116	0.114	0.159	0.129	0.129	0.107
1989	0.119	0.143	0.121	0.120	0.137	0.325	0.170	0.104	0.149	0.141	0.141	0.106
1988	0.121	0.135	0.113	0.113	0.092	0.230	0.159	0.107	0.146	0.131	0.132	0.074
1987	0.110	0.114	0.090	0.090	0.083	0.020	0.136	0.071	0.126	0.104	0.105	0.102
1986	0.114	0.099	0.084	0.084	0.062	0.000	0.086	0.065	0.104	0.080	0.081	0.081
1985	0.099	0.085	0.080	0.079	0.053	0.000	0.066	0.067	0.090	0.083	0.084	0.072
1984	0.082	0.065	0.068	0.068	0.026	0.000	0.056	0.064	0.061	0.087	0.071	0.065
1983	0.061	0.037	0.059	0.059	0.000	0.000	0.010	0.044	0.039	0.005	0.048	0.065
1982	0.043	0.028	0.041	0.041	0.003	0.000	0.008	0.034	0.033	0.024	0.031	0.041
1981	0.035	0.020	0.031	0.031	0.003	0.000	0.013	0.043	0.024	0.031	0.032	0.053
1980	0.027	0.018	0.031	0.031	0.010	0.000	0.011	0.042	0.023	0.035	0.029	0.047
1979	0.022	0.020	0.042	0.042	0.030	0.009	0.013	0.053	0.015	0.041	0.035	0.030
1978	0.017	0.018	0.047	0.047	0.123	0.015	0.011	0.045	0.011	0.034	0.025	0.028
1977	0.011	0.015	0.033	0.033	0.050	0.006	0.014	0.035	0.006	0.018	0.019	0.017
1976	0.007	0.012	0.021	0.021	0.049	0.000	0.020	0.027	0.003	0.011	0.010	0.031
1975	0.004	0.009	0.012	0.012	0.021	0.001	0.016	0.022	0.003	0.011	0.005	0.032
1974	0.003	0.009	0.007	0.007	0.016	0.003	0.020	0.023	0.003	0.012	0.009	0.019
1973	0.003	0.009	0.006	0.006	0.030	0.003	0.018	0.021	0.003	0.012	0.007	0.013
1972	0.002	0.006	0.004	0.004	0.027	0.008	0.026	0.013	0.002	0.009	0.004	0.010
1971	0.001	0.004	0.003	0.003	0.002	0.009	0.033	0.008	0.002	0.005	0.003	0.007

Urban Fractions

	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
Diesel	0.59	0.49	0.49	0.79	0.45	0.45	0.45	0.53	0.51	0.42	0.26	1
Gas	0.59	0.49	0.49	0.79	0.44	0.44	0.44	0.31	0.46	0.21	0	0

03/26/1992

Vehicle Sales (Thousands)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	11560.34	2355.09	969.06	422.99	16.59	9.93	6.67	46.57	74.74	33.08	104.90	4.43
1994	11431.79	2316.02	951.04	415.88	16.34	9.68	6.68	48.13	73.65	32.95	105.13	4.30
1993	11290.29	2276.24	942.23	412.92	17.36	9.40	6.63	49.80	72.32	32.97	103.83	4.07
1992	11123.27	2210.50	927.15	406.99	17.69	9.46	6.90	50.56	70.46	32.59	101.90	4.10
1991	10973.80	2143.52	913.44	401.37	16.01	9.53	7.35	51.73	68.50	31.81	100.37	4.06
1990	9301.00	2866.00	756.91	340.06	20.87	27.45	5.06	38.21	85.35	29.12	92.21	3.99
1989	9772.00	2854.00	895.21	402.19	19.20	27.00	7.20	39.10	93.40	34.82	110.28	4.19
1988	10530.00	2926.00	919.84	413.26	14.20	21.20	8.30	53.60	103.00	35.62	112.78	3.02
1987	10277.00	2696.00	810.82	364.28	14.00	2.10	8.20	44.30	102.60	31.49	99.71	4.22
1986	11460.00	2541.00	837.38	376.22	11.60	0.00	5.90	44.80	100.70	27.10	85.80	3.38
1985	11042.00	2408.00	883.34	396.86	10.90	0.00	5.10	48.40	97.00	32.06	101.54	3.37
1984	10391.00	2031.00	844.56	379.44	6.00	0.00	5.00	56.00	78.00	38.64	99.36	3.89
1983	9182.00	1314.00	832.83	374.17	0.00	0.00	1.00	47.00	59.00	2.46	79.54	4.08
1982	7982.00	1102.00	663.09	297.91	1.00	0.00	1.00	44.00	62.00	15.20	60.80	2.96
1981	8536.00	896.00	586.50	263.50	1.00	0.00	2.00	72.00	51.00	24.00	76.00	4.06
1980	8979.00	985.00	672.75	302.25	4.00	0.00	2.00	90.00	58.00	32.76	84.24	4.57
1979	10673.00	1271.00	1086.06	487.94	15.00	3.00	3.00	146.00	50.00	48.72	125.28	3.44
1978	11314.00	1334.00	1476.60	663.40	73.00	6.00	3.00	156.00	41.00	50.22	111.78	3.81
1977	11183.00	1306.00	1244.07	558.93	36.00	3.00	5.00	163.00	28.00	32.43	108.57	2.44
1976	10110.00	1318.00	966.69	434.31	43.00	0.00	9.00	153.00	22.00	26.19	70.81	4.75

Annual Vehicle Miles Travelled for Gas and Diesel Vehicles (Billions)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	151.719	36.844	17.112	7.614	0.347	0.215	0.129	1.687	3.073	1.453	9.061	0.259
1994	141.338	33.558	15.336	6.847	0.306	0.186	0.117	1.579	2.765	1.330	8.326	0.238
1993	131.099	30.422	13.827	6.193	0.289	0.160	0.105	1.473	2.469	1.218	7.509	0.220
1992	120.567	27.167	12.346	5.542	0.262	0.143	0.099	1.336	2.167	1.092	6.668	0.218
1991	109.980	24.117	10.991	4.942	0.210	0.126	0.093	1.204	1.872	0.953	5.860	0.213
1990	84.464	29.320	8.175	3.761	0.241	0.318	0.056	0.771	2.038	0.767	4.725	0.187
1989	77.993	26.354	8.610	3.965	0.191	0.273	0.070	0.660	1.803	0.793	4.875	0.154
1988	70.762	24.133	7.802	3.599	0.124	0.186	0.066	0.631	1.650	0.685	4.233	0.105
1987	55.316	19.647	5.998	2.767	0.107	0.016	0.055	0.388	1.302	0.499	3.124	0.128

1986	46.902	16.168	5.340	2.457	0.076	0.000	0.033	0.325	1.002	0.355	2.221	0.096
1985	33.027	13.198	4.791	2.203	0.059	0.000	0.023	0.316	0.803	0.344	2.148	0.076
1984	21.989	9.496	3.853	1.774	0.027	0.000	0.018	0.281	0.511	0.341	1.701	0.074
1983	13.573	5.172	3.151	1.462	0.000	0.000	0.003	0.184	0.312	0.017	1.096	0.066
1982	8.161	3.610	2.078	0.958	0.003	0.000	0.002	0.136	0.250	0.085	0.672	0.046
1981	5.959	2.428	1.506	0.697	0.003	0.000	0.004	0.168	0.180	0.107	0.670	0.057
1980	4.291	2.195	1.407	0.650	0.009	0.000	0.003	0.157	0.164	0.118	0.592	0.060
1979	3.505	2.336	1.836	0.848	0.026	0.005	0.004	0.195	0.107	0.135	0.700	0.030
1978	2.536	2.003	2.010	0.928	0.102	0.008	0.003	0.164	0.073	0.109	0.495	0.035
1977	1.725	1.596	1.361	0.628	0.041	0.003	0.004	0.127	0.039	0.057	0.382	0.019
1976	1.060	1.309	0.849	0.392	0.039	0.000	0.005	0.096	0.023	0.037	0.197	0.028
Total	1085.968	311.074	128.380	58.227	2.462	1.640	0.890	11.880	22.604	10.494	65.255	2.312

Survival Rates (%)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
1994	99.6	99.3	99.3	99.3	99.3	99.3	99.7	99.7	99.7	99.7	99.7	99.9
1993	98.9	98.2	98.2	98.2	98.2	98.2	99.0	99.0	99.0	99.0	99.0	99.8
1992	97.6	96.8	96.8	96.8	96.8	96.8	97.4	97.4	97.4	97.4	97.4	99.5
1991	95.4	95.0	95.0	95.0	95.0	95.0	94.5	94.5	94.5	94.5	94.5	99.1
1990	91.4	92.6	92.6	92.6	92.6	92.6	90.2	90.2	90.2	90.2	90.2	98.5
1989	85.0	89.6	89.6	89.6	89.6	89.6	84.6	84.6	84.6	84.6	84.6	97.5
1988	75.6	85.8	85.8	85.8	85.8	85.8	78.1	78.1	78.1	78.1	78.1	95.5
1987	64.0	81.3	81.3	81.3	81.3	81.3	70.9	70.9	70.9	70.9	70.9	93.5
1986	51.4	76.1	76.1	76.1	76.1	76.1	63.7	63.7	63.7	63.7	63.7	90.5
1985	39.7	70.3	70.3	70.3	70.3	70.3	56.6	56.6	56.6	56.6	56.6	87.0
1984	29.7	64.3	64.3	64.3	64.3	64.3	49.8	49.8	49.8	49.8	49.8	83.0
1983	21.9	58.1	58.1	58.1	58.1	58.1	43.6	43.6	43.6	43.6	43.6	79.0
1982	16.0	52.0	52.0	52.0	52.0	52.0	38.0	38.0	38.0	38.0	38.0	74.0
1981	11.6	46.2	46.2	46.2	46.2	46.2	33.0	33.0	33.0	33.0	33.0	69.0
1980	8.4	40.8	40.8	40.8	40.8	40.8	28.6	28.6	28.6	28.6	28.6	64.0
1979	6.1	35.8	35.8	35.8	35.8	35.8	24.7	24.7	24.7	24.7	24.7	59.0
1978	4.4	31.3	31.3	31.3	31.3	31.3	21.3	21.3	21.3	21.3	21.3	54.0
1977	3.2	27.3	27.3	27.3	27.3	27.3	18.4	18.4	18.4	18.4	18.4	50.0
1976	2.3	23.8	23.8	23.8	23.8	23.8	15.8	15.8	15.8	15.8	15.8	46.0

VMT Fractions

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
1995	0.140	0.118	0.133	0.131	0.141	0.131	0.145	0.142	0.136	0.138	0.139	0.112
1994	0.130	0.108	0.120	0.118	0.124	0.114	0.132	0.133	0.122	0.127	0.128	0.103
1993	0.121	0.098	0.108	0.106	0.118	0.098	0.119	0.124	0.109	0.116	0.115	0.095
1992	0.111	0.087	0.096	0.095	0.106	0.087	0.111	0.113	0.096	0.104	0.102	0.094
1991	0.101	0.078	0.086	0.085	0.085	0.077	0.105	0.101	0.083	0.091	0.090	0.092
1990	0.078	0.094	0.064	0.065	0.098	0.194	0.063	0.065	0.090	0.073	0.072	0.081
1989	0.072	0.085	0.067	0.068	0.078	0.167	0.078	0.056	0.080	0.076	0.075	0.067
1988	0.065	0.078	0.061	0.062	0.051	0.113	0.074	0.053	0.073	0.065	0.065	0.045
1987	0.051	0.063	0.047	0.048	0.044	0.010	0.061	0.033	0.058	0.048	0.048	0.056
1986	0.043	0.052	0.042	0.042	0.031	0.000	0.037	0.027	0.044	0.034	0.034	0.042
1985	0.030	0.042	0.037	0.038	0.024	0.000	0.026	0.027	0.036	0.033	0.033	0.033
1984	0.020	0.031	0.030	0.031	0.011	0.000	0.020	0.024	0.023	0.033	0.026	0.032
1983	0.013	0.017	0.025	0.025	0.000	0.000	0.003	0.016	0.014	0.002	0.017	0.029
1982	0.008	0.012	0.016	0.017	0.001	0.000	0.003	0.011	0.011	0.008	0.010	0.020
1981	0.006	0.008	0.012	0.012	0.001	0.000	0.004	0.014	0.008	0.010	0.010	0.025
1980	0.004	0.007	0.011	0.011	0.004	0.000	0.003	0.013	0.007	0.011	0.009	0.026
1979	0.003	0.008	0.014	0.015	0.011	0.003	0.004	0.017	0.005	0.013	0.011	0.013
1978	0.002	0.006	0.016	0.016	0.042	0.005	0.003	0.014	0.003	0.010	0.008	0.015
1977	0.002	0.005	0.011	0.011	0.016	0.002	0.004	0.011	0.002	0.005	0.006	0.008
1976	0.001	0.004	0.007	0.007	0.016	0.000	0.006	0.008	0.001	0.004	0.003	0.012

Urban Fractions

	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
Diesel	0.59	0.49	0.49	0.79	0.45	0.45	0.45	0.53	0.51	0.42	0.26	1
Gas	0.59	0.49	0.49	0.79	0.44	0.44	0.44	0.31	0.46	0.21	0	0

03/26/1992

Vehicle Sales (Thousands)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	12219.57	2604.30	1023.76	440.36	17.08	11.34	6.98	44.68	79.98	34.97	111.47	4.55
1999	12048.81	2575.14	1026.66	443.59	17.11	11.22	6.92	45.51	78.98	35.02	111.48	4.43
1998	11900.16	2531.74	1032.18	447.69	18.98	11.07	6.83	45.73	78.09	34.70	110.08	4.27
1997	11777.45	2479.15	1013.02	440.45	17.46	10.67	6.78	45.88	76.84	33.88	108.97	4.22
1996	11672.01	2424.92	992.02	432.24	17.51	10.23	6.78	46.09	75.59	33.29	106.84	4.34
1995	11558.82	2355.09	969.06	422.99	16.59	9.93	6.67	46.57	74.74	33.08	104.90	4.43
1994	11431.18	2316.02	951.04	415.88	16.34	9.68	6.68	48.13	73.65	32.95	105.13	4.30
1993	11293.33	2276.24	942.23	412.92	17.36	9.40	6.63	49.80	72.32	32.97	103.83	4.07
1992	11125.12	2210.50	927.15	406.99	17.69	9.46	6.90	50.56	70.46	32.59	101.90	4.10
1991	10974.68	2143.52	913.44	401.37	16.01	9.53	7.35	51.73	68.50	31.81	100.37	4.06
1990	9301.00	2866.00	756.91	340.06	20.87	27.45	5.06	38.21	85.35	29.12	92.21	3.99
1989	9772.00	2854.00	895.21	402.19	19.20	27.00	7.20	39.10	93.40	34.82	110.28	4.19
1988	10530.00	2926.00	919.84	413.26	14.20	21.20	8.30	53.60	103.00	35.62	112.78	3.02
1987	10277.00	2696.00	810.82	364.28	14.00	2.10	8.20	44.30	102.60	31.49	99.71	4.22
1986	11460.00	2541.00	837.38	376.22	11.60	0.00	5.90	44.80	100.70	27.10	85.80	3.38
1985	11042.00	2408.00	883.34	396.86	10.90	0.00	5.10	48.40	97.00	32.06	101.54	3.37
1984	10391.00	2031.00	844.56	379.44	6.00	0.00	5.00	56.00	78.00	38.64	99.36	3.89
1983	9182.00	1314.00	832.83	374.17	0.00	0.00	1.00	47.00	59.00	2.46	79.54	4.08
1982	7982.00	1102.00	663.09	297.91	1.00	0.00	1.00	44.00	62.00	15.20	60.80	2.96
1981	8536.00	896.00	586.50	263.50	1.00	0.00	2.00	72.00	51.00	24.00	76.00	4.06

Annual Vehicle Miles Travelled for Gas and Diesel Vehicles (Billions)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	160.411	40.802	18.135	7.913	0.357	0.248	0.135	1.734	3.309	1.536	9.628	0.266
1999	149.001	37.346	16.588	7.291	0.320	0.218	0.121	1.601	3.003	1.413	8.829	0.245
1998	138.218	33.855	15.158	6.703	0.317	0.190	0.109	1.452	2.717	1.282	7.961	0.231
1997	127.695	30.475	13.488	5.988	0.259	0.162	0.097	1.303	2.424	1.135	7.130	0.224
1996	116.991	27.284	11.929	5.312	0.230	0.137	0.086	1.154	2.132	0.997	6.237	0.228
1995	104.990	24.093	10.458	4.667	0.192	0.116	0.074	1.011	1.843	0.872	5.376	0.208
1994	91.338	21.386	9.146	4.090	0.165	0.099	0.063	0.890	1.560	0.750	4.647	0.158
1993	75.905	18.776	7.993	3.582	0.152	0.082	0.053	0.775	1.300	0.642	3.897	0.141
1992	59.880	16.117	6.864	3.081	0.133	0.071	0.046	0.646	1.049	0.529	3.193	0.125

1991	44.873	13.647	5.832	2.621	0.102	0.060	0.040	0.539	0.839	0.427	2.598	0.116
1990	27.783	15.723	4.112	1.890	0.111	0.146	0.022	0.321	0.850	0.320	1.950	0.090
1989	20.656	13.360	4.095	1.884	0.091	0.119	0.024	0.258	0.705	0.311	1.888	0.079
1988	15.527	11.543	3.502	1.613	0.056	0.075	0.023	0.233	0.610	0.253	1.554	0.049
1987	10.473	8.876	2.545	1.171	0.045	0.006	0.019	0.138	0.464	0.178	1.101	0.066
1986	8.007	6.934	2.152	0.988	0.031	0.000	0.011	0.112	0.345	0.122	0.757	0.048
1985	5.282	5.412	1.848	0.846	0.022	0.000	0.008	0.106	0.269	0.116	0.714	0.044
1984	3.412	3.737	1.430	0.654	0.010	0.000	0.006	0.093	0.168	0.112	0.555	0.034
1983	2.056	1.973	1.143	0.521	0.000	0.000	0.001	0.060	0.101	0.005	0.352	0.038
1982	1.226	1.349	0.731	0.333	0.001	0.000	0.001	0.044	0.081	0.027	0.214	0.023
1981	0.889	0.897	0.519	0.238	0.001	0.000	0.001	0.054	0.060	0.036	0.211	0.024
Total	1164.612	333.583	137.669	61.385	2.596	1.729	0.938	12.525	23.830	11.064	68.794	2.437

Survival Rates (%)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
1999	99.6	99.3	99.3	99.3	99.3	99.3	99.7	99.7	99.7	99.7	99.7	99.9
1998	98.9	98.2	98.2	98.2	98.2	98.2	99.0	99.0	99.0	99.0	99.0	99.8
1997	97.6	96.8	96.8	96.8	96.8	96.8	97.4	97.4	97.4	97.4	97.4	99.5
1996	95.4	95.0	95.0	95.0	95.0	95.0	94.5	94.5	94.5	94.5	94.5	99.1
1995	91.4	92.6	92.6	92.6	92.6	92.6	90.2	90.2	90.2	90.2	90.2	98.5
1994	85.0	89.6	89.6	89.6	89.6	89.6	84.6	84.6	84.6	84.6	84.6	97.5
1993	75.6	85.8	85.8	85.8	85.8	85.8	78.1	78.1	78.1	78.1	78.1	95.5
1992	64.0	81.3	81.3	81.3	81.3	81.3	70.9	70.9	70.9	70.9	70.9	93.5
1991	51.4	76.1	76.1	76.1	76.1	76.1	63.7	63.7	63.7	63.7	63.7	90.5
1990	39.7	70.3	70.3	70.3	70.3	70.3	56.6	56.6	56.6	56.6	56.6	87.0
1989	29.7	64.3	64.3	64.3	64.3	64.3	49.8	49.8	49.8	49.8	49.8	83.0
1988	21.9	58.1	58.1	58.1	58.1	58.1	43.6	43.6	43.6	43.6	43.6	79.0
1987	16.0	52.0	52.0	52.0	52.0	52.0	38.0	38.0	38.0	38.0	38.0	74.0
1986	11.6	46.2	46.2	46.2	46.2	46.2	33.0	33.0	33.0	33.0	33.0	69.0
1985	8.4	40.8	40.8	40.8	40.8	40.8	28.6	28.6	28.6	28.6	28.6	64.0
1984	6.1	35.8	35.8	35.8	35.8	35.8	24.7	24.7	24.7	24.7	24.7	59.0
1983	4.4	31.3	31.3	31.3	31.3	31.3	21.3	21.3	21.3	21.3	21.3	54.0
1982	3.2	27.3	27.3	27.3	27.3	27.3	18.4	18.4	18.4	18.4	18.4	50.0
1981	2.3	23.8	23.8	23.8	23.8	23.8	15.8	15.8	15.8	15.8	15.8	46.0

VMT Fractions

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2000	0.138	0.122	0.129	0.118	0.114	0.105	0.140	0.079	0.094	0.040	0.000	0.000
1999	0.128	0.112	0.119	0.111	0.105	0.098	0.128	0.081	0.085	0.037	0.000	0.000
1998	0.119	0.101	0.109	0.104	0.106	0.091	0.115	0.081	0.077	0.033	0.000	0.000
1997	0.110	0.091	0.098	0.094	0.088	0.082	0.104	0.080	0.069	0.029	0.000	0.000
1996	0.101	0.082	0.087	0.085	0.080	0.073	0.093	0.077	0.060	0.026	0.000	0.000
1995	0.090	0.072	0.077	0.076	0.068	0.065	0.080	0.074	0.058	0.023	0.000	0.000
1994	0.079	0.064	0.067	0.067	0.060	0.058	0.069	0.070	0.054	0.019	0.000	0.000
1993	0.065	0.057	0.059	0.060	0.057	0.051	0.058	0.065	0.049	0.017	0.000	0.000
1992	0.052	0.049	0.051	0.052	0.052	0.046	0.051	0.059	0.043	0.014	0.000	0.000
1991	0.039	0.041	0.043	0.046	0.041	0.045	0.045	0.053	0.037	0.011	0.000	0.000
1990	0.024	0.047	0.031	0.034	0.047	0.120	0.025	0.033	0.040	0.008	0.000	0.000
1989	0.018	0.040	0.030	0.035	0.067	0.102	0.008	0.030	0.057	0.008	0.000	0.000
1988	0.013	0.035	0.026	0.032	0.041	0.058	0.024	0.051	0.059	0.132	0.000	0.000
1987	0.009	0.027	0.019	0.023	0.033	0.005	0.025	0.040	0.058	0.187	0.000	0.000
1986	0.007	0.021	0.016	0.017	0.023	0.000	0.015	0.032	0.054	0.129	0.000	0.000
1985	0.005	0.016	0.014	0.014	0.012	0.000	0.011	0.024	0.040	0.121	0.000	0.000
1984	0.003	0.011	0.010	0.011	0.006	0.000	0.006	0.024	0.027	0.058	0.000	0.000
1983	0.002	0.006	0.008	0.011	0.000	0.000	0.001	0.017	0.016	0.012	0.000	0.000
1982	0.001	0.004	0.005	0.007	0.001	0.000	0.001	0.013	0.016	0.043	0.000	0.000
1981	0.001	0.003	0.004	0.006	0.001	0.000	0.002	0.018	0.008	0.054	0.000	0.000

Urban Fractions

	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
Diesel	0.59	0.49	0.49	0.79	0.45	0.45	0.45	0.53	0.51	0.42	0.26	1
Gas	0.59	0.49	0.49	0.79	0.44	0.44	0.44	0.31	0.46	0.21	0	0

04/28/1992

Vehicle Sales (Thousands)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	14092.03	3053.28	1184.15	493.06	19.95	13.31	7.80	49.70	89.06	38.78	123.96	4.95
2009	13897.98	3015.53	1173.75	490.56	19.85	13.21	7.72	49.21	88.17	38.57	123.17	4.91
2008	13711.51	2984.84	1159.62	486.17	19.49	12.95	7.71	48.82	87.50	38.20	122.03	4.75
2007	13523.53	2939.59	1139.07	478.96	19.30	12.33	7.66	48.03	86.51	37.69	120.47	4.81
2006	13349.73	2899.56	1124.45	474.41	19.02	12.16	7.54	47.53	85.54	37.20	118.86	4.72
2005	13169.36	2860.96	1111.04	470.21	18.75	12.05	7.45	47.17	84.73	36.98	118.08	4.69
2004	12986.57	2810.22	1094.29	464.46	18.38	11.94	7.37	46.72	83.45	36.84	116.85	4.57
2003	12796.59	2738.43	1078.99	459.31	17.92	11.82	7.20	45.97	82.23	35.34	115.11	4.64
2002	12599.03	2691.14	1055.81	450.93	17.66	11.69	7.12	45.30	81.45	35.32	113.33	4.47
2001	12405.85	2640.89	1036.43	444.14	17.33	11.53	7.06	45.10	80.55	35.13	112.47	4.54
2000	12219.57	2604.30	1023.76	440.36	17.08	11.34	6.98	44.68	79.98	34.97	111.47	4.55
1999	12048.81	2575.14	1026.66	443.59	17.11	11.22	6.92	45.51	78.98	35.02	111.48	4.43
1998	11900.16	2531.74	1032.18	447.69	18.98	11.07	6.83	45.73	78.09	34.70	110.08	4.27
1997	11777.45	2479.15	1013.02	440.45	17.46	10.67	6.78	45.88	76.84	33.88	108.97	4.22
1996	11672.01	2424.92	992.02	432.24	17.51	10.23	6.78	46.09	75.59	33.29	106.84	4.34
1995	11558.82	2355.09	969.06	422.99	16.59	9.93	6.67	46.57	74.74	33.08	104.90	4.43
1994	11431.18	2316.02	951.04	415.88	16.34	9.68	6.68	48.13	73.65	32.95	105.13	4.30
1993	11293.33	2276.24	942.23	412.92	17.36	9.40	6.63	49.80	72.32	32.97	103.83	4.07
1992	11125.12	2210.50	927.15	406.99	17.69	9.46	6.90	50.56	70.46	32.59	101.90	4.10
1991	10974.68	2143.52	913.44	401.37	16.01	9.53	7.35	51.73	68.50	31.81	100.37	4.06

Annual Vehicle Miles Travelled for Gas and Diesel Vehicles (Billions)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	185.058	47.959	21.036	8.839	0.417	0.296	0.150	1.928	3.685	1.703	10.707	0.290
2009	171.925	43.814	18.996	8.042	0.372	0.261	0.136	1.754	3.352	1.557	9.754	0.272
2008	159.304	39.962	17.041	7.258	0.326	0.226	0.123	1.593	3.044	1.411	8.825	0.257
2007	146.666	36.159	15.162	6.493	0.287	0.190	0.109	1.421	2.729	1.263	7.883	0.256
2006	133.859	32.631	13.504	5.812	0.250	0.164	0.095	1.257	2.413	1.114	6.939	0.248
2005	119.661	29.263	11.964	5.172	0.217	0.142	0.082	1.097	2.103	0.974	6.051	0.220
2004	103.798	25.937	10.492	4.553	0.186	0.122	0.070	0.940	1.790	0.839	5.165	0.168
2003	86.035	22.573	9.121	3.972	0.157	0.104	0.057	0.789	1.506	0.688	4.320	0.161
2002	67.831	19.606	7.787	3.403	0.133	0.087	0.047	0.648	1.244	0.573	3.551	0.136

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2001	50.735	16.800	6.591	2.890	0.110	0.072	0.038	0.534	1.018	0.472	2.912	0.129
2000	36.506	14.278	5.542	2.438	0.091	0.059	0.031	0.434	0.828	0.385	2.358	0.103
1999	25.471	12.047	4.684	2.069	0.075	0.048	0.025	0.353	0.663	0.312	1.908	0.084
1998	17.548	9.985	3.923	1.738	0.068	0.039	0.019	0.283	0.529	0.250	1.517	0.069
1997	12.002	8.164	3.178	1.410	0.051	0.030	0.015	0.225	0.418	0.196	1.204	0.066
1996	8.156	6.619	2.549	1.132	0.041	0.023	0.012	0.178	0.329	0.154	0.942	0.061
1995	5.532	5.296	2.027	0.901	0.031	0.018	0.010	0.142	0.258	0.122	0.738	0.058
1994	3.758	4.263	1.609	0.716	0.024	0.014	0.008	0.115	0.201	0.097	0.587	0.038
1993	2.534	3.418	1.285	0.573	0.021	0.011	0.006	0.093	0.156	0.077	0.460	0.037
1992	1.717	2.701	1.016	0.453	0.017	0.008	0.005	0.074	0.120	0.061	0.359	0.032
1991	1.151	2.129	0.804	0.359	0.012	0.007	0.004	0.062	0.098	0.050	0.279	0.024
Total	1339.248	383.604	158.313	68.225	2.885	1.921	1.043	13.920	26.485	12.296	76.459	2.709

Survival Rates (%)

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
2009	99.6	99.3	99.3	99.3	99.3	99.3	99.7	99.7	99.7	99.7	99.7	99.9
2008	98.9	98.2	98.2	98.2	98.2	98.2	99.0	99.0	99.0	99.0	99.0	99.8
2007	97.6	96.8	96.8	96.8	96.8	96.8	97.4	97.4	97.4	97.4	97.4	99.5
2006	95.4	95.0	95.0	95.0	95.0	95.0	94.5	94.5	94.5	94.5	94.5	99.1
2005	91.4	92.6	92.6	92.6	92.6	92.6	90.2	90.2	90.2	90.2	90.2	98.5
2004	85.0	89.6	89.6	89.6	89.6	89.6	84.6	84.6	84.6	84.6	84.6	97.5
2003	75.6	85.8	85.8	85.8	85.8	85.8	78.1	78.1	78.1	78.1	78.1	95.5
2002	64.0	81.3	81.3	81.3	81.3	81.3	70.9	70.9	70.9	70.9	70.9	93.5
2001	51.4	76.1	76.1	76.1	76.1	76.1	63.7	63.7	63.7	63.7	63.7	90.5
2000	39.7	70.3	70.3	70.3	70.3	70.3	56.6	56.6	56.6	56.6	56.6	87.0
1999	29.7	64.3	64.3	64.3	64.3	64.3	49.8	49.8	49.8	49.8	49.8	83.0
1998	21.9	58.1	58.1	58.1	58.1	58.1	43.6	43.6	43.6	43.6	43.6	79.0
1997	16.0	52.0	52.0	52.0	52.0	52.0	38.0	38.0	38.0	38.0	38.0	74.0
1996	11.6	46.2	46.2	46.2	46.2	46.2	33.0	33.0	33.0	33.0	33.0	69.0
1995	8.4	40.8	40.8	40.8	40.8	40.8	28.6	28.6	28.6	28.6	28.6	64.0
1994	6.1	35.8	35.8	35.8	35.8	35.8	24.7	24.7	24.7	24.7	24.7	59.0
1993	4.4	31.3	31.3	31.3	31.3	31.3	21.3	21.3	21.3	21.3	21.3	54.0
1992	3.2	27.3	27.3	27.3	27.3	27.3	18.4	18.4	18.4	18.4	18.4	50.0
1991	2.3	23.8	23.8	23.8	23.8	23.8	15.8	15.8	15.8	15.8	15.8	46.0

VMT Fractions

Year	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
2010	0.138	0.125	0.133	0.130	0.145	0.154	0.144	0.139	0.139	0.139	0.140	0.107
2009	0.128	0.114	0.120	0.118	0.129	0.136	0.130	0.126	0.127	0.127	0.128	0.100
2008	0.119	0.104	0.108	0.106	0.113	0.118	0.118	0.114	0.115	0.115	0.115	0.095
2007	0.110	0.094	0.096	0.095	0.099	0.099	0.105	0.102	0.103	0.103	0.103	0.094
2006	0.100	0.085	0.085	0.085	0.087	0.086	0.092	0.090	0.091	0.091	0.091	0.092
2005	0.089	0.076	0.076	0.076	0.075	0.074	0.079	0.079	0.079	0.079	0.079	0.081
2004	0.078	0.068	0.066	0.067	0.065	0.063	0.067	0.068	0.068	0.068	0.068	0.062
2003	0.064	0.059	0.058	0.058	0.054	0.054	0.055	0.057	0.057	0.056	0.057	0.060
2002	0.051	0.051	0.049	0.050	0.046	0.045	0.045	0.047	0.047	0.047	0.046	0.050
2001	0.038	0.044	0.042	0.042	0.038	0.038	0.037	0.038	0.039	0.038	0.038	0.048
2000	0.027	0.037	0.035	0.036	0.031	0.031	0.030	0.031	0.031	0.031	0.031	0.038
1999	0.019	0.031	0.030	0.030	0.026	0.025	0.024	0.025	0.025	0.025	0.025	0.031
1998	0.013	0.026	0.025	0.026	0.024	0.020	0.019	0.020	0.020	0.020	0.020	0.026
1997	0.009	0.021	0.020	0.021	0.018	0.016	0.015	0.016	0.016	0.016	0.016	0.024
1996	0.006	0.017	0.016	0.017	0.014	0.012	0.012	0.013	0.012	0.013	0.012	0.023
1995	0.004	0.014	0.013	0.013	0.011	0.009	0.009	0.010	0.010	0.010	0.010	0.022
1994	0.003	0.011	0.010	0.011	0.008	0.007	0.007	0.008	0.008	0.008	0.008	0.014
1993	0.002	0.009	0.008	0.008	0.007	0.006	0.006	0.007	0.006	0.006	0.006	0.014
1992	0.001	0.007	0.006	0.007	0.006	0.004	0.005	0.005	0.005	0.005	0.005	0.012
1991	0.001	0.006	0.005	0.005	0.004	0.004	0.004	0.005	0.004	0.004	0.004	0.009

Urban Fractions

	LDV	I	IIA	IIB	III	IV	V	VI	VII	VIIIA	VIIIB	Buses
Diesel	0.59	0.49	0.49	0.79	0.45	0.45	0.45	0.53	0.51	0.42	0.26	1
Gas	0.59	0.49	0.49	0.79	0.44	0.44	0.44	0.31	0.46	0.21	0	0

04/28/1992

Unleaded Gasoline Particulate Emissions

	Catalyst Type	THC g/mile	Particulate mg/mile	Particulate as % THC
Hammerle, et al., 1987 (SAE 920731) (1)				
1991 Escorts (4 cars)	3-way/no air	0.2	2.3	1.2%
1991 Explorers (4 cars)	dual 3-way/no air	0.22	2.5	1.1%
Average		**0.21**	**2.4**	**1.1%**
Lewtas, 1991 (POM Report) (2)				
1978 Mustang II	ox cat		5.3	
Lang et al., 1981 (SAE 811186) (3)				
composite with 1976 Ply Fury (16 cars)	ox; 3-way+ox	2.52	31.7	1.3%
composite without Fury (15 cars)	ox; 3-way+ox	1.05	21	2.0%
ox cat with air (with Fury) (6 cars)		5.05	52.3	1.0%
ox cat with air (without Fury) (5 cars)		1.16	24.4	2.1%
ox cat/no air (5 cars)		1.46	22.2	1.5%
3-way+ox with air (5 cars)		0.53	16.4	3.1%
Volkswagen, 1991 (4)				
1982 Scirocco	ox cat	0.13	8.04	6.2%
1982 Jetta	ox cat	0.21	4.68	2.2%
1982 Jetta	ox cat	0.17	7.34	4.3%
1985 Jetta	ox cat	0.32	4.57	1.4%
1984 Audi 5000S	ox cat	0.26	7.07	2.7%
1985 Audi 5000S	ox cat	0.31	8.38	2.7%
1986 Audi 5000S	ox cat	0.2	6.27	3.1%
Average		**0.23**	**6.62**	**3.2%**
Smith, 1981 (EPA 460/3-81-024 , High Mileage)				
1978 Buick Regal	ox cat/no air	1.71	50.23	2.9%
1979 Mercury Marquis	3-way with air	0.69	26.95	3.9%
1979 Mercury Marquis	3-way with air	0.68	38.15	5.6%
1978 Ford Granada	ox cat with air	1.74	58.2	3.3%
1978 Volvo 245DL	3-way/no air	0.61	13.48	2.2%
1978 Olds Cutlass	ox cat/no air	0.87	31.02	3.6%
1978 Chev Malibu	ox cat with air	0.64	49.54	7.7%
1978 Chev Monte Carlo	ox cat/no air	0.45	32.02	7.1%
1978 Ford Fiesta	ox cat with air	0.64	56.46	8.8%
1978 Chrysler New Yorker	ox cat/no air	4.57	425.15	9.3%
Averages by catalyst type	**ox/no air**	**1.01**	**37.76**	**4.5%**
(New Yorker excluded)	**ox with air**	**1.01**	**54.73**	**6.6%**
	3-way with air	**0.69**	**32.55**	**4.8%**
	3-way/no air	**0.61**	**13.48**	**2.2%**
Urban, 1980a (EPA 460/3-80-003)				
1978 Chev Malibu	ox/no air	0.5	9.41	1.9%
1978 Chev Malibu (Calif.)	ox with air	0.31	5.5	1.8%

Unleaded Gasoline Particulate Emissions

1978 Ford Granada	ox with air	0.43	12.23	2.8%
1978 Ford Mustang II	ox/no air	0.51	9.27	1.8%
Averages by catalyst type	**ox/no air**			**1.8%**
	ox with air			**2.3%**

Urban, 1980b (EPA 460/3-80-004)

1978 Ford Pinto	3way+ox with air	0.11	24.62	22.4%
1978 Pont. Sunbird	3-way	0.26	9.01	3.5%
1978 Saab 99	3-way	0.16	7.88	4.9%
Averages by catalyst type	**3-way**			**4.2%**

Urban, 1980c (EPA 460/3-80-005)

1979 Merc. Marquis	3-way+ox with a	0.21	3.23	1.5%

CARB, 1986 (assumed all 3-way) (5)

1 1986 GM Cutlass 22,834mi	0.329	10.5	3.2%
2 1986 Ford Marquis 21,535mi		6.7	
3 1985 Ford Thunderbird 59,697mi	0.582	13.1	2.3%
4 1986 Toyota Camry 9,672mi	0.21	5.7	2.7%
5 1986 GM DeVille 15,477mi	0.175	72.1	41.2%
6 1986 VW Golf 24,002mi		1.3	
7 1984 Mitsubishi Colt 67,570mi	0.436	10.4	2.4%
9 1976 Chrysler Valiant 130,274mi		81.8	
10 1978 Ford Fairmont 71,362mi			
11 1985 Nissen Sentra 52,317mi		3.5	
		8.9	
Average (all)	**0.35**	**21.4**	**10.3%**
Average (without vehicle 5 and removed)	**0.39**	**9.93**	**2.6%**

STUDY AVERAGES

3-Way/No Air

Hammerle, et al. (8 cars)	0.21	2.40	1.1%
EPA 460/3-81-024 (1 car)	0.61	13.48	2.2%
EPA 460/3-80-004 (2 cars)	0.21	8.45	4.2%
CARB, 1986 (4 cars)	0.39	9.93	2.6%
Average by Study	**0.36**	**8.57**	**2.5%**
Average by Number of Cars	**0.28**	**5.95**	**2.0%**

3-Way+Ox/Air

Lang et al. (5 cars)	0.53	16.4	3.1%
EPA 460/3-80-005 (1 car)	0.21	3.23	1.5%
Average by Study	**0.37**	**9.82**	**2.3%**
Average by Number of Cars	**0.48**	**14.21**	**2.8%**

Ox Cat/Air

Lang et al. (5 cars)	1.16	24.4	2.1%
EPA 460/3-81-024 (3 cars)	1.01	54.73	6.6%
EPA 460/3-80-003 (2 cars)	0.37	8.87	2.3%
Average by Study	**0.85**	**29.33**	**3.7%**
Average by Number of Cars	**0.96**	**30.39**	**3.5%**

Ox Cat/No Air

Lang et al. (5 cars)	1.46	22.2	1.5%
EPA 460/3-81-024 (3 cars)	1.01	37.76	4.5%
EPA 460/3-80-003 (2 cars)	0.51	9.34	1.8%
Average by Study	**0.99**	**23.10**	**2.6%**
Average by Number of Cars	**1.14**	**24.30**	**2.5%**

All Ox Cat

Lang et al. (10 cars)	1.31	23.3	1.8%
VW (7 cars)	0.23	6.62	3.2%
EPA 460/3-81-024 (6 cars)	1.01	46.25	5.6%
EPA 460/3-80-003 (4 cars)	0.44	9.11	2.1%
Average by Study	**0.75**	**21.32**	**3.2%**
Average by Number of Cars	**0.83**	**21.97**	**3.1%**

SUMMARY AVERAGES (# OF CARS)

3-Way/No Air	0.28	5.95	**2.0%**
3-Way+Ox/Air	0.48	14.21	**2.8%**
Ox Cat/Air	0.96	30.39	**3.5%**
Ox Cat/No Air	1.14	24.3	**2.5%**
All Ox Cat	0.83	21.97	**3.1%**

Footnotes:

(1) There were four Escorts and four Explorers tested. The average numbers in this table are the averag
sites except the single sample for the Explorer collected with the TX40 filter. The Escort value is the aver
Explorer value is the average of 27 data points. The vehicles were tested with and without MMT; the 'nc
this table.

(2) The Mustang II was used by EPA to develop a unit risk for gasoline particulate, based on the compa
Note that emissions for this vehicle were measured using the HFET cycle.

(3) The 1976 Plymouth Fury had an oil consumption problem which produced abnormally high particula
 A total of 16 catalyst-equipped vehicles were tested; six were oxidation catalyst-equipped with air, fiv
were oxidation catalyst-equipped without air, and five were 3-way plus oxidation catalyst-equipped w

(4) All the vehicles tested in the VW program were equipped with oxidation catalysts. Particle-bound c

(5) Vehicle 5 exhibited exceptionally high Si, Cl, and SO4.

6/17/92

ges of all the samples at all
age of 30 data points and the
MMT' data were used for

rative potency method.

ate emission rates.
e
ith air.

organics were not measured.