Relative Cancer Risk
of Reformulated Gasoline
and Conventional Gasoline
Sold in the Northeast

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About NESCAUM

The Northeast States for Coordinated Air Use Management, or NESCAUM, is a non-profit association of the eight Northeast states from New Jersey to Maine. Established in 1967, NESCAUM provides technical assistance and policy guidance to member agencies on regional air pollution problems or concern in the Northeast.

Peer Review

We are indebted to the following specialists who reviewed a draft version of this study. Their comments greatly strengthened the final product. Their listing here does not constitute an endorsement of the findings or policy recommendations contained in this study.

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## UNITS, SYMBOLS AND ACRONYMS

### A
- **ACGIH**: American Conference of Governmental Industrial Hygienists
- **ATSDR**: Agency for Toxic Substances and Disease Registry

### B
- **BaP**: Benzo(a)pyrene
- **BTEX**: Benzene, toluene, ethylbenzene, and xylene

### C
- **CAA**: Clean Air Act Amendments of 1990
- **CARB**: California Air Resources Board
- **CFS**: Chronic fatigue syndrome
- **CG**: Conventional gasoline
- **CNS**: Central nervous system
- **CO**: Carbon monoxide

### D
- **DOE**: U.S. Department of Energy
- **DEP**: Department of Environmental Protection

### E
- **EPA**: U.S. Environmental Protection Agency
- **ETBE**: Ethyl tertiary-butyl ether

### H
- **HAPs**: Hazardous air pollutants
- **HC**: Hydrocarbons
- **HEI**: Health Effects Institute

### I
- **IARC**: The International Agency for Research on Cancer
- **IRIS**: Integrated Risk Information System, an EPA database

### M
- **MCS**: Multiple chemical sensitivity
- **MLEs**: Maximum likelihood estimates
- **MTBE**: Methyl tertiary butyl ether
<table>
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<th>Abbreviation</th>
<th>Definition</th>
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<td>NAAQS</td>
<td>National Ambient Air Quality Standards</td>
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<td>NESCAUM</td>
<td>Northeast States for Coordinated Air Use Management</td>
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<tr>
<td>NOx</td>
<td>Nitrogen oxide or oxides of nitrogen</td>
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<tr>
<td>NPRM</td>
<td>Notice of Proposed Rulemaking</td>
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<tr>
<td>ORNL</td>
<td>Oak Ridge National Laboratory</td>
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<tr>
<td>OSHA</td>
<td>Occupational Safety and Health Administration</td>
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<td>OSTP</td>
<td>Office of Science and Technology Policy</td>
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<td>PADDs</td>
<td>Petroleum Allocation for Defense Districts</td>
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<tr>
<td>PAHs</td>
<td>Polycyclic aromatic hydrocarbons</td>
</tr>
<tr>
<td>PAMS</td>
<td>Photochemical Assessment Monitoring Station</td>
</tr>
<tr>
<td>POM</td>
<td>Polycyclic organic matter</td>
</tr>
<tr>
<td>psi</td>
<td>Pounds per square inch</td>
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<tr>
<td>QA/QC</td>
<td>Quality Assurance/Quality Control</td>
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<tr>
<td>Reg-Neg</td>
<td>Regulatory negotiation</td>
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<tr>
<td>RFG</td>
<td>Federal reformulated gasoline</td>
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<tr>
<td>RVP</td>
<td>Reid Vapor Pressure</td>
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<tr>
<td>SIPs</td>
<td>State Implementation Plans</td>
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<tr>
<td>SNPRM</td>
<td>Supplemental Notice of Proposed Rulemaking</td>
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<tr>
<td>TAME</td>
<td>Tertiary amyl methyl ether</td>
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<tr>
<td>TBA</td>
<td>Tertiary butyl alcohol</td>
</tr>
<tr>
<td>TBF</td>
<td>Tertiary butyl formate</td>
</tr>
<tr>
<td>μg/m³</td>
<td>Micrograms per cubic meter</td>
</tr>
<tr>
<td>USTs</td>
<td>Underground storage tanks</td>
</tr>
<tr>
<td>VOCs</td>
<td>Volatile organic compounds</td>
</tr>
<tr>
<td>VMT</td>
<td>Vehicle miles traveled</td>
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Executive Summary

This study evaluated the relative risk of cancer in members of the general population from conventional gasoline compared to federal reformulated gasoline (RFG). The analysis in this study demonstrated that Phase I RFG served to reduce the relative cancer risk associated with gasoline vapors and automobile exhaust compared to conventional gasoline in the Northeast. These public health benefits are projected to increase with the introduction of Phase II RFG in the year 2000. Notably, the demonstrated benefits associated with the use of RFG were found even after including methyl tertiary butyl ether (MTBE) in the analysis as a potential carcinogen.

A. Overview

Gasoline-powered motor vehicles are a major source of carbon monoxide (CO), volatile organic compounds (VOCs) and oxides of nitrogen (NOx). Gasoline also represents a ubiquitous source of other toxic air pollutants emitted as gasoline evaporates and when it is combusted. Three primary strategies have been employed to reduce emissions from internal combustion engines: (1) engine modifications, such as fuel injection; (2) aftertreatment, such as catalytic converters; and (3) fuel reformulation.

The Clean Air Act Amendments of 1990 (CAA) required the use of RFG in the country's nine worst ozone nonattainment areas and provided other nonattainment areas with the authority to opt into the federal reformulated gasoline (RFG) program. In 1995, RFG was introduced in seven of the Northeast states and other urban areas across the nation in order to reduce ambient concentrations of ground-level ozone in the summer and toxic air pollutants throughout the year (see Table ES-2). Most of these new gasoline blends contained about 10 percent by volume of a chemical compound called methyl tertiary butyl ether, or MTBE.¹

Public reaction to this federally-required reformulated gasoline was mixed. Analyses of ambient air quality data appeared to find significant reductions in several toxic air pollutants, such as benzene, associated with the introduction of RFG. However, some individuals reported experiencing acute effects from exposure to RFG, which included headaches, dizziness and irritation of the eyes and throat. These reports raised questions about the

¹ MTBE has been present in gasoline in small amounts for many years prior to the introduction of RFG, which contains MTBE at 2 percent by weight, or 10 percent by volume.
overall public health benefits of RFG compared to conventional gasoline. Epidemiological studies conducted in several states demonstrated that the general population was not experiencing adverse acute health effects from exposure to MTBE, but did not rule out the possibility that certain individuals may experience acute symptoms from exposure to RFG containing MTBE.

B. Scope of Study

When designing a public health protection program, it is critical to ensure that the strategy employed does not result in a transfer of risks from one segment of the population to another or an increase in one risk while causing a reduction in another. For example, the RFG program’s mandatory addition of an oxygenate, primarily MTBE, to gasoline in areas of high carbon monoxide and ozone pollution has raised concerns that the “cleaner” gasoline has caused more of a public health risk than the product it replaced.

This study assessed the relative cancer risk in members of the general population from conventional and reformulated gasoline blends. The goal of this study was to inform policy makers and the public about the impact of reformulated gasoline as a long-term public health protection option. While there are many known and suspected toxic compounds associated with gasoline, this study relied on an assessment of the subset of five toxic air pollutants identified by Congress in the Clean Air Act for reduction in the aggregate. We have also included MTBE as a toxic in this assessment given the public concern expressed in some areas regarding its potential adverse health effects. While most of these six pollutants present both acute (short-term) and chronic (long-term) health risks, this study assessed only the carcinogenic risk associated with these pollutants.

Carcinogenicity was selected as the appropriate toxicity endpoint for use in this comparison due to several factors. First, gasoline contains many known and suspected human carcinogens. Second, specific concerns have been raised about the potential carcinogenicity of emissions of gasoline containing MTBE. Third, the toxins regulated by the RFG program are all known or suspected human carcinogens. Finally, since the carcinogenic potency of the compounds of interest and emissions factors for these compounds are quantifiable, a reasonable toxicity comparison can be made.

Previous estimates of the toxicity of gasoline blends have provided incomplete evidence that RFG was more protective of public health than conventional gasoline. This study improved upon previous assessments in several ways. This analysis recognized that the carcinogenicity of different compounds varies (e.g., benzene is an order of magnitude more carcinogenic than MTBE), and assigned relative cancer potencies to the six toxic air
pollutants of interest. This study evaluated MTBE in its analysis as a carcinogen in order to provide the greatest margin of safety. Additionally, this study used 1996 conventional gasoline sold in the Northeast as a baseline, which more accurately reflects the choice faced by Northeast policy makers who must decide whether or not to continue with the RFG program.

C. Methodology

A multi-step approach was used to quantify the relative cancer risk of conventional gasoline, Phase I RFG and Phase II RFG. The first step involved developing estimates of the average fuel quality parameters for each of the gasoline blends available in the Northeast. The second step included quantifying cancer unit risk values and cancer potency ratios for each of the six toxins of interest. The third step utilized a modified version of the Complex Model, which incorporated MTBE emission rates, to derive motor vehicle evaporative and exhaust emission estimates for the six toxic compounds. The final step quantified the relative cancer risk for each gasoline blend by multiplying the emissions of each toxic air pollutant by the appropriate cancer potency ratio.

C.1 Fuel Quality

The Northeast receives the majority of its gasoline supply from refineries located in three areas: the Northeast, the Gulf Coast, and foreign refineries. For national security reasons, refineries in the United States are regulated by the Department of Defense, and refinery regions are called Petroleum Allocation for Defense Districts, or PADDs. Northeast refineries are part of PADD I and provide about 40 percent of this region’s gasoline supply. Gulf Coast refineries are located in PADD III, and provide about 40 percent of the Northeast’s gasoline supply. Foreign refineries, or imports, provide the remaining 20 percent of gasoline to the Northeast.

Average fuel formulations for Conventional Gasoline (CG), and Phase I RFG for each PADD and imports were provided to NESCAUM by the Department of Energy (DOE) and the U.S. Environmental Protection Agency (EPA). The EPA data base for 1996 gasoline quality was used to estimate the volume-weighted average fuel formulations for baseline CG and Phase I RFG from PADD I and foreign refineries. The results from a 1996 survey of petroleum refineries conducted for the American Petroleum Institute (API) were used to generate average fuel formulations for baseline CG and Phase I

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2 It is possible, though we believe unlikely, that the analysis overestimated the risk posed by MTBE, and that MTBE will not be shown to be carcinogenic with subsequent review. The risk assessment approach used for MTBE in this study is described in Appendix A.
RFG in PADD III, but only for that subset of refineries producing both CG and Phase I RFG.

Estimates of the chemical composition of Phase II RFG were based on PADD-specific modeling conducted by DOE's Oak Ridge National Laboratory. Since foreign refineries were not included in the Oak Ridge analysis, NESCAUM used DOE's prediction for Phase II gasoline properties expected from foreign refineries. The chemical compositions of average baseline conventional gasoline, Phase I and Phase II RFG are found in Table ES-3.

C.2 Health Effects and Relative Cancer Risk

This study developed numerical estimates of cancer potency for benzene, 1,3-butadiene, acetaldehyde, polycyclic organic matter (POM), formaldehyde, and MTBE. MTBE was considered a potential carcinogen in order to provide a "worse case" scenario, although it is possible that future studies will not characterize MTBE as a carcinogen. An assessment of acute effects was not included in this modeling analysis due to difficulties in quantification. However, the known and suspected acute effect associated with each of the six pollutants are summarized in Chapter IV.

When establishing a health protective standard for a cancer-causing compound, public health experts have traditionally, and appropriately, used the highest plausible cancer risk, also known as the "upper bound" cancer risk, in order to provide an adequate margin of safety to the public. One source of numerical estimates of cancer risk associated with various compounds is an EPA database, called IRIS,3 which contains a mixture of upper bound cancer risk estimates and average cancer risk estimates, better known as maximum likelihood estimates (MLE's). NESCAUM and its member states believe that it is necessary to apply the upper bound or IRIS risk values when setting regulatory limits on toxic emissions.

However, the analysis in this study did not seek to establish health-protective cancer risk standards, but instead sought to quantify the relationship between the cancer risk of conventional gasoline and federal reformulated gasoline. Therefore, an innovative third approach (Method 3) was developed which utilized the MLE to derive numerical cancer potency estimates for each toxic air pollutant. MLEs are based on average cancer risk estimates from each peer-reviewed study and provide the most appropriate comparison of relative cancer risk. The Upper Bound and IRIS approaches (Method 1 and Method 2) were used to explore the sensitivity of the Base Case results to changes in assumptions of cancer risk.

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3 IRIS is an acronym for Integrated Risk Information System.
Since this study was designed to assess relative cancer risk, rather than absolute cancer risk, the numerical estimates of cancer risk were converted to cancer potency ratios compared to benzene.\(^4\) The cancer potency ratios are meaningful only as they related to one another, and should not be taken out of context. Three sets of cancer potency values used for these analyses are presented in Table ES-1.\(^5\)

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Method 1</th>
<th>Method 2</th>
<th>Method 3 (MLE)</th>
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<tbody>
<tr>
<td></td>
<td>IRIS</td>
<td>Upper Bound</td>
<td>Base Case</td>
</tr>
<tr>
<td>Benzene</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>MTBE</td>
<td>0.5</td>
<td>0.16</td>
<td>0.14</td>
</tr>
<tr>
<td>1,3-butadiene</td>
<td>33.7</td>
<td>3.1</td>
<td>3.58</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>1.6</td>
<td>0.9</td>
<td>0.004</td>
</tr>
<tr>
<td>Acetaldehyde</td>
<td>0.3</td>
<td>0.16</td>
<td>0.07</td>
</tr>
<tr>
<td>POM</td>
<td>2.0</td>
<td>5.86</td>
<td>7.1</td>
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Cancer potency ratios differ widely between chemical compounds. For example, using the Base Case (Method 3), POM was assigned the highest cancer potency ratio, followed by 1,3-butadiene and benzene. MTBE’s cancer potency ratio was approximately one order of magnitude lower than benzene’s; and acetaldehyde and formaldehyde were determined to have the lowest cancer potency ratios in the set of compounds evaluated. In addition, the same chemical compound was assigned substantially different cancer potency ratios depending on the Method. For example, the cancer potency ratio of 1,3-butadiene varies from 33.7 in Method 1 to 3.1 in Method 2. The wide differences within and between each Method highlights the importance of using appropriate statistical methods and assigning comparable estimates of cancer risk to each compound.

\(^4\) Cancer potency ratios were generated by dividing the numerical cancer risk estimates for each of the six toxic air pollutants by the numerical cancer risk estimate for benzene. For instance, benzene is divided by itself and becomes 1.0 for each method.

\(^5\) Cancer unit risk values are defined as the increased risk of cancer for each incremental increase in exposure.
C.3 Results of Relative Cancer Risk Analysis

The findings in this study suggest that RFG has served to reduce the relative cancer risk associated with gasoline vapors and automobile exhaust when compared against conventional gasoline in the Northeast. Public health benefits from RFG are projected to increase with the introduction of Phase II RFG in the Northeast in the year 2000. Notably, substantial public health benefits were predicted with the use of RFG rather than conventional gasoline, even after including MTBE in the analysis as a carcinogen.

The major findings of this study are presented in Figure ES-1. The Y-axis represents the percent decrease in relative cancer risk associated with the use of Phase I RFG (left bars in white) and Phase II RFG (right bars in gray) as compared with conventional gasoline. The relative cancer risk associated with the use of conventional gasoline is the baseline against which RFG Phase I and Phase II are compared, and thus CG is not depicted in this chart. Moving from left to right, the X-axis identifies the three petroleum production regions which supply the Northeast with gasoline, and then aggregates the relative cancer risk of the gasoline blends from each region by their proportional share of the Northeast gasoline market for the final result, which is labeled "Northeast."

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure.png}
\caption{Percentage Change in Relative Cancer Risk from Conventional Gasoline Sold in the Northeast (Summer)}
\end{figure}

This analysis found that the use of Phase I RFG in the Northeast has resulted in a 12 percent reduction in the relative cancer risk associated
automobile evaporative and exhaust emissions compared to conventional gasoline. These benefits are projected to increase to 20 percent when Phase II RFG is introduced in the Northeast in the year 2000. As a point of reference, federal regulations require a 15 percent reduction in toxic mass emissions from Phase I RFG, and a 20 percent reduction in toxic mass emissions from Phase II RFG, but this requirement does not account for MTBE. The results of this modeling analysis are consistent with the evidence that ambient levels of benzene have declined since the introduction of Phase I RFG in certain parts of the country (Sonoma, 1995).

The second set of findings are found in Figure ES-2, which presents the predicted relative cancer risk for each individual toxic air pollutant. The numbers along the Y-axis provide a measure of the relative cancer risk of the toxic air pollutants, but are not meaningful by themselves and should not be taken out of context. The six toxic air pollutants are listed along the X-axis. Moving from left to right, the relative cancer risk for each chemical compound is estimated based on the emissions from CG, RFG Phase I, and RFG Phase II. These results provide policy makers with an assessment of the appropriateness and effectiveness of Phase I RFG, and an estimate of the additional improvement expected with the introduction of Phase II RFG, for each chemical compound evaluated in this study.

According to this analysis, benzene is the most significant contributor to cancer risk of the six toxic compounds evaluated, accounting for more of the relative cancer risk than the five other pollutants combined. The wisdom of Congress' requirement that petroleum refiners reduce the amount of benzene in gasoline to below 1.0 percent by volume (the benzene cap) is supported by this finding. This study found that petroleum refiners reduced the benzene content in RFG to about 0.7 percent by volume — or 30 percent below the legal limit of 1.0 percent by volume — presumably due to a market demand for this chemical compound. The fact that benzene remains the primary contributor to cancer risk in Phase II RFG as compared with the five other toxic air pollutants indicates that a continued regulatory focus on benzene is appropriate.

Butadiene poses the next highest level of risk in conventional gasoline and Phase I RFG. The cancer potency of 1,3-butadiene is three times that of benzene. Exhause emissions of 1,3-butadiene are directly related to the olefin content in gasoline, which is not directly regulated under the RFG program.

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6 For the Base Case Scenario, which used maximum likelihood estimates to derive numerical cancer risk values.
The 15 percent reduction in butadiene emissions from Phase I RFG as compared with conventional gasoline is presumed to result from the displacement of olefin levels by the addition of MTBE at 11 percent by volume. The additional 15 percent reduction in emissions of 1,3-butadiene predicted from the introduction of Phase II RFG is primarily due to olefin reductions in order to comply with Phase II NOx requirements. Should they deem it necessary, public policy makers might consider direct regulation of gasoline olefin content as a means of reducing the significant contribution of 1,3-butadiene to overall relative cancer risk.

According to this pollutant-by-pollutant analysis, the relative cancer risk from MTBE is slightly greater than the relative cancer risk associated with 1,3 butadiene for Phase II RFG formulations. While this places MTBE as the second largest contributor to relative cancer risk after benzene in Phase II RFG, it is imperative to recognize that this analysis characterized MTBE as a carcinogen despite disagreement from some public health experts, and that the MTBE model employed by this study assumed the highest emissions factor for MTBE evaporative emissions. In simple terms, the MTBE analysis represents a worst case scenario in order to provide the greatest margin of safety. While this approach strengthened the reliability of this study’s major conclusion — that RFG with MTBE provides a substantial long term public
health benefit to the general population — it may provide a less definitive measure of the relative importance of MTBE as compared with the five other toxic air pollutants.

Acetaldehyde, POM, and formaldehyde rank in that order according to estimated relative cancer risk. As with 1,3-butadiene, there is no direct requirement that petroleum refiners reduce emissions from these toxic air pollutants, other than the general requirement that RFG provide a reduction in mass emissions of toxic air pollutants. The 10 percent reduction in emissions from acetaldehyde and POM from Phase I RFG compared to conventional gasoline are presumably due to the corresponding addition of MTBE at about 10 percent by volume. Emissions of formaldehyde are greater with the use of RFG than with CG, but are too small to appear in Figure ES-2 due to the very low cancer potency ratio for formaldehyde.

D. Conclusions

The results of this analysis comparing the relative cancer risk of conventional gasoline to that of RFG suggest that:

- Phase I federal reformulated gasoline sold in the Northeast in 1996 served to reduce the cancer risk associated with gasoline vapors and automobile exhaust compared to conventional gasoline by 12 percent in our Base Case analysis.

- Phase II federal reformulated gasoline is expected to further reduce the public cancer risk from exposure to gasoline vapors and automobile exhaust as compared with conventional gasoline by 20 percent in our Base Case analysis.

- For all gasoline blends, benzene's contribution to overall cancer risk is greater than the sum of the cancer risks of the other five toxic compounds. This finding affirms the decision made by the drafters of the Clean Air Act Amendments of 1990 to establish specific gasoline fuel specifications for benzene. Despite reductions in cancer risk from benzene of about 30 percent from conventional gasoline to RFG Phase I and Phase II, this pollutant continues to represent the primary cancer risk as rated against the five other toxic compounds.

- Since the cancer potency of MTBE is significantly less than that of benzene, 1,3 butadiene and POM, its presence in RFG at 10 percent by volume tends to dilute these and other carcinogens commonly found in conventional gasoline. This “dilution effect” contributes to a reduction in the overall
relative cancer risk associated with RFG as compared with conventional gasoline.

- While the acute health effects from conventional gasoline and RFG oxygenated with MTBE reported by certain segments of the population have neither been proven nor dismissed, it is important to consider the widespread benefits of reductions in cancer risk and ground level ozone formation associated with the use of RFG Phase I and Phase II when evaluating public policy choices.

E. Future Research Objectives

While this study suggests that the federal reformulated gasoline program is providing important public health benefits, there is a need for further study to verify this conclusion and assess the overall implications of the RFG program, particularly with the pending introduction of Phase II RFG in 2000. NESCAUM suggests additional work in the following areas: (1) ambient monitoring; (2) exposure assessment; (3) MTBE health effects; and (4) MTBE fate and transport, including groundwater contamination.

- **Ambient Monitoring.** Measured reduction of toxics in the air represent the best indicator of the effectiveness of the RFG program. Therefore, continued collection and further analysis of ambient monitoring data is necessary to establish a pre-Phase II baseline and to confirm the model-predicted impact of RFG on air quality and public health.

- **Exposure Assessment.** Given the different residence times of the various motor vehicle-related air toxics, an exposure assessment of hazardous air pollutants (HAPs) from fuel combustion and evaporative emissions is warranted to more accurately depict the relative risk of conventional gasoline and RFG.

- **MTBE Health Effects.** RFG is expected to reduce the cancer risk from exposure to gasoline vapors and automobile exhaust compared to conventional gasoline. However, assuming the continued use of ether-based oxygenates, further research is needed on systemic, neurotoxic, reproductive, and developmental effects of MTBE and MTBE inhaled with RFG.

- **MTBE Fate and Transport.** Because MTBE is highly water soluble relative to other constituents in gasoline, and is poorly adsorbed to soils, it moves with the groundwater flow largely unretarded. Therefore, when RFG is released to the environment, it presents an increased potential for
groundwater contamination compared to conventional gasoline. Further studies are necessary to investigate the potential for MTBE groundwater contamination and the resulting public health and ecological effects.

Table ES-2

Mandatory and Opt-In RFG Areas
(Areas in **bold** within the NESCAUM region)

<table>
<thead>
<tr>
<th>Mandatory Areas</th>
<th>Opt-in Areas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hartford - New Britain - Middletown - New Haven - Meriden - Waterbury, Connecticut area</td>
<td>Entire State(s) of Connecticut, Rhode Island, Massachusetts</td>
</tr>
<tr>
<td>New York - northern New Jersey - Long Island - Connecticut area</td>
<td>Delaware (Sussex County)</td>
</tr>
<tr>
<td>Philadelphia - Wilmington - Trenton - Cecil County, Maryland area</td>
<td>Maine (Knox, Lincoln, Androscoggin, Kennebec, Cumberland, Sagadahoc, and York Counties) New York (Dutchess County)</td>
</tr>
<tr>
<td>Baltimore, Maryland area</td>
<td>New Jersey (Warren, Atlantic, and Cape May Counties)</td>
</tr>
<tr>
<td>Chicago - Gary - Lake County, Illinois area</td>
<td>New Hampshire (Hillsborough, Rockingham, Merrimack, and Strafford Counties)</td>
</tr>
<tr>
<td>Milwaukee - Racine, Wisconsin area</td>
<td>Maryland (Calvert, Charles, Frederick, Montgomery, Prince Georges, Queen Anne’s, and Kent Counties)</td>
</tr>
<tr>
<td>Houston - Galveston - Brazoria, Texas area</td>
<td>Virginia (Cities of Alexandria, Fairfax, Falls Church, Manassas, Manassas Park, Colonial Heights, Hopewell, Richmond, Chesapeake, Hampton, Newport News, Norfolk, Poquoson, Suffolk, Virginia Beach, and Williamsburg; Counties of Arlington, Fairfax, Loudoun, Prince William, Stratford, Charles City, Chesterfield, Hanover, Henrico, James City, and York) Texas (Collin, Dallas, Denton, and Tarrant Counties)</td>
</tr>
<tr>
<td></td>
<td>Kentucky (Boone, Campbell, Kenton, Jefferson, partial Bullitt, and partial Oldham Counties) Washington, D.C.)</td>
</tr>
</tbody>
</table>
Table ES-3

Average Fuel Composition of Conventional, Phase I and Phase II Gasolines Sold in the Northeast from RFG-Producing Refineries

**PADD I**

<table>
<thead>
<tr>
<th>Fuel Parameters</th>
<th>Conventional Gasoline</th>
<th>Phase I RFG</th>
<th>Phase II RFG (predicted)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfur, ppm</td>
<td>382</td>
<td>266</td>
<td>132</td>
</tr>
<tr>
<td>Olefins, %vol.</td>
<td>16</td>
<td>15.5</td>
<td>12.4</td>
</tr>
<tr>
<td>Benzene, %vol.</td>
<td>1.1</td>
<td>0.61</td>
<td>0.7</td>
</tr>
<tr>
<td>Oxygen (MTBE), %wt.</td>
<td>0</td>
<td>2.1</td>
<td>2.19</td>
</tr>
<tr>
<td>RVP, psi</td>
<td>8.7</td>
<td>7.9</td>
<td>6.5</td>
</tr>
<tr>
<td>Aromatics, %vol.</td>
<td>23.8</td>
<td>17.50</td>
<td>25.0</td>
</tr>
<tr>
<td>E200</td>
<td>47</td>
<td>50.4</td>
<td>55.0</td>
</tr>
<tr>
<td>E300</td>
<td>77.5</td>
<td>77.5</td>
<td>87.0</td>
</tr>
</tbody>
</table>

**PADD III**

<table>
<thead>
<tr>
<th>Fuel Parameters</th>
<th>Conventional Gasoline</th>
<th>Phase I RFG</th>
<th>Phase II RFG (predicted)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfur, ppm</td>
<td>490</td>
<td>347</td>
<td>127</td>
</tr>
<tr>
<td>Olefins, %vol.</td>
<td>16.0</td>
<td>13.8</td>
<td>9.0</td>
</tr>
<tr>
<td>Benzene, %vol.</td>
<td>0.88</td>
<td>0.68</td>
<td>0.7</td>
</tr>
<tr>
<td>Oxygen (MTBE), %wt.</td>
<td>0.05</td>
<td>1.89</td>
<td>2.19</td>
</tr>
<tr>
<td>RVP, psi</td>
<td>8.2</td>
<td>7.2</td>
<td>6.5</td>
</tr>
<tr>
<td>Aromatics, %vol.</td>
<td>26.6</td>
<td>21.3</td>
<td>25.0</td>
</tr>
<tr>
<td>E200</td>
<td>48.6</td>
<td>51.1</td>
<td>59.7</td>
</tr>
<tr>
<td>E300</td>
<td>77.2</td>
<td>79.70</td>
<td>86.2</td>
</tr>
</tbody>
</table>

**IMPORTS**

<table>
<thead>
<tr>
<th>Fuel Parameters</th>
<th>Conventional Gasoline</th>
<th>Phase I RFG</th>
<th>Phase II RFG (predicted)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfur, ppm</td>
<td>303</td>
<td>192</td>
<td>192</td>
</tr>
<tr>
<td>Olefins, %vol.</td>
<td>12.6</td>
<td>7.7</td>
<td>7.7</td>
</tr>
<tr>
<td>Benzene, %vol.</td>
<td>0.98</td>
<td>0.64</td>
<td>0.64</td>
</tr>
<tr>
<td>Oxygen (MTBE), %wt.</td>
<td>0.04</td>
<td>2.1</td>
<td>2.1</td>
</tr>
<tr>
<td>RVP, psi</td>
<td>8.7</td>
<td>7.9</td>
<td>6.5</td>
</tr>
<tr>
<td>Aromatics, %vol.</td>
<td>23.7</td>
<td>20.70</td>
<td>20.70</td>
</tr>
<tr>
<td>E200</td>
<td>47.00</td>
<td>51.10</td>
<td>51.10</td>
</tr>
<tr>
<td>E300</td>
<td>77.50</td>
<td>79.70</td>
<td>79.70</td>
</tr>
</tbody>
</table>
I. Introduction

A. Study Overview

Through this analysis, the Northeast States for Coordinated Air Use Management (NESCAUM) compared the long-term relative risk associated with human exposure to the exhaust and evaporative emissions of toxic air pollutants from motor vehicles burning conventional gasoline and federal reformulated gasoline (RFG). The study focuses on the carcinogenic effects associated with the subset of toxic constituents from gasoline that are regulated under Section 211 of the Clean Air Act Amendments of 1990 (CAA), as well as the risk posed by the oxygenate methyl tertiary butyl ether or MTBE. The information generated through this study is intended to further inform Northeast decision makers as to the relative risk to public health posed by toxic emissions from RFG compared to conventional gasoline.

Gasoline-powered motor vehicles are a major source of carbon monoxide (CO), volatile organic compounds (VOCs) and oxides of nitrogen (NOx). Under its CAA mandate, the U.S. Environmental Protection Agency (EPA) has established a national ambient air quality standard (NAAQS) for CO. VOCs and NOx are the primary precursors to the criteria pollutant ozone. Gasoline also represents a ubiquitous source of other toxic air pollutants emitted as gasoline evaporates and when it is combusted in motor vehicles.

The RFG program represents an important air quality improvement strategy in the NESCAUM region. Several areas of the Northeast including the New York City, Philadelphia and Hartford metropolitan areas are required under the CAA to participate in the RFG program. Other states in the region have voluntarily opted all or some portion of their jurisdictions into the program as part of a comprehensive effort to lower ozone precursor emissions. This strategy represents an integral component of the state implementation plans (SIPs) for ozone in these states. The decision to adopt RFG rather than some alternative ozone precursor reduction program stems, in part, from the collateral toxic benefits expected to accrue through the implementation of this program.

Studies have found strong correlations between the introduction of cleaner-burning gasoline and ambient air quality improvements in ozone concentrations, ozone precursors and air toxics. Analysis of ambient ozone data from before and after the introduction of low volatility gasoline in the Northeast beginning in 1989 measurably reduced ozone levels in the program areas. The introduction of cleaner-burning gasoline in California in 1996 resulted in overall reductions in ozone ranging from 4 percent to 17 percent, after adjusting for meteorological differences, between 1995 and years prior to
the introduction of cleaner-burning gasoline (CARB, January, 1998). In addition, average benzene concentrations at 11 monitoring sites in Northern California were more than 50 percent less in the Spring of 1996 than in the Spring of 1995 (CARB, July, 1996).

Preliminary analysis of ambient VOC data collected at Photochemical Assessment Monitoring Station (PAMS) sites located in RFG areas found statistically significant changes at some sites consistent with RFG introduction. From 1994 to 1995, significant decreases in ambient benzene median weight percent (benzene to total non-methane hydrocarbons) and benzene ratios (benzene to toluene and benzene to acetylene) were detected at a number of sites. Available gasoline property data verifies that the benzene content of the gasoline sold in these RFG areas decreased between 1994 and 1995. Ambient levels of certain aromatic species such as total xylenes and trimethylbenzenes also showed significant declines at a number of the monitoring sites in RFG areas (Sonoma Technology, 1998).

Despite data suggesting significant reductions in ambient levels of several toxic air pollutants associated with the introduction of RFG, some individuals have reported experiencing acute effects from exposure to RFG including headaches, dizziness and irritation of the eyes and throat, raising questions as to the overall public health benefits of RFG compared to conventional gasoline. Since the addition of relatively higher levels of MTBE is among the primary formulation changes associated with RFG, it has been hypothesized that this additive may contribute to such acute effects. The June, 1997 Office of Science and Technology Policy (OSTP) report, Interagency Assessment of Oxygenated Fuels, concluded from the limited number of studies conducted to date that MTBE exposures to the overall population did not produce a significant increase in acute symptoms or illnesses over background levels. However, the report recognized that “[a]nedotal reports of acute health symptoms among some individuals at very low levels of exposure to oxygenate cannot be adequately explained, but cannot be dismissed.”

Several areas which initially opted into the program — including parts of Maine, Pennsylvania, and New York — subsequently dropped out of the RFG program, primarily due to concerns about potential price spikes and possibly some concerns about health effects associated with oxygenate exposure. Several mandated oxygenated fuel program areas have modified their programs in response to concerns regarding MTBE exposure. The State of Alaska, for example, switched to an ethanol program in Anchorage and New Jersey reduced the length of the oxyfuel season. These actions have had an adverse economic impact on gasoline refiners who invested capital to produce RFG and oxygenated fuels to meet expected market demands. The decision by certain jurisdictions to opt out of the program resulted in a smaller than anticipated market for this product.
In order to enable gasoline refiners to recover a portion of the capital invested to comply with the Phase II requirements and meet RFG demand, states were required to determine by the end of 1997 whether their voluntary opt-in areas will remain in the federal RFG program after the Phase II program takes effect on January 1, 2000. States that have chosen to remain in the program are committed to do so through the end of 2003. To date, no states have opted-out of the program. The state of Maine has been granted an extension to the opt-out deadline.

In the CAA, Congress requires the use of federal RFG in the country’s nine worst ozone nonattainment areas and provides other nonattainment areas with the authority to opt into the RFG program. The CAA included specific formulation requirements and performance standards for reformulated gasoline including: a minimum 2 wt percent oxygen requirement, a 1 percent benzene cap, and a 15 percent and 20 percent - 25 percent reduction in VOCs and toxics for Phase I and Phase II, respectively. The performance standards for toxics apply over the entire year, even though ozone noncompliance is a seasonal problem in most areas, revealing Congress’ intent that the RFG program was to serve broader air quality needs than strictly helping areas comply with the ozone standard.

The CAA establishes a performance standard for RFG requiring a percent reduction in mass emissions of five specified air toxics. The term “toxic air pollutants”, as it applies to RFG, is defined by the CAA as the aggregate mass emissions of: benzene, 1,3-butadiene, polycyclic organic matter (POM), acetaldehyde, and formaldehyde. Compliance is demonstrated using the “Complex Model” which estimates exhaust and evaporative emissions of gasoline formulations used in vehicles with 1990 emission control technologies. This model does not account for differences in the potency of the five toxic air pollutants.

Analysis conducted with the Complex Model suggests that typical reformulation strategies will decrease some toxic constituents, increase emissions of some toxics and leave others relatively unchanged. While each of the regulated pollutants are considered known or probable human carcinogens, they have a wide range of applicable “cancer potency factors”. Consequently, use of the Complex Model alone does not suggest the degree to which the achieved reduction in mass toxic emissions translate to commensurate public health benefits.

In order to better assess the aggregate toxic benefits of the RFG program, NESCAUM used a toxics “weighting” technique. Under this approach, the emission rates for each of the toxic compounds quantified by the Complex Model are multiplied by an appropriate cancer potency ratio to generate a “Relative Cancer Risk” value for several complying fuel formulations. Relative Cancer Risk is a term used in this study for the product of a
particular toxic air pollutant's mass emissions multiplied by its corresponding cancer potency ratio. Significantly, this study includes MTBE in addition to the five CAA-regulated toxic pollutants. NESCAUM believes that including this potential carcinogen in the risk assessment provides a more conservative and conservative comparison of the relative toxicity of conventional and reformulated fuels.

All gasoline, whether conventional or reformulated, presents acute and chronic health hazards. The critical question addressed in this study is whether RFG is more protective of public health than the conventional gasoline it has replaced from the perspective of reducing the aggregate risk associated with exposure to these toxic air pollutants. Rather than attempting to quantify typical exposure levels and assign health risk estimates, we assume that emissions are proportional to exposure and compare the relative toxicity of various fuel formulations. The base assumption being: the more toxic the fuel, the greater the exposure, the greater the public health risk.

Three sets of cancer unit risk values for RFG's five regulated toxic compounds and MTBE were considered in this analysis: (1) EPA's IRIS values; (2) the 95 percent upper bound estimates; and (3) maximum likelihood estimates (MLEs). Since NESCAUM believes that the maximum likelihood estimates provide the most appropriate set of cancer unit risk values for the purpose of a relative risk analysis, this study relies primarily on the MLE approach. NESCAUM and its member states believe, however, that it is necessary to apply the upper bound or IRIS cancer risk values when setting regulatory limits on toxic emissions. A sensitivity analysis was conducted to assess the effect of two alternative cancer unit risk value approaches would have on the results of this modeling exercise.

Using survey data collected by EPA, industry-generated data and best engineering judgment, NESCAUM and the U.S. Department of Energy (DOE) estimated average fuel properties for conventional gasoline (CG), Phase I RFG and Phase II RFG formulations sold in the Northeast. These fuel formulas were entered into the Complex Model to generate mass emission rates for each of the fuels. The predicted mass emission rates for each of the six toxic air pollutants were subsequently multiplied by the applicable cancer unit risk value. The results for the individual pollutants were summed, providing a weighted risk estimate for each formulation. These resulting risk estimates were then compared to assess the relative cancer risk associated with each fuel studied.

Section II of this Report provides background information on the regulatory context of the RFG program. Section III discusses fuel quality and estimates average fuel parameters for conventional and reformulated gasoline blends. Section IV provides background information on the health effects and explains the derivation of the cancer unit risk values for each of
the six toxic compounds of interest. Section V describes the methodology used to estimate the relative cancer risk associated with the various fuels and presents the results of this assessment.

B. Risk Management in Environmental Policy

Elected officials and environmental regulators face difficult risk management decisions in designing and implementing programs to protect public health and the environment from the adverse effects of pollution. The control of air emissions associated with the combustion of gasoline is a particularly complex challenge given the ubiquitous nature of this product and its social and economic importance.

In the effort to safeguard the public health and welfare, environmental programs are targeted to benefit those persons and ecosystems most likely to be adversely affected by pollution. Control programs are then designed to reduce exposures and impacts to levels which are protective of those most sensitive individuals and environments. Providing protection to these sensitive populations and places helps to ensure that risks to general health and welfare are minimized.

Because the goals of health and environmental protection must comport with other social and economic goals, compromise solutions are the norm rather than the exception. Gasoline is a case in point. This product contains hundreds of toxic chemicals and creates many other toxic pollutants when combusted. Many of these pollutants are a direct threat to public health, others contribute to smog, fine particle pollution, acid deposition and climate change problems which have a variety of adverse health and welfare effects. From a narrow public health and environmental protection perspective, the optimal solution would be to take this product off the market. When social and economic concerns are brought to bear, however, this option is not deemed to be in the best overall interest of society.

The search for a compromise solution has led to strategies aimed at reformulating gasoline to improve its emission characteristics in ways which minimize public health risks. Several distinct efforts with related but different goals have been initiated over the past twenty-five years. The first, initiated in the mid-1970s, involved taking the lead out of gasoline to protect against serious health effects associated with exposure to this additive and to permit the use of catalytic converters capable of dramatically reducing many of the harmful constituents of automobile exhaust. This was followed by regulations in the 1980s aimed at lowering gasoline volatility and thereby reducing smog-forming pollutants. Oxygenated fuels were mandated in some areas to reduce carbon monoxide levels in urban areas. The reformulated
gasoline (RFG) program began in 1995 as a strategy to further reduce smog-related pollutants and toxic emissions from gasoline vapors and automobile exhaust.

When designing strategies such as the RFG program, it is critical to ensure that the formulation changes do not result in the transfer of risks from one population to another or increasing one risk while reducing another. For example, the mandatory addition of the oxygenate, primarily in the form of methyl-tertiary butyl ether (MTBE), to gasoline in areas of high carbon monoxide and ozone pollution has raised concerns among some that the “cleaner” gasoline was causing more of a risk than the product it replaced.

The RFG program requires a reduction in the aggregate mass of emissions of the five listed toxic compounds. While refiners have generally overcomplied with the Phase I RFG toxic reduction requirements, some of the regulated toxic constituents increase, some decrease and others remain relatively unchanged as a result of gasoline reformulation strategies. Since these toxic compounds vary significantly in their potential to harm exposed populations, simply comparing the change in mass emissions of these toxics is an inadequate measure of the risk reduction potential of this public health strategy.

This study is intended to assess the relative risk of conventional and reformulated gasoline blends in order to provide better information about the merits of reformulated gasoline as a long-term public health protection option. In this case, we rely on air quality data on ambient levels of ozone to conclude that RFG is an effective smog reduction strategy. Since ozone smog adversely affects the public health and welfare, the RFG program represents an effective risk reduction strategy. Questions remain, however, regarding the extent to which the toxic emission reduction element of this program are achieving the intended goal. This current assessment attempts to shed light on this issue through a comparison of the relative cancer risk posed by exposure to the toxic constituents of gasoline vapors and automobile exhaust.

While there are many hundreds of known and suspected toxic compounds associated with gasoline, this study relies on an assessment of the subset of five toxic air pollutants identified by Congress in the Clean Air Act as needing to be reduced in aggregate. We have also included MTBE in this assessment given the public concern expressed in some areas regarding its potential adverse health effects. While most of these six pollutants present both acute (short-term) and chronic (long-term) health risks, this study assesses only the carcinogenic risk associated with these pollutants. This quantitative assessment of relative risk accounts for both the comparative level of emissions and the capacity of each component of the mixture to produce injury.
We believe that carcinogenicity is the appropriate toxicity endpoint to be used in this comparison due to several factors. First, specific concerns have been raised about the potential carcinogenicity of emissions of gasoline containing MTBE. Second, it is well understood that several components of conventional gasoline are known carcinogens. Finally, since the carcinogenic compounds of interest and emissions factors for these compounds are quantifiable, a reasonable toxicity comparison can be made.
II. Regulatory Context

A. Clean Air Act Amendments of 1990

Section 211(k)(1) of the Clean Air Act Amendments of 1990 (CAA or the Act) established the reformulated gasoline program. The program is intended to improve air quality by requiring that gasoline be reformulated to reduce motor vehicle emissions of toxic and ozone-forming compounds. This section of the Act mandates the sale of reformulated gasoline in nine large metropolitan areas designated as nonattainment of the ozone NAAQS. The Act allows other ozone nonattainment areas to voluntarily opt into the program. It also provides insurance that conventional gasoline sold in the rest of the country does not become more polluting. This requirement ensures that refiners do not "dump" fuel components that are restricted in reformulated gasoline, and that cause harmful emissions, into conventional gasoline.

In section 211(k)(l), Congress directs EPA to issue regulations that "require the greatest reduction in emissions of ozone-forming and toxic air pollutants ("toxics") achievable through the reformulation of conventional gasoline, taking into consideration the cost of achieving such emission reductions, any non air-quality and other air-quality related health and environmental impacts and energy requirements." The Act establishes a two-phase program: Phase I began on January 1, 1995 and Phase II begins on January 1, 2000. RFG must meet both emission performance standards and compositional specifications.

Section 211(k)(3) specifies that Phase I RFG must reduce emissions of VOCs and toxics by 15 percent (measured on a mass basis) compared to baseline emissions. Baseline emissions are defined as those from 1990 model year vehicles operated on a specified baseline gasoline. CAA compositional specifications for reformulated gasoline include a 2.0 weight percent oxygen minimum and a 1.0 volume percent benzene maximum.

For Phase II of the program, the Act sets VOC and toxics performance standards requiring a reduction of no less than a 25 percent from baseline emissions. EPA was given the discretion to adjust this standard upward or downward taking into account such factors as feasibility and cost, but in no case was it to be less than 20 percent.
B. Regulatory Negotiation

Shortly after passage of the 1990 CAA, EPA entered into a regulatory negotiation (Reg-Neg) with interested parties to develop specific proposals for implementing the reformulated gasoline program and the related anti-dumping requirements. In a negotiated rulemaking, a Reg-Neg committee composed of affected parties, attempts to develop a proposed rule acceptable to all parties. If consensus is reached on a proposed rule, it is published as a Notice of Proposed Rulemaking (NPRM). The committee members and the entities they represent agree to support the proposal and not to seek judicial review of the final rule if it has the same substance and effect as the consensus proposal. The RFG Reg-Neg Committee included representatives of the oil and automobile industries, vehicle owners, state air pollution control officials, oxygenate suppliers, gasoline retailers, environmental organizations, and citizens' groups.

In August 1991 the committee reached consensus on a program outline and signed an "Agreement in Principle" describing that consensus. According to the regulatory negotiation agreement, EPA agreed to propose a two-step approach to Phase I reformulated gasoline. The first step took effect in 1995 and utilizes the "Simple Model" to certify that a gasoline meets applicable standards. The Simple Model allows certification based on a fuel's oxygen, benzene, heavy metal and aromatics content and Reid Vapor Pressure (RVP).

Under the second step, EPA would propose a "Complex Model" to supplant the Simple Model for certifying compliance with these standards. The Complex Model provides a more accurate assessment of the emissions associated with different formulations because it includes several fuel parameters not incorporated in the Simple Model. The Complex Model became effective at the beginning of 1998 during Phase I of the RFG program, and will be used to certify gasoline in Phase II starting in 2000.

C. Rulemakings

The first Notice of Proposed Rulemaking (NPRM) for the reformulated gasoline program was published on July 9, 1991, prior to the conclusion on the regulatory negotiations. An NPRM is normally published at the conclusion of the advisory committee negotiations. In this case, however, EPA deemed that although consensus of the members on an acceptable rule was possible, an NPRM was required before that time to meet the statutory deadline.
The 1991 NPRM described the provisions of both a program to require the sale of gasoline which reduces emissions of toxics and ozone-forming volatile organic compounds (VOCs) in certain nonattainment areas and a program to prohibit the gasoline sold in the rest of the country from becoming more polluting. The 1991 notice described the outline of the reformulated gasoline program as required by statutory provisions and options that the regulatory negotiation committee members were considering. Topics included in the 1991 proposal consisted of the derivation of the emission standards, fuel certification by modeling, opt-in provisions, credits, anti-dumping requirements, and enforcement provisions for all aspects of the reformulated gasoline program.

EPA issued a Supplemental Notice of Proposed Rulemaking (SNPRM) on April 16, 1992 which reflected the agreement reached in the regulatory negotiation. The SNPRM described the standards and enforcement scheme for both reformulated and conventional gasoline. It also included specific proposals for the simple emission model to be used in gasoline certification and enforcement for the 1995 to 1997 period.

Also included in the February 26, 1993 NPRM were the proposed Complex Model for certification of reformulated gasoline and the proposed Phase II performance standards. The Complex Model took effect January 1, 1998. The Complex Model provides a method of certification based on the fuel characteristics such as oxygen, benzene, aromatics, RVP, sulfur, olefins and the percent of fuel evaporated at 200 and 300 degrees Fahrenheit (E200 and E300, respectively).

The NPRM also proposed Phase II standards for reformulated gasoline which are to take effect on January 1, 2000, as prescribed by section 211(k)(3) of the CAA. The proposed VOC performance standard was a 30-35 percent reduction for Class B areas and a 20-32 percent for Class C areas. EPA proposed to set the toxic standard at 20 or 25 percent reduction since additional toxics control was not found to be cost effective and, in most cases, these greater toxics reductions were expected to occur through fuel reformulation for VOC control purposes. The NPRM also included proposed NOx performance standards of a 6.5 to 7.5 percent reduction for summertime (VOC-controlled) RFG and no NOx increase for wintertime RFG. While the CAA only specified that no NOx increase could occur through reformulation, EPA proposed the NOx performance standard — under the authority of section 211(c)(1) — since additional NOx control was deemed beneficial and cost effective in reducing ambient ozone levels.

The final rule was promulgated on February 16, 1994. Major provisions of the final rule include the performance standards for VOCs, NOx and toxics, the complex model, and anti-dumping and enforcement provisions. The essential performance requirements on an averaged basis are provided in the
table below. There are also standards for compliance on a per gallons basis, but petroleum refiners have typically chosen to use the averaged standards. The Northeast is a class C area, VOC control region 2.

Table II-1

<table>
<thead>
<tr>
<th></th>
<th>VOC</th>
<th>NOx</th>
<th>Toxics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase I Simple Model (1995-1997)</td>
<td>No performance standards, but RVP, oxygen and benzene fuel specifications which achieve reductions.</td>
<td></td>
<td>16.5%</td>
</tr>
<tr>
<td>Phase I, Complex Model (1997-1999)</td>
<td>17.1% (class C)</td>
<td>1.5%</td>
<td>16.5%</td>
</tr>
<tr>
<td>Phase II, Complex Model (2000+)</td>
<td>27.4% (class C)</td>
<td>6.8% (summer) 1.5% (winter)</td>
<td>21.5%</td>
</tr>
</tbody>
</table>

D. Program Opt-In Rules

In an effort to address interests expressed by state and local air quality control officials, the environmental community and others, on March 28, 1997 EPA published a proposal in the Federal Register to expand the opportunity for states to opt into the RFG program. This rule originally allowed any area classified as an ozone nonattainment area to participate in the RFG program on petition by the Governor of the State in which the area is located. This proposal expanded the opt in provision by allowing states to include in the RFG program any areas previously classified as ozone nonattainment, but which had subsequently redesignated to attainment. The goal of this provision is to provide additional options to help ensure that these areas continue to achieve and maintain compliance with the ozone standard.
E. Phase II Opt-Out Rules

In October, 1997, EPA issued rules governing the procedures for states to opt areas out of the RFG program. The primary purpose of the opt out rule was to establish a greater degree of regulatory certainty as it relates to demand for reformulated gasoline. Greater certainty was expected to encourage capital investment in refinery equipment needed for manufacturing RFG Phase II and provide a greater probability that refiners would be able to recover that investment. To that end, states will be required to decide whether their voluntary opt in areas will participate in the Phase II program. The final rule mandated the Governor of each state with voluntary opt in areas to submit an opt out petition by December 31, 1997 or remain in the Federal RFG program until December 31, 2003. The state of Maine was granted an extension to the opt-out deadline.

The program termination date for those states opting out of the program after December 31, 1997 will be January 1, 2004, or 90 days after the Agency approves a revision to the state plan for removing RFG as a control, or 90 days from the date EPA notifies the state that the petition has been approved, whichever date is applicable. The opt out procedures revert back to the previously-published rule's 90 day requirement as of January 1, 2004. Any area redesignated as ozone attainment between 1998 and 2004 may petition to opt out of the RFG program, regardless of any prior commitment to remain in the program. The new opt out rules also cover those states that opt into the program in the interim period between December 31, 1997 and December 31, 2003.
III. Fuel Quality

A. Overview

This study uses gasoline fuel quality survey data from 1996 to characterize average conventional gasoline and Phase I RFG sold in the Northeast. This information and predictions as to the strategies petroleum refiners will use to comply with the pending requirements of the program served as the basis for estimating the composition of Phase II RFG.

Petroleum refiners will adjust their current RFG blends and refining processes to comply with two significant regulatory changes scheduled between 1996 and 2000. In 1998, EPA has upgraded its fuel certification procedure by requiring refiners to use the Complex Model, rather than the Simple Model, to demonstrate the compliance of RFG blends. The Complex Model includes gasoline fuel properties not accounted for in the Simple Model and adds a Phase I NOx performance standard. In 2000, the more stringent Phase II RFG requirements take effect, including an additional 5 percent reduction in toxic emissions, 10 percent reduction in VOC emissions, and 5 percent reduction in NOx emissions compared to Phase I levels.

Given the uncertainty associated with predicting the composition of Phase II RFG, a two step process was used in this study to determine the properties of this fuel. This assessment was conducted to provide Northeast decision makers with information about the magnitude of the toxic benefits already achieved with Phase I RFG, and to estimate the types of changes necessary to achieve Phase II RFG compliance in the year 2000. The first step involved: (1) calculating how close average 1996 RFG fuel formulations were to meeting the Phase II RFG performance standards; (2) analyzing the fuel properties adjustments needed to achieve the additional necessary reductions; and (3) projecting the range of formulations which may be used to produce average Phase II RFG. In conducting this assessment, NESCAUM relied on gasoline survey data and Complex Model analyses of the relationship between reductions in various gasoline fuel properties and emissions rates of VOCs and NOx.

The second step relied on DOE’s prediction of how petroleum refineries in Petroleum Allocation for Defense Districts (PADD) I and III, and foreign refineries will comply with the NOx and VOC performance standards for Phase II RFG. DOE’s analysis used refinery models developed specifically for each PADD, and reflects the unique characteristics of these refineries and their gasoline feedstocks. As there is no available model designed specifically for foreign refineries, our Base Case analysis assumes that foreign refineries will use their current Phase I RFG formulation with a reduction in RVP to achieve the necessary VOC controls.
In addition to the Base Case, three additional analyses were performed to explore the effect of changes in three gasoline fuel properties — sulfur, olefins, and aromatics — on toxic emissions. Changes in the content of these three gasoline fuel properties are correlated to changes in NOx and toxic emissions. These analyses were performed for PADDs I and III and Imports.

B. Estimating Average Fuel Quality Parameters for Gasoline Blends Sold in the Northeast

While the quality of individual batches of fuel may vary considerably, for the purposes of this assessment, it was necessary to estimate regional average parameters for conventional gasoline, Phase I RFG and Phase II RFG sold in the Northeastern U.S. Industry survey data were used to estimate the average quality of currently marketed conventional and RFG blends of gasoline.

The U.S. is geographically divided into petroleum area defense districts (PADDs). The average quality of gasoline produced in these different regions tends to vary somewhat due to differences in the age and design of the refineries operating in the different PADDs and the use of different quality crudes in the refining process. Foreign refineries have yet a different mix of refinery technologies and sources of crude oil. The Northeast retail market offers gasoline refined in PADD I (East Coast), PADD III (Gulf Coast), and at foreign facilities. Survey data indicate that 40 percent of the gasoline sold in the Northeast comes from PADD I refineries, 40 percent from PADD III refineries and the remaining 20 percent from foreign refineries.

Nearly all of the PADD I and foreign refineries produce both conventional and reformulated gasoline. In PADD III, however, less than half of the refineries currently produce RFG. The EPA database for 1996 gasoline quality was used to estimate the volume-weighted average qualities for PADD I CG and RFG and for imported CG and RFG. Summer data from the 1996 API survey were used to estimate volume-weighted qualities for PADD III CG and RFG. This analysis accounts only for that subset of refineries producing both CG and RFG. These data sets allow the most direct comparison of the relative qualities of RFG and CG from the three areas supplying gasoline to the Northeast. The average fuel properties for conventional and reformulated gasoline blends from each petroleum supply region are presented in Tables III-1, III-2 and III-3 below.
Table III-1

Average Fuel Composition for Gasoline from RFG-Producing Refineries PADD I

<table>
<thead>
<tr>
<th>Fuel Parameters</th>
<th>Conventional Gasoline</th>
<th>Phase I RFG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfur, ppm</td>
<td>382</td>
<td>266</td>
</tr>
<tr>
<td>Olefins, %vol.</td>
<td>16</td>
<td>15.5</td>
</tr>
<tr>
<td>Benzene, %vol.</td>
<td>1.1</td>
<td>0.61</td>
</tr>
<tr>
<td>Oxygen (MTBE), %wt.</td>
<td>0</td>
<td>2.1</td>
</tr>
<tr>
<td>RVP, psi</td>
<td>8.7</td>
<td>7.9</td>
</tr>
<tr>
<td>Aromatics, %vol.</td>
<td>23.8</td>
<td>17.50</td>
</tr>
<tr>
<td>E200</td>
<td>47</td>
<td>50.4</td>
</tr>
<tr>
<td>E300</td>
<td>77.5</td>
<td>77.5</td>
</tr>
</tbody>
</table>

Table III-2

Average Fuel Composition for Gasoline From RFG-Producing Refineries PADD III

<table>
<thead>
<tr>
<th>Fuel Parameters</th>
<th>Conventional Gasoline</th>
<th>Phase I RFG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfur, ppm</td>
<td>490</td>
<td>347</td>
</tr>
<tr>
<td>Olefins, %vol.</td>
<td>16.0</td>
<td>13.8</td>
</tr>
<tr>
<td>Benzene, %vol.</td>
<td>.88</td>
<td>.68</td>
</tr>
<tr>
<td>Oxygen (MTBE), %wt.</td>
<td>.05</td>
<td>1.89</td>
</tr>
<tr>
<td>RVP, psi</td>
<td>8.2</td>
<td>7.2</td>
</tr>
<tr>
<td>Aromatics, %vol.</td>
<td>26.6</td>
<td>21.3</td>
</tr>
<tr>
<td>E200</td>
<td>48.6</td>
<td>51.1</td>
</tr>
<tr>
<td>E300</td>
<td>77.2</td>
<td>79.70</td>
</tr>
</tbody>
</table>
Table III-3

Average Gasoline Composition Foreign Refineries

<table>
<thead>
<tr>
<th>Fuel Parameters</th>
<th>Conventional Gasoline</th>
<th>Phase I RFG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfur, ppm</td>
<td>303</td>
<td>192</td>
</tr>
<tr>
<td>Olefins, %vol.</td>
<td>12.6</td>
<td>7.7</td>
</tr>
<tr>
<td>Benzene, %vol.</td>
<td>.98</td>
<td>.64</td>
</tr>
<tr>
<td>Oxygen (MTBE), %wt.</td>
<td>0.04</td>
<td>2.1</td>
</tr>
<tr>
<td>RVP, psi</td>
<td>8.7</td>
<td>7.9</td>
</tr>
<tr>
<td>Aromatics, %vol.</td>
<td>23.7</td>
<td>20.70</td>
</tr>
<tr>
<td>E200</td>
<td>47.00</td>
<td>51.10</td>
</tr>
<tr>
<td>E300</td>
<td>77.50</td>
<td>79.70</td>
</tr>
</tbody>
</table>

As shown in Tables III-1, III-2 and III-3, the average quality and composition of Phase I RFG differs significantly among PADD I, PADD III and foreign refiners. The most significant differences in fuel composition from these different sets of refineries are the relative levels of sulfur, olefins and aromatics, the primary determinants of NOx and toxics emissions.

C. Predicting Phase II RFG Fuel Quality

This section describes the first step used by NESCAUM to predict the range of average characteristics of Phase II RFG. It involves calculating how close 1996 RFG fuel formulations were to meeting the Phase II RFG emission performance standards. Once the necessary additional reductions needed for Phase II compliance are quantified, the range of options available to petroleum refiners are identified. This analysis is intended to predict changes in gasoline composition associated with Phase II RFG requirements, with the goal of quantifying how typical reformulation strategies will affect toxic emissions.

C.1 Comparison of 1996 RFG with Phase II RFG Performance Standards

The 1996 average fuel formulation estimates were used as a basis for predicting how refiners will modify RFG formulations to comply with the Phase II performance standards. The average fuel composition information for each of the three refinery groups were run through the Complex Model to assess the current emission reductions — compared to the CAA baseline —
achieved by these fuels. These model results were compared to the Phase II performance standards to estimate the additional reductions needed for year 2000 compliance.

C.1.1 Comparison of 1996 RFG with Phase II Toxics Performance Standard

The 1996 RFG supplied to the Northeast typically achieves the 21.5 percent toxics reduction requirement for Phase II RFG — primarily through benzene reductions.\(^7\) Although no additional reductions of toxic air pollutants are needed from either PADD to comply with the Phase II standards in 2000, NOx and VOC requirements will require fuel parameter adjustments which will also affect toxic emissions. Table III-4 depicts the compliance status of regular and premium grade RFG from each petroleum supply region with Phase II RFG toxics requirements.

<table>
<thead>
<tr>
<th>Entire PADD Production</th>
<th>2000 Standard with Annual Averaging</th>
<th>Regular Grade (75%)</th>
<th>Premium Grade (25%)</th>
<th>Total (%)</th>
<th>Change Needed for 2000 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PADD 1</td>
<td>&gt;= -21.5%</td>
<td>-30.77</td>
<td>-29.24</td>
<td>-30.4</td>
<td>None (-8.9)</td>
</tr>
<tr>
<td>PADD 3</td>
<td>&gt;= -21.5%</td>
<td>-27.54</td>
<td>-27.30</td>
<td>-27.5</td>
<td>None (-6.0)</td>
</tr>
<tr>
<td>imports</td>
<td>&gt;= -21.5%</td>
<td>-32.16</td>
<td>-35.74</td>
<td>-33.1</td>
<td>None (-11.6)</td>
</tr>
</tbody>
</table>

C.1.2 Comparison of 1996 RFG with Phase II VOC Performance Standard

The NESCAUM region is within VOC control region 2, but the fuel characteristics for PADD I and especially PADD III are for RFG produced in the entire PADD for VOC control regions 1 and 2. This is deliberate since there is no guarantee that distribution patterns will remain consistent from year to year.

Fuel formulations for premium grade gasoline from PADD I and III were from the API refinery survey for summer, 1996 (May 1 to August 31, 1996). Fuel formulations for premium grade gasoline from imports were generated by EPA. The regular grade fuel characteristics are from the EPA database for the entire year, while the premium grade gasoline characteristics were from API's survey because this data was not requested from EPA. EPA's figures were chosen because they represent the entire year, and because for PADD I, API's survey only accounted for half the gasoline by volume.
The Phase II RFG performance standard will require an averaged 27.6% reduction in summertime VOC emissions from the CAA baseline. As shown in Table III-5 below, most domestic and foreign RFG refiners will need to further reduce emissions of VOCs to meet the Phase II standard. On average, PADD I refiners will need to achieve an additional 11.5 percent VOC reduction, PADD III refiners an additional 7.5 percent reduction from the levels achieved in 1996 RFG, and foreign refineries will need an additional 10.9 percent VOC reduction.

Table III-5

Average VOC Emissions from 1996 Summer Gasoline Compared with Phase II Performance Standards

<table>
<thead>
<tr>
<th>Entire PADD Production</th>
<th>2000 Standard with Annual Averaging</th>
<th>Regular Grade (75%)</th>
<th>Premium Grade (25%)</th>
<th>Total (%)</th>
<th>Change Needed for 2000 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PADD 1</td>
<td>&gt;= -27.6</td>
<td>-16.87</td>
<td>-13.70</td>
<td>-16.1</td>
<td>+11.5</td>
</tr>
<tr>
<td>PADD 3</td>
<td>&gt;= -27.6</td>
<td>-22.29</td>
<td>-13.44</td>
<td>-20.1</td>
<td>+7.5</td>
</tr>
<tr>
<td>Imports</td>
<td>&gt;= -27.6</td>
<td>-16.46</td>
<td>-17.57</td>
<td>-16.7</td>
<td>+10.9</td>
</tr>
</tbody>
</table>

C.1.3 Comparison of 1996 RFG to Phase II NOx Performance Standard

The Phase II performance standard requires an average 6.8 percent reduction in NOx emissions in the summertime from the 1990 CAA baseline. On average, PADD I and PADD III refiners need an additional NOx reduction of about 5 percent from Phase I RFG levels, while foreign refineries are already in compliance with the Phase II NOx standard. According to this analysis, most RFG sold in the Northeast in 1996 achieved the 1.6 percent NOx reduction requirement from the CAA baseline which will take effect with the advent of the Complex Model in 1998.
Table III-6
Average NOx Emissions from 1996 Summer Gasoline Compared With Phase II Performance Standards

<table>
<thead>
<tr>
<th>Entire PADD Production</th>
<th>Phase II Standards</th>
<th>Regular Grade (75%)</th>
<th>Premium Grade (25%)</th>
<th>Total Change</th>
<th>Change Needed for 2000</th>
</tr>
</thead>
<tbody>
<tr>
<td>PADD 1</td>
<td>&gt;= -6.8</td>
<td>-1.27</td>
<td>-1.9</td>
<td>-1.4</td>
<td>+5.4</td>
</tr>
<tr>
<td>PADD 3</td>
<td>&gt;= -6.8</td>
<td>+0.9</td>
<td>-5.2</td>
<td>-1.9</td>
<td>+4.9</td>
</tr>
<tr>
<td>Imports</td>
<td>&gt;= -6.8</td>
<td>-6.9</td>
<td>-11.5</td>
<td>-8.1</td>
<td>None (-1.2)</td>
</tr>
</tbody>
</table>

In sum, RFG produced in 1996 in PADD I must on average achieve an additional 11.5 percent VOC reduction and a 5.4 percent NOx reduction to comply with Phase II RFG requirements. PADD III refineries must reduce VOCs by 7.5 percent and NOx by 4.8 percent. Foreign refineries must reduce VOC emissions by 10.9 percent and have already achieved the Phase II performance standard for NOx.

C.2 Compliance Options

In evaluating the range of options available to petroleum refineries as they respond to the challenges of meeting the additional VOC and NOx reductions estimated above, NESCAUM utilized data from the Complex Model to correlate the impact of changes in gasoline fuel properties to changes in emissions of VOCs and NOx. Table III-7 and Table III-8 below show the effect of incremental fuel parameter changes on automobile emissions of VOCs and NOx.

C.2.1 VOC Reduction Strategies

In order to comply with the 15 percent VOC performance standard for Phase I RFG, most refineries in PADD I and foreign refineries have lowered RVP to 7.9 psi, while PADD 3 refineries have lowered RVP for regular grade RFG to 7.2 psi and premium grade to 7.9 psi. As previously stated, on average, PADD I RFG refiners will need an additional 11.5 percent reduction in VOC emissions, PADD III refiners an additional 7.5 percent reduction, and foreign refineries an additional 10.9 percent reduction to achieve the Phase II VOC performance standard in 2000. As shown in Table III-7, reducing RVP control from a 7.9 psi to 6.5 psi achieves an 11.6 percent VOC reduction. Reducing RVP from 7.2 psi to 6.5 psi achieves a more modest reduction of 4.8 percent.
Table III-7
VOCs Reductions From Various Decreases in RVP Calculated Using the Complex Model

<table>
<thead>
<tr>
<th>RVP Level (psi)</th>
<th>VOC Emissions Reductions from CAA Baseline</th>
<th>Change in Reductions from Previous RVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.9</td>
<td>-19.23%</td>
<td>--</td>
</tr>
<tr>
<td>7.2</td>
<td>-25.99%</td>
<td>-6.76%</td>
</tr>
<tr>
<td>6.5</td>
<td>-30.81%</td>
<td>-11.57%</td>
</tr>
</tbody>
</table>

Table III-7 shows that PADD I and Imports will be able to comply with the Phase II RFG VOC performance standard solely through a reduction of RVP to 6.5 psi. PADD III refineries will fall short by about 2.5 percent of reaching the needed VOC reductions with an RVP reduction to 6.5 psi. However, this analysis only accounts for regular gasoline RVP. Since premium grade RFG produced in PADD III has 7.9 psi RVP, the additional VOC reductions achieved from premium gasoline will bridge the shortfall. In addition, analysis performed with the Complex Model confirms that Phase II RFG blends with RVP of 6.5 psi will comply with the Phase II VOC standard.

Given current RVP levels and the predicted benefits from further lowering this parameter, it appears possible that many refineries will be able to achieve the Phase II RFG VOC requirements with further RVP reductions alone. RVP reductions are one of the most cost-effective options for reducing VOCs. Consequently, for the purposes of this study we assume that refineries will opt for RVP control to achieve the needed VOC reductions for Phase II compliance.

C2.2 NOx Reduction Strategies

Most domestic refineries will need to achieve significant additional reductions to comply with Phase II NOx standards. On average, PADD I refineries will need slightly more than a 5.4 percent reduction while PADD III refineries will need to lower NOx emissions by about 4.8 percent. On average, foreign refineries already achieve the Phase II NOx performance standard.

NOx reductions are likely to be achieved through sulfur, olefin or aromatic adjustments. In general, refineries are expected to achieve the bulk of NOx reductions through de-sulfurization, with some additional reductions in
olefins or aromatics. Individual refiners may choose to focus more heavily on reducing olefins and aromatics due to particular refinery configurations.

Table III-8

The Impact of Various Levels of Sulfur, Olefins and Aromatics on Emissions

<table>
<thead>
<tr>
<th>Case</th>
<th>Sulfur, ppm</th>
<th>Olefins, %vol.</th>
<th>Aromatics, %vol.</th>
<th>RVP, psi</th>
<th>NOx</th>
<th>Toxics</th>
<th>VOCs</th>
</tr>
</thead>
<tbody>
<tr>
<td>National Average 1996 RFG</td>
<td>314</td>
<td>12.9</td>
<td>22.9</td>
<td>7.6</td>
<td>-0.3</td>
<td>-27.7</td>
<td>-19.1</td>
</tr>
<tr>
<td>Sulfur</td>
<td>164</td>
<td>12.9</td>
<td>22.9</td>
<td>6.5</td>
<td>-5.6</td>
<td>-31.2</td>
<td>-29.3</td>
</tr>
<tr>
<td>Ole./ Sulfur</td>
<td>114</td>
<td>12.9</td>
<td>22.9</td>
<td>6.5</td>
<td>-7.65</td>
<td>-32.2</td>
<td>-29.8</td>
</tr>
<tr>
<td>Ole. / Sulfur</td>
<td>214</td>
<td>8.9</td>
<td>20.0</td>
<td>6.5</td>
<td>-6.6</td>
<td>-32.4</td>
<td>-28.8</td>
</tr>
<tr>
<td>Ole. / Sulfur</td>
<td>164</td>
<td>8.9</td>
<td>20.0</td>
<td>6.5</td>
<td>-8.4</td>
<td>-33.3</td>
<td>-29.3</td>
</tr>
<tr>
<td>Sulfur / Aro.</td>
<td>114</td>
<td>8.9</td>
<td>15.0</td>
<td>6.5</td>
<td>-10.4</td>
<td>-34.2</td>
<td>-29.8</td>
</tr>
<tr>
<td>S/O/A</td>
<td>214</td>
<td>12.9</td>
<td>15.0</td>
<td>6.5</td>
<td>-8.8</td>
<td>-34.6</td>
<td>-31.0</td>
</tr>
<tr>
<td>S/O/A</td>
<td>164</td>
<td>8.9</td>
<td>20.0</td>
<td>6.5</td>
<td>-6.5</td>
<td>-32.7</td>
<td>-29.9</td>
</tr>
<tr>
<td>S/O/A</td>
<td>114</td>
<td>12.9</td>
<td>15.0</td>
<td>6.5</td>
<td>-10.4</td>
<td>-35.6</td>
<td>-31.5</td>
</tr>
</tbody>
</table>

An infinite number of Phase II RFG complying fuel formulations exist. Table III-8 sketches the rough boundaries of the ranges of sulfur, olefin and aromatic levels which might be found in complying Phase II RFG. Sulfur levels in Phase II RFG range from 214 to 114, olefin levels range from 12.9 to 8.9, and aromatic levels range from 20.0 to 15.0. For VOC reductions, this analysis assumes a RVP of 6.5 psi. These parameters establish the range within which complying Phase II RFG are likely to fall for RFG produced for sale in the Northeast.

D Estimates of Phase II RFG Fuel Quality

Individual petroleum refineries decide on the best approach for producing compliant blends of RFG based on the age and design of the refinery, the quality of the crude oil used to make gasoline, and cost considerations. Given the large number of refineries producing RFG and the wide variability in plant designs, the quality of the crude oil used in refining gasoline, cost considerations and corporate goals, individual refineries may
achieve compliant fuels through any number of strategies. Since Phase II RFG is not currently produced, it is not possible at this time to determine the precise strategies refiners will employ to produce complying fuel formulations. However, our analysis from the previous section indicates that Phase II RFG is likely to have an RVP of 6.5 and that refineries will adjust the content of sulfur, olefins and aromatics in order to achieve the required NOx reductions.

The Base Case scenario was generated from an Oak Ridge National Laboratory (ORNL) modeling run designed to determine the most likely refinery response to the Phase II NOx performance standard within each PADD. This analysis was performed in response to API's petition in opposition to the NOx performance standards for Phase II RFG. The Oak Ridge modeling analysis is adopted for this study because of the strong correlation between gasoline sulfur levels and NOx emissions, and the recognition that the need for NOx reductions will drive most of the formulation changes in Phase II RFG.

Table III-9

Phase II RFG Fuel Properties

<table>
<thead>
<tr>
<th>Properties</th>
<th>National RFG Phase I, 1996 (API)</th>
<th>PADD 1</th>
<th>PADD 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfur, ppm</td>
<td>314</td>
<td>132</td>
<td>127</td>
</tr>
<tr>
<td>Olefins, %vol.</td>
<td>12.9</td>
<td>12.4</td>
<td>9.0</td>
</tr>
<tr>
<td>Benzene, %vol.</td>
<td>0.7</td>
<td>0.7</td>
<td>0.7</td>
</tr>
<tr>
<td>Oxygen (MTBE), wt %</td>
<td>2.19</td>
<td>2.19</td>
<td>2.19</td>
</tr>
<tr>
<td>RVP, psi</td>
<td>7.6</td>
<td>6.5</td>
<td>6.5</td>
</tr>
<tr>
<td>Aromatics, vol. %</td>
<td>22.9</td>
<td>25.0</td>
<td>25.0</td>
</tr>
<tr>
<td>E200 %</td>
<td>49.4</td>
<td>55.0</td>
<td>59.7</td>
</tr>
<tr>
<td>E300 %</td>
<td>82.5</td>
<td>87.0</td>
<td>86.2</td>
</tr>
</tbody>
</table>

Three sensitivity runs were completed to explore the impact of high sulfur, high olefins and high aromatics on toxic emissions. These runs were variations on a national 1996 RFG average gasoline fuel properties and, unlike the Base Case, are not PADD-specific.
### Table III-10

**Other Property Variations**

<table>
<thead>
<tr>
<th>Properties</th>
<th>National RFG Phase I, 1996</th>
<th>High Sulfur</th>
<th>High Olefins</th>
<th>High Aromatics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfur, ppm</td>
<td>314</td>
<td>184</td>
<td>111</td>
<td>92</td>
</tr>
<tr>
<td>Olefins, %vol.</td>
<td>12.9</td>
<td>9.2</td>
<td>14.5</td>
<td>12.9</td>
</tr>
<tr>
<td>Benzene, %vol.</td>
<td>0.7</td>
<td>0.7</td>
<td>0.7</td>
<td>0.7</td>
</tr>
<tr>
<td>Oxygen (MTBE), wt %</td>
<td>2.19</td>
<td>2.19</td>
<td>2.19</td>
<td>2.19</td>
</tr>
<tr>
<td>RVP, psi</td>
<td>7.6</td>
<td>6.5</td>
<td>6.5</td>
<td>6.5</td>
</tr>
<tr>
<td>Aromatics, vol. %</td>
<td>22.9</td>
<td>22.9</td>
<td>22.9</td>
<td>33.2</td>
</tr>
<tr>
<td>E200 %</td>
<td>49.4</td>
<td>57.4</td>
<td>57.4</td>
<td>57.4</td>
</tr>
<tr>
<td>E300 %</td>
<td>82.5</td>
<td>86.6</td>
<td>86.6</td>
<td>86.6</td>
</tr>
</tbody>
</table>

The High Sulfur formulation assumes a sulfur level of 184 ppm which is at the top of the likely range. Refineries are expected to compensate for high sulfur with an olefin level of only 9.2 percent by volume. These sulfur levels are still below the levels found in 1996 RFG, which range from 347 ppm (PADD III) to 192 (Imports).

Olefins are created during hydrogen-deficient refining processes and are directly related to automobile emissions of 1,3-butadiene, one of the most toxic constituents found in vehicle exhaust. The high olefin case assumes an olefin content of 14.5 percent by volume, which is at the top of the modeled range for complying Phase II RFG fuels. Interestingly, it is within the range of olefins levels found in Phase I RFG, which range from 15.5 percent by volume (PADD I) to 7.7 percent by volume (Imports). The high olefin case predicts that refineries will offset the olefin increase with a substantial reduction in sulfur to 111 ppm.

Aromatics are high octane blending components that have a benzene ring in their molecular structure. Common aromatics include benzene, toluene and xylene. In Phase I RFG, aromatics ranged from 21.3 percent by volume (PADD III) to 17.5 percent by volume (PADD I). The high aromatic case assumes an aromatic content of 33.2 percent by volume, which is well above the aromatic levels found in Phase I RFG. Refiners are expected to adjust for the high aromatic level with a sulfur content of 92 ppm, the lowest of the three alternative RFG formulas. These three fuel formulations identify the compliance boundaries of Phase II RFG and demonstrate the tradeoffs refineries are likely to encounter when choosing their compliance path.
E. Summary

Analysis of 1996 gasoline fuel properties for PADDs I and III and Imports into the Northeast, results from the Complex Model, and review of DOE's recommendations for Phase II RFG fuel formulations led to the following conclusions.

- The Phase II RFG toxics performance standard is currently being achieved by most refiners and this standard is not likely to drive further formulation changes.

- On average, PADD I refiners will need to lower VOCs by an additional 11.5 percent; PADD III refiners will need to lower VOCs by an additional 7.5 percent; and foreign refineries by 10.9 percent. These reductions are expected to be achieved primarily through a reduction in RVP to 6.5 psi.

- On average, PADD I and PADD III refiners will need to achieve NOx reductions of about 5 percent from 1996 Phase I levels. Significant reductions in sulfur levels, supplemented by reductions in aromatic and olefin content, are assumed to represent the most likely compliance strategies for these sets of refineries. On average, RFG produced by foreign refineries already meets the Phase II RFG NOx performance standard.

- The Oak Ridge National Laboratory PADD-specific modeling analysis was chosen as the Base Case for this study due to focus on how refineries are most likely to achieve a 6.8 percent reduction in summertime NOx emissions to comply with the performance standard in Phase II RFG.

- Since all predictions are by definition uncertain, this study also examines Phase II RFG fuel formulations with high sulfur, high olefin, and high aromatic content. These three gasoline fuel properties were chosen because of their correlation to both NOx emissions and toxic emissions.
IV. Health Effects and Quantitative Risk Factors

The federal RFG program is intended as both an ozone and toxic air pollutant reduction strategy. As previously mentioned, however, the mass-based composite toxic reduction approach used for RFG compliance is an inexact measure of the likely public health benefits. In an effort to better understand the benefits associated with the toxic reduction component of the RFG program, NESCAUM has employed a risk-weighted approach\(^8\) to compare the relative benefits of conventional and reformulated gasoline blends.

This section presents background information on the cancer and non-cancer effects of the five air toxic compounds regulated under the RFG program: benzene, 1,3-butadiene, formaldehyde, acetaldehyde and POM. Given the concern over the potential adverse health effects associated with the oxygenate MTBE, we have included this pollutant in the comparative risk assessment. This section also presents the cancer unit risk factors and cancer potency ratios used in this assessment and explains their derivation. While most of the six toxic compounds evaluated in this assessment present both acute and chronic risk to exposed individuals, it is important to note that the risk-weighted assessment conducted through this study focuses strictly on carcinogenic effects.

The section begins with a general discussion of the acute effects of these toxic compounds and then moves to a summary of the known or suspected carcinogenic effects of each pollutant. This section also includes some basic information about: the source(s) of each pollutant, its atmospheric residence time, average ambient concentrations, and other (noncancer) chronic health effects.

A. Health Effects and Toxicity

A.1 Acute Effects

Acute effects are those which occur after a single or limited exposure and whose impacts are temporary in nature. Example of acute symptoms are nausea or vomiting, coughing, dizziness, and headache. The primary acute health effects associated with the six toxic compounds evaluated in this study are: (1) irritation of the eyes, nose and throat, and nausea; and/or (2) central

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\(^8\) The risk-weighted approach first determines relative cancer potencies for the six constituents.
nervous system (CNS) effects — dizziness and headaches (Fiedler, N., et al., 1994; Health Effects Institute, 1996).

Some individuals report experiencing acute effects from exposure to RFG including headaches, dizziness and irritation of the eyes and throat. (Health Effects Institute, 1996; Interagency Assessment of Oxygenated Fuels, 1997; Fiedler N., 1994). Since the addition of relatively higher levels of MTBE is among the primary formulation changes associated with RFG, it has been hypothesized that this additive may contribute to such acute effects. Individuals have associated the disagreeable odor of MTBE with nausea and vomiting, and headaches (Health Effects Institute, 1996). Patients receiving short-term, high doses of MTBE for dissolution of gall stones have exhibited clinical symptoms of CNS depression, mild liver abnormalities, dizziness and headache. Some motorists and gas station workers have reported similar subjective symptoms of headache, dizziness, and irritation which were attributed to exposure to gasoline containing MTBE. However, chamber studies with healthy human volunteers exposed to up to 50 ppm MTBE for two hours did not report the health effects described above (Cain, W.S., et al. 1996). Human sensitivity differences, and exposure variables (pure MTBE versus a mixture of gasoline constituents) may be some of the reasons for the response differences reported in the chamber studies.

Given the addition of substantially higher levels of MTBE in reformulated gasoline, it is possible that the combined acute effects are greater with RFG than with conventional gas. Further, the discernible odor associated with ether-based oxygenates may increase sensitivity to the other sensory responses. These factors may result in a larger group of sensitive individuals noticing the acute effects of gasoline exhaust and vapors than in the past. Since reports of acute health effects have tended to be concentrated in a few specific locations and the incidence of complaints has diminished significantly since the early stages of the RFG program, it appears that other factors are also at play. For example, the perception of odors diminish with repeated exposure, which may in part explain the decline in the incidence of reported complaints (Vetrano K., 1993a, Shusterman D., 1992)

**A.2 General Information and Carcinogenic Effects**

**Benzene**

**Background**

Benzene is a highly volatile aromatic hydrocarbon that is added to gasoline as an "anti-knock" agent (ATSDR, 1996a). Gasoline vapors and exhaust emissions from gasoline-powered motor vehicles and nonroad equipment are the primary sources of benzene at most locations. Some
stationary sources also emit benzene. Cigarette smoke is another source of benzene. Inhalation of benzene is the principal route of human exposure.

This pollutant is chemically stable. Atmospheric residence times are estimated to range from as short as one to two days in the summer to as long as ten to twenty days in the winter. Nationally, average ambient concentrations of benzene range from about 4 to 7 micrograms per cubic meter (µg/m³) (ATSDR, 1996a, Landrigan, P.L. 1990).

The primary noncancer effects associated with chronic exposure to benzene are hematotoxic in nature (blood disorders) and target bone marrow tissue (ATSDR, 1996a, ATSDR, 1996b, Benzene toxicity and risk assessment, 1972-1992). Long-term exposure to high levels of benzene can disrupt blood production and decrease the number of red blood cells and platelets. Benzene exposure can also be harmful to the immune system. Animal studies indicate that this pollutant is a developmental and reproductive toxicant. Benzene can also cause chromosomal damage in laboratory animals. The chromosomal aberrations induced by benzene exposure are useful as biomarkers of exposure and as a potential indicator of leukemogenesis.

Carcinogenicity

Benzene is classified by EPA as a Group A, known human carcinogen (ATSDR, 1996a). The International Agency for Research on Cancer (IARC) has also classified this pollutant as a human carcinogen. Chronic workplace exposure to relatively high levels of benzene is generally accepted as a cause of acute myeloid leukemia (AML) in humans. Further, animals exposed to benzene have developed leukemia, lymphomas and other types of tumors. Benzene exposure has also been linked to genetic changes in humans and animals.

A benzene cancer potency estimate has been derived by EPA, OSHA, and American Conference of Governmental Industrial Hygienists (ACGIH) based largely on data from a cohort study of rubber workers (Benzene toxicity and risk assessment, 1972-1992). The present report derived a potency factor for benzene based on rat data and the close agreement between these two estimates is noteworthy.

MTBE

Background

MTBE is a volatile oxygenate that is added to gasoline to enhance octane and decrease CO emissions. The addition of this oxygenate also serves to lower the mass emissions of other toxic compounds such as benzene through a dilution effect. Humans are exposed to MTBE primarily through
the inhalation of gasoline vapors and exhaust emissions. Atmospheric concentrations of MTBE are estimated to be less than 1 ppm and workplace exposure levels are reported to range from 0.1 to 10 ppm (National Science and Technology Council, 1997).

In addition, MTBE has been found in a number of underground drinking water supplies. Drinking MTBE-contaminated water is another potentially significant exposure pathway. Because MTBE is highly water soluble relative to other constituents in gasoline, is poorly adsorbed to soils, and moves with the groundwater flow largely unretarded, this is an area for future research and investigation.

MTBE is not a cumulative toxicant and the chemical is rapidly eliminated from the body through exhalation. No published information is available on the human health impacts of long-term exposure to MTBE. Chronic bioassays have, however, shown that, at high doses, MTBE is carcinogenic to rats and mice (Personal Communication with Hari Rao, Methyl Tertiary Butyl Ether). Experimental studies found that after inhaling high doses of MTBE, some male rats developed kidney and testis tumors; some of the mice developed liver tumors. The gavage of high doses of MTBE was also shown to result in some of the male rats developing testis tumors and some of the female rats developing leukemia and lymphoma (National Science and Technology Council, 1997). The mechanisms by which this toxicant causes cancer in animals is not known.

Neither the Department of Health and Human Services, IARC, nor the EPA have classified MTBE as a known or suspected human carcinogen. Nevertheless, the tumor responses in rats and mice through both inhalation and gavage suggest that MTBE may be carcinogenic to humans. A recent White House Office of Science and Technology Policy (OSTP) report, Interagency Assessment of Oxygenated Fuels, states that, “There is sufficient evidence to indicate that MTBE is an animal carcinogen and to regard MTBE as having a human hazard potential” (National Science and Technology Council, 1997). The federal 1997 cancer risk assessment guidelines substituted the term “human health hazard” for the previous “possible human carcinogen” designation.

The OSTP report looked at all the available data and concluded that, “[a]t the lower concentrations that are experienced by the general population, the limited epidemiological studies and controlled exposure studies conducted to date do not support the contention that MTBE as used in the winter oxygenated fuels program is causing significant increases over background in acute symptoms or illnesses.” At the same time, the OSTP report acknowledged the limits of its own understanding of the health effects of RFG. It stated, “The largely anecdotal reports of acute health symptoms
among some individuals at very low levels of exposure to oxygenated fuels cannot be adequately explained, but cannot be dismissed."

1,3-Butadiene

Background

The pollutant 1, 3-butadiene is a reactive chemical by-product of the incomplete combustion of hydrocarbons in fuels. This pollutant is a flammable gas at room temperature. Exposure to 1, 3-butadiene comes primarily from the inhalation of vehicle exhaust emissions. Butadiene emissions are positively correlated to hydrocarbon emissions. The use of a catalyst on a vehicle reduces these exhaust emissions and serves to mitigate human exposure to this contaminant. Other sources of this pollutant include facilities producing 1,3-butadiene and manufacturing processes such as rubber production.

Since butadiene is highly reactive, it has a short atmospheric lifetime (Health Effects Institute, 1996, Landrigan P.L. 1994). The residence time during daylight hours, under clear conditions in the summer is estimated to be one hour or less. Under wintertime, cloudy sky conditions, however, the atmospheric lifetime can be much longer and significant day-to-day carryover of 1,3-butadiene concentrations are possible. in addition, 1,3-butadiene decomposes in the atmosphere into other toxic compounds.

Epidemiological studies have reported an association between chronic workplace exposure to 1, 3-butadiene and an increased risk of certain cardiovascular diseases, mainly arteriosclerotic heart diseases. An increased rate of emphysema among rubber workers exposed to 1, 3-butadiene has also been reported. Bone marrow damage and gonadal atrophy have been reported in studies on mice following subchronic and chronic exposure (Landrigan P.L. 1990).

Carcinogenicity

The pollutant 1, 3-butadiene is classified by EPA as a Group B-2 probable human carcinogen based on the results of studies which found this pollutant to be carcinogenic in rats and mice but without adequate epidemiological data. Butadiene is metabolized to mutagenic and carcinogenic epoxides in rats, mice, and humans. The DNA-reactive epoxides are thought to play a critical role in 1, 3-butadiene carcinogenicity. Mice are more sensitive to this toxicant than rats, and are affected at lower doses.

The results of epidemiological studies of cancer among workers exposed to 1,3-butadiene have been inconsistent. The utility of the results from these studies are limited due to the concurrent occupational exposure to
other toxic compounds and the lack of data on workplace concentrations of butadiene. Nevertheless, researchers found excess mortalities among the male workers at several rubber tire manufacturing plants including statistically significant increases in deaths due to stomach and prostate cancer, lymphosarcoma, and leukemia.

In February 1998, the EPA’s National Center for Environmental Assessment concluded that 1,3-butadiene was a known carcinogen (National Center for Environmental Assessment, 1998).

**Formaldehyde**

**Background**

Formaldehyde is formed by the incomplete combustion of the hydrocarbons in fuel. It is also formed through secondary processes when automobile VOC emissions are photo-oxidized in the atmosphere. The use of catalytic converters effectively reduces formaldehyde emissions from motor vehicle exhaust. It is not a component of evaporative emissions. Motor vehicle exhaust is thought to be the source of about one-third of ambient formaldehyde. Other key sources include formaldehyde production, resin manufacturing and as a by-product of various combustion processes.

Formaldehyde is a relatively reactive pollutant (ATSDR, 1996a, Casserette and Doull, 1986). Under clear sky summer daytime conditions, the residence time is estimated to be about two to four hours. Winter residence times exceed those of the summer by a factor of ten. Annual average atmospheric concentrations of formaldehyde are 1.71 µg/m³ in those areas where measurements have been taken. EPA has estimated that maximum concentrations of nearly 5.0 µg/m³ are likely at service stations and concentrations as high as 41.8 µg/m³ may be found in parking garages.

There is data suggesting, but not concluding, that formaldehyde can affect immune function. Studies have also found adverse effects on the liver and kidney of experimental animals exposed to high levels of the pollutant.

**Carcinogenicity**

EPA has classified formaldehyde as a Group B-1 probable human carcinogen based on limited evidence in humans and sufficient evidence in animal studies (ATSDR, 1996a). IARC agrees that formaldehyde is probably carcinogenic to humans. The epidemiologic data showed statistically significant association between site-specific respiratory neoplasms and exposure to formaldehyde. An increased incidence of nasal tumors was reported in chronic studies in rats and mice.
Acetaldehyde

Acetaldehyde is a reactive chemical formed by the incomplete combustion of the hydrocarbons in fuel (US EPA, 1991). Inhalation of motor vehicle exhaust is the primary exposure pathway for acetaldehyde. The use of catalysts reduce vehicle emissions of the toxicant at about the same rate as they reduce overall hydrocarbon emissions. This pollutant is not a component of evaporative and refueling emissions. Motor vehicles are estimated to be the source of about 40 percent of the acetaldehyde found in the ambient air. Other important manmade sources include wood combustion and coffee roasting. Acetaldehyde is also released through natural processes related to plant respiration and alcohol fermentation. Motor vehicles also contribute to secondary acetaldehyde formation.

Daytime residence times in the summer are estimated at five hours or less under clear conditions. At night and under cloudy conditions, the summer residence time increases to as much as eighteen hours. Winter, clear sky conditions may result in residence times ranging from 20 to 60 hours. Average ambient levels are around 3.0 µg/m3 in cities with available measurements.

Acute exposure to acetaldehyde is irritating to the eyes, nose, and throat. The nasal cavity is the most severely affected and animal studies have shown concentration dependent degeneration of the olfactory and respiratory epithelium (Casserette and Doull's Toxicology, 3rd Ed. 1986).

Carcinogenicity

Chronic exposure studies in rats have reported the occurrence of two types of nasal tumors: adenocarcinoma from the olfactory epithelium and squamous cell carcinoma from the respiratory epithelium. Laryngeal tumors were observed in hamsters after inhalation exposure. The human carcinogenicity data are limited and considered inadequate to assess the carcinogenicity of acetaldehyde. EPA has classified this chemical as a Group B-2, probable human carcinogen and IARC has classified Acetaldehyde as a Group 2B possible human carcinogen. An epidemiological study of occupationally exposed workers found that the evidence was inadequate to suggest that long-term exposure to this toxicant was associated with an increase in total incidence of cancer.

Polycyclic Organic Matter (POM)

POM is a general term used to describe a complex mixture of polycyclic aromatic compounds including diverse classes of hydrocarbons — polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs, and heterocyclic aromatic compounds. POM are emitted during the combustion of gasoline, diesel.
wood, and plastics. People come in contact with POM through exposure to vehicle exhaust and the emissions from other combustion sources. Cigarette smoke is another source of POM (National Science and Technology Council, 1997).

The residence time for POM under clear sky summer conditions is calculated at about 60 hours. In the winter the residence time essentially doubles to about 120 hours. The particulate fraction has a relatively short atmospheric lifetime ranging from 0.5 to 4.0 hours under rainy conditions.

Carcinogenicity

PAHs associated with particulate phase emissions appear to account for most, if not all the carcinogenic activity of POM. Although benzo(a)pyrene (BaP) has been used as a surrogate for PAHs, BaP alone does not appear to be a good marker for carcinogenicity of POM. Since many other carcinogenic chemicals are present in complex POM mixtures, the extractable organic matter from respirable particles has been used as a surrogate exposure measure (ATSDR, 1996a, Meek M.E., et al. 1994).

Because human exposure to POM emissions occurs as the whole complex mixture containing hundreds of carcinogenic and non-carcinogenic chemicals, quantitative risk estimates have been based on source categories such as, diesel exhaust and tobacco smoke and on whole emissions rather than using the additivity of the components.

This concludes the brief overview of toxicity and human health effects associated with six of the toxic pollutants present in emissions from automotive vehicles using CG and RFG. A potency ratio approach has been suggested to evaluate human exposures to these toxic emissions from different blends of gasoline including RFG. With the exception of POM, this report used a consistent methodology (Global 86) to derive chemical-specific potency factors from appropriate animal data (Cancer Risk Assessment Guidelines, 1997). These factors were developed for the specific purpose of comparing emissions associated with the combustion and evaporation of reformulated and conventional gasoline in automotive vehicles.

B. Cancer Unit Risk Factors

B.1 Overview

As explained in the preceding section, most of the six toxic compounds evaluated in this assessment present both acute and chronic risk to exposed
individuals. The relative risk assessment conducted in this study, however, focuses strictly on carcinogenic effects. For the purposes of this comparison, all compounds were considered a non-threshold carcinogen, which means that there is no threshold below which the compounds are not carcinogenic. Some experts do not agree with this assumption.

In the 1994 Report, *Health Risk Perspectives on Fuel Oxygenates*, EPA suggested that MTBE has a cancer unit risk value somewhat lower than benzene and about 40 fold less than that of 1,3-butadiene. The EPA assessment made comparisons of pollutant toxicity using sources of data that are not directly comparable. Specifically, its comparisons were based on differences between upper bound estimates from animal studies and maximum likelihood estimates (MLEs) from human studies, which introduces a high degree of variability into the estimate of relative risk.

NESCAUM attempted to develop a more appropriate and uniform estimate of unit risk for use in this comparative assessment of the difference in toxicity between conventional and reformulated gasolines. We began by identifying the primary sources of variability in the risk estimates and where appropriate, attempted to reduce this variability. Much of the variability is reduced when more uniform cancer unit risks are used which have a consistent toxicological basis, such as comparisons relying strictly on either animal or human data.

Cancer unit risk defines the increased risk of cancer for each incremental increase in daily exposure. For example, a unit risk of 1E-06 per micrograms/cubic meter suggests that one additional cancer case would be expected in a population of one million if that population were exposed to an additional microgram per cubic meter of air over a lifetime. The cancer unit risk value is used to estimate the potencies of different carcinogens.

Cancer unit risk estimates are typically derived from one of two sources of data: human epidemiological studies and animal studies. To account for extrapolation, higher uncertainty factors (which in part incorporate a dose scaling adjustment for the difference between animals and humans) are used when animal studies are used to predict human risk. The lifetime cancer unit risk estimate is calculated using mathematical models which consider the outcomes in the higher dose animal studies and extrapolate to an estimate of the risk at low dose exposures found in the environment. Where more than one animal or human study is available, the study believed to be most reliable is selected for the estimate. If adequate human data is available, it takes precedence over animal data.

EPA's 1994 assessment and the Health Effects Institute's (HEI’s) review of that report cautioned that large uncertainties remain in projecting human risk of cancer from MTBE, air toxics, and other components of evaporative
and exhaust emissions of conventional gasoline or the change in emissions when fuel is oxygenated (Health Effects Institute, 1996). The HEI committee expressed the opinion that the band of uncertainty around any calculation of unit risk is wide and that risk estimates must be interpreted with caution. We agree with these observations and have designed this analysis to help quantify the bands of uncertainty.

The three approaches presented in this section for calculating relative risks suggest the bands of uncertainty associated with these comparisons. Analyses of relative risk among gasoline blends using different risk factors indicates the amount of variability introduced by the use of alternative methods of calculating relative risk.

B.2 Methodology for Quantifying Cancer Unit Risk

The cancer unit risk calculations developed with the Global 86 model for estimating cancer potency assume similar exposures and animal sensitivity. Two reference points were selected for each carcinogen, the upper bound risk estimate, and the maximum-likelihood estimate (MLE). The upper bound estimate is defined as the point at which estimated risk would exceed actual risk less than five times in one hundred. In other words, it represents the 95 percent confidence interval. By contrast, the MLE is defined as the point most likely to represent actual risk. For the purpose of relative risk analysis, the MLE comparison is most appropriate, and was used as the Base Case for this analysis. However, when setting regulatory limits on toxic emissions, the upper bound or IRIS risk values are most appropriate.

The IRIS database is an EPA-reviewed summary of the health effects of chemicals and includes cancer unit risk values for suspected carcinogens (IRIS data base for Benzene, 1997). The cancer unit risk estimates contained in the IRIS database are largely based on animal bioassays and typically represent upper bound risk estimates. Where adequate human epidemiological studies exist for a specific toxicant, these data are used to estimate the risk and the MLE value is listed in the IRIS database. The IRIS values also include uncertainty factors based on the quality of the studies being reviewed. Consequently, these values may not be directly comparable and could either over- or under-estimate the differences in risk between compounds. Nevertheless, the IRIS data base represents a logical starting point for an analysis of comparative risks.

In an effort to bound the uncertainty associated with this type of analysis, three sets of cancer risk estimates for the six toxic compounds of interest to this study are presented in Table IV-X. Method 1 relies on IRIS values which includes a mixture of MLEs from human data and upper bound risks from animal data. Method 2 uses a comparison of upper bound risks based solely on animal bioassay data. Method 3 compares the maximum
likelihood estimates from the animal data. In order to minimize methodological differences, Methods 2 and 3 risks are all calculated from comparable animal data using the Global 86 model. Due to technical difficulties, POM risks were not calculated using the Global 86 model (see appendix B).

Table IV-1

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Method 1 IRIS</th>
<th>Method 2 Upper Bound⁹</th>
<th>Method 3 (MLE) BASE CASE</th>
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<td>9.1E-06</td>
<td>8.6E-06</td>
</tr>
<tr>
<td>Formaldehyde</td>
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<td>Acetaldehyde</td>
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<td>POM¹²</td>
<td>1.7E-05</td>
<td>1.7E-05</td>
<td>n/a¹³</td>
</tr>
</tbody>
</table>

Table IV-1 shows the cancer unit risk estimates derived through each of the three methods. The upper bound approach tends to yield the most conservative risk estimates.

Method 1 relies on data from IRIS and on a POM unit risk value developed in this report. The benzene value represents a geometric average of unit risks based on MLE from several human epidemiological studies. It incorporates fewer uncertainty factors than would be found with unit risks derived from animal studies because there is no need to extrapolate between animal data and human data.

⁹ For Method 2 and Method 3, cancer unit risk values are estimated from most sensitive animal study, which may overestimate risk.
¹⁰ Cancer unit risk values for benzene are derived from human MLE data found in IRIS for Method 1 and animal data for Method 2 and Method 3.
¹¹ In February, 1998, EPA issued a new best estimate of human lifetime cancer risk from chronic exposure to 1,3-butadiene based on linear extrapolation of increased leukemia risks in occupationally exposed workers (National Center for Environmental Assessment, 1998). The best estimate is 9x10⁻³ per ppm. This would be 4.1x10⁻⁶ μg/m³. This number compares favorably with the risk derived in this report of 9.1x10⁻⁶ μg/m³ from animal data. An abstract may be found on the web at www.epa.gov/ncea/butadiene.htm.
¹² Estimated from components of POM, a mixture, and unit risk for BaP equivalent.
¹³ For POM, this report used the Upper Bound cancer unit risk value.
Except for POM, Method 2 is based on upper bound estimates of unit risk calculated exclusively from animal bioassay data using Global 86. Global 86 uses the maximum likelihood multi-stage model to evaluate quantal animal tumor data and calculates: (1) the maximum likelihood estimates (MLE) and (2) statistical confidence limits for extra risk over the background at a given dose, and for the dose corresponding to a given value of extra risk. The model is designed to provide plausible linear upper confidence limits to lifetime carcinogenic risk at low doses. Such linear upper confidence limits are believed to be conservative and protective of public health. Since the objective of the conventional risk assessment is to estimate lifetime extra risk in humans, the use of 95 percent upper bound estimate of unit risk is considered appropriate. The unit risks are influenced by the quality of the animal studies and the variability in the findings. The upper bound risk describes the statistical upper limit of the actual risk at a 95 percent confidence level. When incorporated in a relative risk calculation the variability among studies increases the uncertainty.

Method 3 is based on the MLE of unit risk calculated from the animal studies using the Global 86 model, except for POM. Since the principal objective of this report is to quantify and compare the relative risk posed by gasoline blends sold in the Northeast, the use of the best estimate of unit risk (i.e., the MLE of unit risk) was considered appropriate. This is the best estimate of the actual unit risk from each study. While variability introduced by differences among studies of the individual carcinogens are not controlled for in this approach, variability within the studies is controlled by using the MLE.

B.2.1 Benzene

The unit risk values for benzene are based on human studies in the IRIS database (Method 1) and on animal studies in the upper bound estimate (Method 2) and MLE (Method 3) approaches. The IRIS risk estimate is three to four times higher than the estimates based on animal data. This difference likely reflects the uncertainty factors incorporated into the human studies and species differences.

B.2.2 MTBE

Currently, MTBE unit risks are based on animal studies which detected tumors through either inhalation or ingestion. These findings raise concern about the potential carcinogenicity of MTBE in humans, but require further evaluation before designating MTBE as a possible or probable human carcinogen. Some of the tumors found in the animal studies are possibly specific to animals exposed in these studies — mouse liver tumors and rat kidney tumors — and may not indicate a potential human cancer risk. While this precaution is noted, this study did not attempt to address the issue of
whether or not MTBE is carcinogenic to humans. Rather, the risk comparisons in this study assume the worst case, i.e., MTBE cancer unit risk is based on the animal study which showed the highest potency based on tumor outcomes. This approach was taken in order to provide the greatest margin of safety in our analysis. It is possible that this approach overestimates cancer potency or that MTBE will not be shown to be carcinogenic with subsequent review. The risk assessment approach used for MTBE in this study is described in Appendix A.

B.2.3 1,3-butadiene

The 1,3-butadiene unit risk value in IRIS is based on animal studies and represents an upper bound estimate of risk. Data from the same animal studies were used in conjunction with the Global 86 approach to derive the risk values in Method 2 and Method 3. Certain assumptions and correction factors used in the IRIS calculation, however, were not used in order to maintain a uniform methodology. This omission may somewhat underestimate the absolute risk of 1,3-butadiene. Prior comparative risk studies have assumed a potency ratio of 50 between 1,3-butadiene and benzene. A ratio of 33.7 to 1 between butadiene and benzene was derived using Method 1. A primary factor in the differences between the unit risk in IRIS and the unit risks found in this study's Base Case is related to estimates of absorption.

B.2.4 Formaldehyde and Acetaldehyde

The differences in the unit risk values for formaldehyde and acetaldehyde, used in the three methods, reflect differences in methodology and assumptions. The relative potencies suggest that these compounds are not responsible for a large portion of the overall cancer risk from gasoline, regardless of the potency ratio used.

B.2.5 POM

Polycyclic organic matter is a mixture of chemicals, including some highly carcinogenic polycyclic hydrocarbons (PAHs). There is no cancer unit risk value for POM in IRIS because it is a mixture and because there are significant differences in the components in the POM depending on the source of emissions. Two approaches were considered to derive a cancer unit risk value for POM from gasoline exhaust. The first relied on the findings from a skin painting study designed to compare the potency of POM from different sources including coke oven emissions, diesel emissions and gasoline exhaust condensate. It is possible to extrapolate a unit risk of 7.0E-05 from this study. This value is similar to the upper bound estimates. It is possible that the condensate contained other carcinogens or that the skin
painting study vary from the inhalation or oral studies in terms of relative potency.

In order to further understand the POM cancer unit risk from gasoline exhaust an estimate was made of the risk from the various components which comprise POM. Benzo(a)pyrene (BaP) is a potent carcinogen which has been widely studied as an ingredient in cigarette smoke. A cancer unit risk has been published for BaP. Comparative risk estimates of polyaromatic hydrocarbons typically express the cancer risk in terms of BaP equivalents. EPA provided some preliminary findings from an analysis of POM in a series of studies of gasoline automobile exhaust. About 1 percent of the POM is BaP or 'BaP equivalents'. This is consistent with a cancer unit risk of $1.7 \times 10^{-5}$. While this estimate may have not considered certain carcinogenic components of the POM, the derivation methodology for the unit risk from the animal studies is consistent with that used for the other components.

C. Epidemiological and Anecdotal Studies

Following the introduction of MTBE-oxygenated gasoline in Fairbanks and Anchorage, Alaska in November 1992, numerous reports of acute health symptoms in connection with the new fuel were registered by residents in those areas. In response to the concerns expressed by officials of the State of Alaska, a research program involving government, industry, and academia was initiated to investigate the acute human health effects of MTBE. These research studies did not provide definitive evidence that the acute symptoms were due to the new fuel introduced in Alaska. A chamber study in Sweden (Johanson et al., 1995) using 2-hour exposures to MTBE concentrations up to 180 mg/m$^3$ reported that other than detection of MTBE odor and the reported that poor air quality in the chamber, no reactions to exposure to MTBE were observed in ten healthy human volunteers.

Field studies of human populations were also conducted in various locations. A study in New Jersey (Mohr et al., 1994) evaluated 237 garage workers from two groups: (1) northern New Jersey workers sampled during the wintertime oxyfuel program and (2) southern New Jersey workers sampled 10 weeks after the phase-out date for the program in that area. Essentially no differences in symptom reports were found between the northern (high-exposure) and southern (low-exposure) groups. Subgroups of refuelers differed significantly in pre/postshift symptom reports between north and south, but not significantly when matched for age, gender, and education. Another study in the New Jersey area (Fiedler et al., 1994) investigated MTBE-related symptom reports in 14 persons with known multiple chemical sensitivity (MCS), 5 persons with chronic fatigue syndrome (CFS), and 6 normal controls. Both MCS and CFS subjects reported
more symptoms than controls, but the pattern of their reports "did not provide clear evidence to support that an unusually high rate of symptoms or an increase of symptoms was occurring uniquely where MTBE was most prevalent" (i.e., refueling or driving an automobile).

Milwaukee, Wisconsin began to use MTBE in its gasoline as part of the federal RFG program in November 1994. Similar health complaints, as voiced in Alaska, were registered in Wisconsin. The EPA, Wisconsin Department of Health, Centers for Disease Control, and the University of Wisconsin investigated complaints from approximately 1500 people. They wrote two reports and concluded that they could find no definitive relationship between reported health effects and MTBE exposure (Wisconsin Department of Health and Social Services, May and September, 1995).

These studies suggest that the general population is not experiencing adverse health effects from exposure to MTBE. However, these studies do not rule out the possibility that sensitive human subjects may experience acute health symptoms from exposure to oxygenated or reformulated gasolines.

D. Cancer Potency Ratios

Since this study is designed to assess relative, rather than absolute risk, the unit risk values are converted to cancer potency ratios. The potency ratios are calculated based on the applicable unit risk values and represent a cancer risk relative to benzene (1.0). Potency ratios were developed for all three sets of risk factors. It is important to keep in mind that the methodology used to derive the cancer unit risk affects the potency ratios since differences in the unit risks are an important variable in uncertainty.

The results of the potency ratio calculations are presented in Table IV-2. According to the Method 1 approach, 1,3-butadiene is the most toxic of the pollutants, some thirty times more toxic than benzene. For both Method 2 and 3, butadiene is shown to be about three times more toxic than benzene. POM is the most toxic of the six pollutants according to both Methods 2 and 3.

Since the potency ratios shown are relatively sensitive to slight changes in the cancer unit risks, they provide only an approximation of the relative risks. But the ratios do indicate the extent to which changes in the emissions of the various toxic compounds affect the overall cancer potency of different blends of gasoline.
Table IV-2
Cancer Potency Ratios

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Method 1 IRIS</th>
<th>Method 2 Upper Bound</th>
<th>Method 3 MLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzene</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>MTBE</td>
<td>0.5</td>
<td>0.16</td>
<td>0.14</td>
</tr>
<tr>
<td>1,3-butadiene</td>
<td>33.7</td>
<td>3.1</td>
<td>3.58</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>1.6</td>
<td>0.9</td>
<td>0.004</td>
</tr>
<tr>
<td>Acetaldehyde</td>
<td>0.3</td>
<td>0.16</td>
<td>0.07</td>
</tr>
<tr>
<td>POM</td>
<td>2.0</td>
<td>5.86</td>
<td>7.1</td>
</tr>
</tbody>
</table>

E. Limitations of a Risk-Weighted Approach

Unlike a traditional risk assessment, the risk-weighted approach used in this study does not include an exposure assessment. An exposure assessment is used to adjust the cancer potency estimates in order to account for the level of expected exposure to human populations. In this study, the impact of an exposure assessment will depend upon the estimated level of exposure to human populations from each of the six toxic air pollutants. If the levels of exposure are identical for the six toxins in question, then the addition of an exposure assessment will not alter the current estimates of relative risk. Since this study is concerned exclusively with relative cancer risk, our conclusions would be influenced by the addition of an exposure assessment only if the exposure levels of the six toxic compounds differed significantly.

Toxic emissions degrade in the environment with time and ambient conditions. In order to evaluate the health risks from air toxics, it is necessary to use photokinetic (sunlight degradation) rate data to determine if there is any significant decomposition of the toxic emissions before human exposure. According to a study by the Colorado School of Mines, Microenvironmental Exposure Analysis: Evaluation of the Toxicity of Conventional and Oxygenated Motor Fuels, in the spring, fall and winter, the half-life of the benzene, formaldehyde, 1,3-butadiene, POM, acetaldehyde, and MTBE are sufficiently long (weeks to months) so that it can be assumed that no decomposition occurs before human exposure. In the summertime, significant degradation occurs with 1,3-butadiene, especially in the south,
within one to four hours. However, the Colorado School of Mines study suggested that even the short half-life of 1,3-butadiene may be long enough to cause direct human contact since the principal exposure to vehicle exhaust occurs during commuting and at refueling stations along busy streets directly downwind of exhaust emissions (D. Mowery, M.S. Graboski, 1996). In addition, the oxidation products from 1,3-butadiene degradation are acrolein, which is also a potentially toxic compound, and formaldehyde.

The dominant pathway for human exposure to toxic emissions from motor vehicles is inhalation of exhaust emissions while seated in a moving vehicle. The major source of vehicle cabin exhaust emissions is other vehicles on the road in close proximity to the vehicle in question. The Colorado School of Mines study estimates that about 70 percent of the exposure to motor vehicle toxics occurs in the vehicle cabin.

A secondary pathway for exposure to motor vehicle toxic emissions is evaporative emissions which occur primarily during vehicle refueling. Of the six toxics studied, only benzene and MTBE are found in evaporative emissions. The relative cancer risk estimates presented in this study weight evaporative and exhaust emissions equally. The Colorado School of Mines study estimates that evaporative emissions account for about 30 percent of the human exposure to toxic emissions in vehicle exhaust. An exposure assessment would diminish slightly the importance of evaporative emissions of benzene and MTBE in relation to the emissions found in vehicle exhaust.

Based on the information available about the half-life and major exposure pathways associated with the six toxic compounds relevant to this study, it is possible to draw three general conclusions about the potential impact of an exposure assessment upon our findings. First, since the primary exposure pathway is exhaust emissions inhaled while seated in the vehicle cabin, the equal treatment of each toxic pollutant in our risk-weighted analysis is sound and is likely to remain largely unchanged (D. Mowery, M.S. Graboski, 1996). Second, the shorter half-life of 1,3-butadiene will probably result in a slight narrowing of the relative benefit of RFG over CG during the summer months since 1,3-butadiene’s contribution to the reduction in relative cancer risk will be diminished. Third, while our analysis apparently overestimates the relative impact of evaporative emissions on risk to the public health, the impact on our relative risk analysis is probably marginal since the benefits of evaporative benzene reductions and the detriments associated with evaporative MTBE increases will to some extent offset each other.

These observations are presented here to provide the reader with a rough sense as to how the results in this risk-weighted study would compare to a traditional risk assessment which would account for differences in levels of exposure. NESCAUM fully supports a full risk assessment to achieve a more accurate depiction of the relative risk of CG as compared with RFG.
V. Results of Relative Cancer Risk Analysis

A. Overview

This analysis compares the relative cancer risk associated with human exposure to the exhaust and evaporative emissions of toxic air pollutants from motor vehicles burning conventional gasoline and federal reformulated gasoline (RFG). The information generated in this study is intended to further inform Northeast decisionmakers regarding the public health benefits associated with the toxic emission reduction component of the federal RFG program. The results presented in this section provide a quantitative representation of the relative cancer risk associated with exposure to conventional gasoline, Phase I blends and Phase II RFG.

The two primary variables in this analysis are the gasoline fuel compositions and the cancer potency ratio values for each of the five regulated toxic air pollutants and MTBE. Our Base Case Scenario uses the average fuel properties calculated in Section III of this study and the cancer potency ratios derived from the maximum likelihood estimate (Method 3) approach described in Section IV.

Additional analyses were performed to explore the sensitivity of the results to changes in assumptions about the composition of Phase II RFG and alternative cancer potency ratios. In Sensitivity Case #1, three alternative Phase II RFG formulations were modeled in conjunction with the Method 3 cancer potency ratios. Sensitivity Case #2 used the Base Case fuel quality assumptions in conjunction with the Method 1 and Method 2 cancer potency ratios.

B. Methodology

This analysis used the fuel formulation data from Section III and the relative cancer potency ratios from Section IV (see table below) to calculate the differences in relative cancer risk between RFG and conventional gasoline sold in the Northeast. The PADD-weighted average formulations for CG, Phase I RFG and Phase II RFG sold in the Northeast were run through the Complex Model to generate milligram per mile emission estimates for each of the five toxics pollutants regulated under the federal RFG program. The Complex Model does not, however, estimate MTBE emissions. In order to incorporate MTBE into this analysis, NESCAUM, with assistance from EPA and DOE, developed an MTBE emissions factor model to generate comparable emission estimates for this pollutant. This model estimates both exhaust and
nonexhaust MTBE emissions; it estimates nonexhaust MTBE emissions based on percentage of hydrocarbons (HC).

Hydrocarbon exhaust emissions are significantly affected by the RVP of a fuel. The MTBE model estimates two different levels of nonexhaust MTBE emissions based on different assumptions about RVP. "Method A" assumes that the percent MTBE of HC does not change, which means that decreasing RVP from the base case will dramatically reduce MTBE emissions. "Method B" estimates MTBE nonexhaust emissions based on MTBE percent of HC at base RVP. This assumes that the level of MTBE is the same regardless of a fuel's RVP. In order to err on the conservative side, we chose to use Method B in this report because of the two nonexhaust models, Method B results in greater nonexhaust MTBE emissions.

The predicted emissions for each of the toxic air pollutants were subsequently weighted according to the relative cancer potency ratio values to provide a quantitative representation of the relative cancer risk from RFG and conventional gasoline sold in the Northeast.

The RFG program requires reductions in emissions of VOCs, NOx, and toxics as compared with emissions from a CAA baseline gasoline intended to represent a national average fuel in 1990. It is important to note, however, that this analysis uses 1996 conventional gasoline sold in the Northeast as the baseline against which to compare the relative toxic emissions benefits from Phase I and Phase II of the RFG program. This approach was selected since opt-in states cannot choose CAA baseline fuel; rather the choice is between current conventional gasoline or RFG.

<table>
<thead>
<tr>
<th>BASE CASE</th>
<th>Cancer Potency Ratio Values*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzene</td>
<td>1.0</td>
</tr>
<tr>
<td>1,3-butadiene</td>
<td>3.58</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>0.004</td>
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<td>0.07</td>
</tr>
<tr>
<td>POM</td>
<td>7.1</td>
</tr>
<tr>
<td>MTBE</td>
<td>0.14</td>
</tr>
</tbody>
</table>

*Method 3 (Max. Likelihood Estimate)

C. Results

C.1 Base Case

The relative toxicity of conventional gasoline, Phase I RFG and Phase II RFG are compared using units designated as "relative cancer risk" — defined in this study as the product of mass emissions and the associated cancer risk.
potency ratio. Cancer potency ratios are derived by setting the cancer unit risk value of benzene to one (1.0) and then adjusting each of the other five cancer unit risk values by the same factor. The units of relative cancer risk and the cancer potency ratios are meaningful only as they relate to one another, and should not be taken out of context.

C1.1 Relative Cancer Risk of CG and RFG Phase I and II

The findings in this study suggest that RFG has served to reduce the relative cancer risk associated with gasoline vapors and automobile exhaust when compared against conventional gasoline in the Northeast. Public health benefits from RFG are projected to increase with the introduction of Phase II RFG in the Northeast in the year 2000. Notably, substantial public health benefits were predicted with the use of RFG rather than conventional gasoline, even after including MTBE in the analysis as a carcinogen.

The major findings of this study are presented in Figure ES-1. The Y-axis represents the percent decrease in relative cancer risk associated with the use of Phase I RFG (left bars in white) and Phase II RFG (right bars in gray) as compared with conventional gasoline. The relative cancer risk associated with the use of conventional gasoline is the baseline against which RFG Phase I and Phase II are compared, and thus CG is not depicted in this chart. Moving from left to right, the X-axis identifies the three petroleum production regions which supply the Northeast with gasoline, and then aggregates the relative cancer risk of the gasoline blends from each region by their proportional share of the Northeast gasoline market for the final result, which is labeled “Northeast.”

Our analysis shows a 12 percent reduction in relative cancer risk experienced by the Northeast through the use of Phase I RFG and an expected further reduction in relative cancer risk to 20 percent in 2000 and thereafter due to the introduction of Phase II RFG into the gasoline retail market. These reductions in relative cancer risk are as compared with 1996 conventional gasoline sold in the Northeast. The results of this analysis support the conclusion that the federal reformulated gasoline program serves to reduce the cancer risk associated with gasoline vapors and automobile exhaust compared to conventional gasoline.

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14 As a point of reference, the federal regulations require a 15 percent reduction in toxic mass emissions from Phase I RFG, and a 20 percent reduction in toxic mass emissions from Phase II RFG.
The largest reductions in relative cancer risk come from Gulf Coast (PADD III) and East Coast (PADD I) refineries which are projected to reduce relative cancer risk by 23 percent and 18 percent respectively in Phase II RFG as compared with conventional gasoline produced by the same refineries. Three formulation changes are primarily responsible for the predicted relative cancer risk reductions: (1) lower benzene content; (2) lower olefin content; (3) lower RVP. The sharp decline expected to occur in the benzene content of Phase II RFG produced by East Coast refineries as compared to CG is the dominant factor in the aggregate reduction in relative cancer risk seen in PADD I.

The bulk of PADD III's 23 percent relative cancer risk reduction is due to the substantial difference in 1,3-butadiene emissions from CG as compared to Phase II RFG. Butadiene emissions are dependent upon olefin content. Olefin levels in Gulf Coast CG are 16 percent by volume while our analysis predicts olefin levels will drop to 9.0 percent by volume for Phase II RFG produced in the Gulf Coast. RVP adjustments in Phase II RFG reduced evaporative emissions of benzene and MTBE, but the percentage contribution to the reduction in relative cancer risk was not as substantial as those generated by benzene and 1,3-butadiene exhaust emission reductions.

Aromatics are another source of toxic emissions. Aromatic content in Phase II RFG is expected to increase slightly for each PADD under this analysis, which will offset some of the gains from reductions in benzene,
olefins and, to a lesser extent, RVP. Aromatic levels for Phase I RFG are 17.5, 21.3, and 20.7 percent by volume for East Coast, Gulf Coast and foreign refineries respectively. Our study estimates aromatic content will increase in Phase II RFG to 25 percent by volume for PADDs I and III, and will stay at Phase I levels for foreign refineries. If the domestic refineries maintain their relatively low aromatic levels currently found in Phase I RFG, it is likely that Phase II RFG will generate a greater reduction in relative cancer risk as compared with conventional gasoline that currently predicted by this analysis.

Foreign refineries are projected to reduce relative cancer risk by 15 percent in Phase II RFG. In absolute terms, foreign refineries are producing some of the cleanest gasoline in the region with benzene and olefin levels well below the regional average for Phase I RFG. The relatively smaller difference in relative cancer risk predicted in Imported CG and RFG gasolines is due to the low benzene (64% vol.) and olefin (12.6% vol.) levels found in conventional gasoline produced by foreign refineries. Moreover, since on average Imported Phase I RFG already meets the Phase II RFG NOx performance standard due to exceptionally low sulfur (192 ppm) and olefin (7.7% vol.) levels, the only change expected in Phase II RFG is a downward adjustment in RVP which has little impact on toxic emissions. Imports account for about 20 percent of the gasoline sold in the Northeast and these results are weighted accordingly in this relative risk analysis.

C.1.2 Pollutant-Specific Relative Cancer Risk

The second set of findings are found in Figure V-2, which presents the predicted relative cancer risk for each individual toxic air pollutant. The numbers along the Y-axis provide a measure of the relative cancer risk of the toxic air pollutants, but are not meaningful by themselves and should not be taken out of context. The six toxic air pollutants are listed along the X-axis. Moving from left to right, the relative cancer risk for each chemical compound is estimated based on the emissions from CG, RFG Phase I, and RFG Phase II. These results provide policy makers with an assessment of the appropriateness and effectiveness of Phase I RFC, and an estimate of the additional improvement expected with the introduction of Phase II RFC, for each chemical compound evaluated in this study.
According to this analysis, benzene is the most significant contributor to cancer risk of the six toxic compounds evaluated, accounting for more of the relative cancer risk than the five other pollutants combined. The wisdom of Congress’ requirement that petroleum refiners reduce the amount of benzene in gasoline to below 1.0 percent by volume (the benzene cap) is supported by this finding. This study found that petroleum refiners reduced the benzene content in RFG to about 0.7 percent by volume — or 30 percent below the legal limit of 1.0 percent by volume — presumably due to a market demand for this chemical compound. The fact that benzene remains the primary contributor to cancer risk in Phase II RFG as compared with the five other toxic air pollutants indicates that a continued regulatory focus on benzene is appropriate.

Butadiene poses the next highest level of risk in conventional gasoline and Phase I RFG. The cancer potency of 1,3-butadiene is three times that of benzene.\(^{15}\) Exhaust emissions of 1,3-butadiene are directly related to the olefin content in gasoline, which is not directly regulated under the RFG program. The 15 percent reduction in butadiene emissions from Phase I RFG as compared with conventional gasoline is presumed to result from the displacement of olefin levels by the addition of MTBE at 10 to 15 percent by volume. The additional 15 percent reduction in emissions of 1,3-butadiene predicted from the introduction of Phase II RFG is primarily due to olefin

\(^{15}\) For the Base Case Scenario, which used maximum likelihood estimates to derive numerical cancer risk values.
reductions in order to comply with Phase II NOx requirements. Should they deem it necessary, public policy makers might consider direct regulation of gasoline olefin content as a means of reducing the significant contribution of 1,3-butadiene to overall relative cancer risk.

The relative cancer risk from MTBE is slightly greater than the relative cancer risk associated with 1,3-butaadiene for Phase II RFG formulations. While this places MTBE as the second largest contributor to relative cancer risk after benzene in Phase II RFG, it is imperative to recognize that this analysis characterized MTBE as a carcinogen despite disagreement from some public health experts, and that the MTBE model employed by this study assumed the highest emissions factor for MTBE evaporative emissions. In simple terms, the MTBE analysis represents a worst case scenario in order to provide the greatest margin of safety. While this approach strengthened the reliability of this study’s major conclusion — that RFG with MTBE provides a substantial long term public health benefit to the general population — it may provide a less definitive measure of the relative importance of MTBE as compared with the five other toxic air pollutants.

Acetaldehyde, POM, and formaldehyde rank in that order according to estimated relative cancer risk. As with 1,3-butadiene, there is no direct requirement that petroleum refineries reduce emissions from these toxic air pollutants, other than the general requirement that RFG provide a reduction in mass emissions of toxic air pollutants. The 10 percent reduction in emissions from acetaldehyde and POM from Phase I RFG compared to conventional gasoline are presumably due to the corresponding addition of MTBE at about 10 percent by volume. Emissions of formaldehyde are greater with the use of RFG than with CG, but are too small to appear in Figure V-2 due to the very low cancer potency ratio for formaldehyde.

C.2 Sensitivity Analyses

Two sensitivity analyses were conducted to examine the effect that changes in assumptions regarding the two key variables would have on the Base Case results. Sensitivity Case #1 explores the effects of changes in fuel formulation assumptions and Sensitivity Case #2 looks at the effect of changing cancer potency ratios.

C.2.1 Sensitivity Case #1 - Fuel Composition

Phase II RFG will require an additional reduction in NOx emissions of about 5 percent on average beyond what is required for Phase I RFG. Petroleum refiners may achieve the NOx standard through a combination of reductions in three gasoline fuel properties: sulfur, olefins and aromatics. The Base Case predicts a Phase II sulfur level of 132 ppm in PADD I and 127 ppm in PADD III; an olefin level of 12.4 percent in PADD I and 9.0 percent in
PADD III; and an aromatics level of 25 percent for both PADDs. As each of these key fuel properties affects emissions of toxic air pollutants differently, this analysis examines the impact of Phase II RFG with the highest practical sulfur, olefin and aromatic content for the compliant fuel. In effect, each of these three scenarios explores a "worse case scenario" for toxic emissions from Phase II RFG.

The level of adjustment to sulfur content does not appear to significantly affect emissions of toxic air pollutants. The High Sulfur Scenario assumed a sulfur content for Phase II RFG of 184 ppm, which is about 50 ppm higher than the Base Case. Figure V-3 shows an increase in relative cancer risk of 21 percent between CG and Phase II RFG. By comparison, the Base Case resulted in a 20 percent reduction in relative cancer risk from CG to Phase II RFG. These results demonstrate a strong insensitivity to a 50 ppm change in sulfur content.

**Figure V-3**
**High Sulfur Scenario**
**Percentage Change in Relative Cancer Risk from Conventional Gasoline Sold in the Northeast (Summer)**

The second alternative fuel analysis explores the relationship between olefin content to relative cancer risk. The olefin content of gasoline is positively correlated to emissions of 1,3-butadiene, one of the most potent toxic air pollutants. The results are depicted in Figure V-4. The relative cancer risk of Phase II RFG is about 17 percent less than that for CG in the High Olefin case. The Base Case Phase II RFG resulted in a 20 percent reduction in relative cancer risk. The difference is due to the change in olefin levels from
14.5 percent in this analysis compared with lower range in the Base Case from 12.4 percent and 7.7 percent.

**Figure V-4**
High Olefin Sensitivity Run
Percentage Change in Relative Cancer Risk from Conventional Gasoline Sold in the Northeast (Summer)

In the Base Case, PADD III fuels showed a 23 percent reduction in relative cancer risk from Phase II RFG as compared with conventional gasoline. In the High Olefin Scenario, PADD III's relative cancer risk benefits are diminished to 19 percent due to an olefin content of 14.5 percent by volume for Phase II RFG, as compared with 9.0 percent by volume for Phase II RFG in the Base Case. More importantly, under this sensitivity analysis, Phase II RFG produced by foreign refineries experienced an increase in relative cancer risk as compared with Phase I RFG. The increase in relative cancer risk predicted for Imported RFG is explained by the near 100 percent increase in olefin content from Phase I RFG at 7.7 percent by volume to Phase II RFG under the High Olefin Scenario at 14.5 percent by volume.
The High Aromatic Scenario assumes an aromatic content at 33.2 percent by volume, which is about 50 percent above the range of aromatic content found in Phase I RFG sold in the Northeast. The results presented in Figure V-5, show a strong sensitivity of relative cancer risk to aromatic levels. The High Aromatics Scenario reduced the public health benefit of RFG compared to CG.

C.2.2 Sensitivity Case #2 - Cancer Potency Ratios

This study derives its Base Case cancer unit risk values through a statistical method called the maximum likelihood estimate (MLE) — referred to as Method 3. As detailed earlier in this study, alternative methods exist for quantifying unit risk. In this sensitivity analysis, we use the two alternative cancer unit risk values and ratios described in Section IV. Method 1 was developed from potency values in the IRIS database and these values were used by EPA in previous risk and cost effectiveness assessments. The cancer unit risk values in the IRIS database are a combination of MLEs and cancer unit risk values derived from a statistical approach called “upper bound.” The “upper bound” approach for setting cancer unit risk values is used when establishing a limit for human exposure to a particular carcinogen. The “upper bound” refers to the large margin of safety used when setting standards to protect human health. In Method 2, a pure “upper bound” approach is utilized.
The table to the right shows the values for Method 1 (IRIS). As with each Method, the cancer unit risk value for benzene is set to 1.0, and other toxic air pollutants cancer unit risk values are adjusted accordingly. The largest change is seen in the cancer potency ratio of 1,3-butadiene which increases by almost an order of magnitude from 3.7 in Method 3 (MLE) to 33.7 in Method 1. Formaldehyde and MTBE’s cancer potency ratios also increase substantially.

The results depicted in Figure V-6 show a 9 percent reduction in relative cancer risk when CG is compared with Phase I RFG, and a dramatic decline of 25 percent in relative cancer risk when Phase 2 RFG is compared with CG. The predicted decline in olefin levels in Phase II RFG, and the associated reductions in relative cancer risk from 1,3-butadiene, resulted in the large reductions in Phase II RFG as compared with CG. Olefin content in Phase I RFG is close to that of CG. Under this analysis for Phase I as compared with CG, predicted increases in the potency of MTBE and formaldehyde—the two toxic compounds whose mass emissions are greater in RFG than in conventional gasoline—resulted in a reduction in relative cancer risk of 9 percent, which is significantly less than the toxic threat reduction of 12 percent in the Base Case.
Interestingly, the relative contribution from the three petroleum producing regions has changed dramatically reflecting the dominance of 1,3-butadiene as a key contributor to overall toxic threat under this sensitivity scenario. Emissions of 1,3-butadiene are primarily a function of olefins levels in gasoline, and PADD III shows the greatest decrease in olefin levels from conventional gasoline at 16 percent by volume to our predictions for Phase II RFG at 9 percent by volume. Foreign refineries have the lowest olefin content in conventional gasoline at 12.6 percent and are not expected to reduce olefin levels by any measurable amount to comply with Phase II RFG requirements. Significant reductions are provided by PADD I RFG due to its expected decline in olefin content in Phase II RFG to 12.4 percent by volume.

<table>
<thead>
<tr>
<th>Method 2 (Upper Bound)</th>
<th>Cancer Potency Ratio Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzene</td>
<td>1.0</td>
</tr>
<tr>
<td>1,3-butadiene</td>
<td>3.1</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>0.9</td>
</tr>
<tr>
<td>Acetaldehyde</td>
<td>0.16</td>
</tr>
<tr>
<td>POM</td>
<td>5.86</td>
</tr>
<tr>
<td>MTBE</td>
<td>0.16</td>
</tr>
</tbody>
</table>

The final sensitivity analysis assumes Method 2 (Upper Bound) values. The cancer potency ratios from this method are provided in the table to the right. These potency ratios establish, in effect, a middle ground between Method 3 used in the Base Case and the cancer potency ratios in Method 1 (IRIS). The major differences from Method 1 are in butadiene’s cancer potency ratio which has dropped from 33.7 to 3.1, and formaldehyde, which has declined from 1.6 to
0.9. MTBE's cancer potency ratio has also declined from 0.5 in Method 1 to 0.16 in Method 2.

The results in Figure V-7 show an overall reduction in relative cancer risk of about 17 percent for Phase I RFG and about 25 percent for Phase II RFG as compared with conventional gasoline produced by the same refineries. These reductions are greater than those predicted in the Base Case (Phase I at 12 percent and Phase II at 20 percent). As with some of the results from Method 1 (IRIS) presented in Figure V-6, these results are explained by the decreased cancer potency ratios for formaldehyde and MTBE — the only toxic compounds whose emissions are greater when vehicles are operated on RFG than on conventional gasoline — as compared with the Base Case.

Figure V-7
Method 2 (Upper Bound)
Percentage Change in Relative Cancer Risk from Conventional Gasoline Sold in the Northeast (Summer)

Under Method 1, the cancer potency ratios of formaldehyde and MTBE saw an increase compared with the Base Case, but these increases were more than offset by relative cancer risk reductions from 1,3-butadiene, whose cancer potency ratio had increased by a factor of ten. Under the Method 2 analysis presented in Figure V-7, 1,3-butadiene's cancer potency ratio was reduced by a factor of ten, and relative cancer risk reductions from 1,3-butadiene were no longer able to offset the increases in relative cancer risk from formaldehyde and MTBE.
D. Conclusions

The results of this analysis comparing the relative cancer risk of conventional gasoline to that of reformulated gasoline suggest that:

- Phase I federal reformulated gasoline sold in the Northeast in 1996 served to reduce the cancer risk associated with gasoline vapors and automobile exhaust compared to conventional gasoline by 12 percent in our base case scenario. For all scenarios evaluated, the cancer risk reduction associated with Phase I RFG as compared with conventional gasoline ranged from a low of 9 percent to a high of 12 percent.

- Decreases in ambient levels of benzene detected at some Photochemical Assessment Monitoring Station (PAMS) sites before and after the introduction of Phase I RFG support the conclusions reached in this study as to the toxic reductions achieved through the use of Phase I RFG.

- Phase II federal reformulated gasoline is expected to further reduce the public cancer risk from exposure to gasoline vapors and automobile exhaust as compared with conventional gasoline by 20 percent in our base case scenario. For all scenarios evaluated, the reduction in cancer risk from Phase II RFG as compared with conventional gasoline ranged from a low of 14 percent to a high of 26 percent.

- For all gasoline blends, benzene's contribution to overall cancer risk is greater than the sum of the cancer risks of the other five toxic compounds. This finding affirms the decision made by the drafters of the Clean Air Act Amendments of 1990 to establish specific gasoline fuel specifications for benzene. Despite reductions in cancer risk from benzene of about 30 percent from conventional gasoline to RFG Phase I and Phase II, this pollutant continues to represent the primary cancer risk as rated against the five other toxic compounds.

- The cancer potency of MTBE is an order of magnitude less than benzene, and some of the combustion byproducts such as POM, and 1,3-butadiene. With the exception of acetaldehyde, these three toxicants represent the major potency-weighted emissions of toxic air pollutants in this study. Typical blends of RFG contain 10 percent MTBE, which tends to dilute these and other carcinogens commonly found in conventional gasoline. This “dilution effect” contributes to a reduction in the overall relative cancer risk associated with RFG as compared with conventional gasoline.

- The toxic benefits of the RFG program accrue in addition to the substantial ozone reduction benefits realized as a consequence of this strategy.
Analyses of ambient ozone data from before and after the introduction of low volatility gasoline in the Northeast beginning in 1989 and the introduction of Phase II California RFG beginning in California in 1995 suggest that these strategies have measurably reduced ozone levels in the program areas.

- While the acute health effects from conventional gasoline and RFG oxygenated with MTBE reported by certain segments of the population have neither been proven nor dismissed, it is important to consider the widespread benefits of reductions in cancer risk and ground level ozone formation associated with the use of RFG Phase I and Phase II when evaluating public policy choices.

VI. Future Research Objectives

A. Overview

While this study suggests that the federal reformulated gasoline program is providing important public health benefits, there is a need for further study to verify this conclusion and assess the overall implications of the RFG program, particularly with the pending introduction of Phase II RFG in 2000. NESCAUM suggests additional work in the following areas: (1) ambient monitoring; (2) exposure assessment; (3) MTBE health effects; and (4) groundwater contamination.

- Measured reduction of toxics in the air represent the best indicator of the effectiveness of the RFG program. Therefore, continued collection and further analysis of ambient monitoring data is necessary to establish a pre-Phase II baseline and to confirm the model-predicted impact of RFG on air quality and public health.

- Given the different residence times of the various motor vehicle-related air toxics, an exposure assessment of hazardous air pollutants (HAPs) from fuel combustion and evaporative emissions is warranted to more accurately depict the relative risk of conventional gasoline and RFG.

- RFG is expected to reduce the cancer risk from exposure to gasoline vapors and automobile exhaust compared to conventional gasoline. However, assuming the continued use of ether-based oxygenates, further research is needed on systemic, neurotoxic, reproductive, and developmental effects of MTBE and MTBE inhaled with RFG.
• Because MTBE is highly water soluble relative to other constituents in gasoline and is poorly adsorbed to soils, it moves with the groundwater flow largely unretarded. Therefore, when RFG is released to the environment, it presents an increased potential for groundwater contamination compared to conventional gasoline. Further studies are necessary to investigate the potential for MTBE groundwater contamination and the resulting public health and ecological effects.

B. Ambient Monitoring

NESCAUM recommends enhanced collection and assessment of ambient toxics (benzene, ethylbenzene, toluene, xylene, NO\textsubscript{2}, formaldehyde, acetaldehyde, and acetone) and ozone data in the Northeast to establish a viable pre-Phase II baseline. Without significant amounts of consistent and valid ambient data for the period prior to and after the introduction of Phase II RFG, it is difficult to confirm and quantify the changes in air toxic concentrations resulting from the introduction of this fuel.

It is important to recognize, however, that factors such as the simultaneous reductions from other control strategies complicate this task and make it difficult to entirely differentiate the influence of RFG on air quality. The following steps are suggested to aid in establishing a viable baseline:

• Analyze and perform QA/QC for ambient monitoring data at PAMS sites within the Northeast to develop a baseline data set for quantifying RFG’s effectiveness in reducing summertime emissions of air toxics and ozone. Future compounds to monitor include: oxygenate used in the region’s RFG and/or their decomposition products and other species predicted to increase or decrease with RFG implementation. PAMS monitors in the Northeast should be evaluated for their ability to measure these compounds and, if necessary, their measurement protocols should be revised.

• Expand wintertime monitoring for specific HAPs, such as benzene, toluene, xylene, associated with fuel combustion and evaporation (PAMS sites only measure 24-hour averages once every six days in the winter).

• Consider co-locating toxic monitors with existing CO and acetylene monitoring stations in various Northeast urban centers.
Isolate peak exposure times (i.e. time periods of maximum VMT) during rush hour traffic for both a.m. and p.m.

Assess meteorological conditions, ambient temperatures, seasonal variation, changes in the length of the oxyfuel season, weekday/weekend effects, and background concentrations from stationary and fugitive sources.

Characterize RFG/MTBE emissions under different meteorological, roadway, and operating conditions to better understand issues such as the effect of cold temperature on the emission rates for MTBE.

Consider conducting tunnel studies in the Northeast which provide data on mobile source emissions with decreased stationary and area source contributions.

Compare ambient monitoring data from RFG areas and non-RFG areas.

C. Exposure Assessment

An exposure assessment would provide a more definitive sense of the absolute and relative risk to the public from exposure to emissions from vehicles operating on various fuel formulations. Toxic emissions degrade in the environment with time and ambient conditions. In order to accurately characterize the health risks from air toxics, it is necessary to use photokinetic (sunlight degradation) rate data to quantify the level of decomposition of the toxic emissions before human exposure.

The dominant pathway for human exposure to toxic emissions from motor vehicles is inhalation of exhaust emissions while seated in a moving vehicle. The major source of vehicle cabin exhaust emissions is other vehicles on the road in close proximity to the vehicle in question. A secondary pathway for exposure to motor vehicle toxic emissions is evaporative emissions which occur primarily during vehicle refueling. A study conducted by the Colorado School of Mines estimates that about 70 percent of the exposure to motor vehicle toxics occurs in the vehicle cabin, while evaporative emissions account for about 30 percent of the human exposure to toxic emissions from motor vehicles.
D. MTBE Health Effects

Although research conducted thus far does not demonstrate a causal association between MTBE exposure at ambient or occupational levels and reported acute health effects (headache, nausea, and sensory irritation), it is necessary to further investigate the acute health effects of oxygenates as a part of RFG mixtures. Research study emphasis should be placed on potentially “sensitive” individuals in primary exposure pathways (in vehicle cabins and while refueling) and workers who may be exposed to high concentrations of RFG. The Environmental and Occupational Health Sciences Institute will be releasing a report later this year on individuals from the “Oxybuster” citizens’ group which claims their members experience acute health effects from breathing MTBE at ambient concentrations. This study should help to inform and target future efforts in this area.

Research should also include human exposure studies to evaluate possible noncancer effects; acute, reversible neurotoxic effects (dizziness and/or spaciness); health effects associated with repeated exposure; and RFG/MTBE metabolism, particularly the increased risk associated with the metabolite by-products, formaldehyde and tertiary butyl alcohol which has a longer half-life and may accumulate in the body.

The following issues and areas need further study:
- a better understanding of how MTBE inhaled with RFG vapors is absorbed in the body and what the uptake rate is;
- how single versus repeated exposure affects RFG/MTBE metabolism; and
- if and why some individuals may have possible predisposing factors and an extreme sensitivity to RFG/MTBE (Health Effects Institute, 1997).

While animal data suggests that there is “limited animal evidence” for MTBE carcinogenicity (Office of Research and Development, EPA, 1993) and MTBE does not increase the cancer risk of RFG compared to conventional gasoline (Maine DEP, 1995) further study is needed, especially for MTBE inhaled with RFG vapors. The potential synergistic effects of MTBE and other gasoline components, such as benzene, should be investigated [e.g., determining the additive effects of two leukemogens such as benzene (confirmed) and MTBE (potential) (Mehlman, 1995)].
E. MTBE Fate and Transport

On a national basis, MTBE is the oxygenate of choice for reformulated gasolines because of its price and availability, transportation properties, high octane rating, distillation temperature depression (T50 and T90), and blending ability and subsequent dilution of undesirable components. It is some of these beneficial properties that when RFG/MTBE is released to the environment present an increased contamination potential when compared to conventional gasoline. Unlike the hydrocarbons in gasoline, MTBE is highly water soluble and poorly adsorbed to soils; therefore, it moves with the groundwater flow largely unretarded. MTBE resists biodegradation and thus increases remediation costs (California EPA, 1998).

In the air, MTBE partitions into atmospheric water. MTBE detected in snow samples supports the hypothesis that MTBE moves with water in the hydrologic cycle (US Geological Survey, 1995). Infiltration of precipitation and dispersion can transport MTBE from urban air to shallow groundwater. MTBE is expected to occur in precipitation in direct proportion to its concentrations in the air (Interagency Assessment of Oxygenated Fuels, EPA, 1997). Nonpoint sources may be the principle source of low concentrations of MTBE detected in ambient urban groundwater as suggested by the infrequent concurrent detection of MTBE with benzene, toluene, ethylbenzene, and xylene (BTEX) compounds (Squillace et al., 1997).

Potential sources of MTBE contamination include leaking underground storage tanks (UST), leaking pipelines, automobile and tanker truck accidents, and spills or exhaust from recreational boats and watercraft. It is not possible to accurately predict whether MTBE from leaking UST will affect deeper aquifers. Mitigation of leaking UST sites in close proximity to active drinking water supply wells may be necessary. The continued replacement and upgrading of USTs should minimize the potential for future MTBE contamination of groundwater. Studies of surface water reservoirs and lakes during the summer boating seasons should be conducted to determine the levels of MTBE (California EPA, 1998).

Further study is needed to understand:

- the behavior of MTBE in the atmosphere;
- the pathways by which MTBE enters groundwater; (i.e. how much does air deposition through precipitation of vehicular or industrial emissions contribute to surface and groundwater contamination);
- the processes by which it is transported and its long-term fate;
- degradation rates (particularly the half-life in water);
• degradation products (TBF, methyl acetate, acetone, TBA, and formaldehyde) and their health and ecological impacts; and
• the extent of exposed populations and ecosystems.

F. Conclusions

To better determine and understand public health and the environment benefits and potential disbenefits from the substitution of RFG for conventional gasoline, there is a need to further identify:

• possible adverse health effects from exposure to MTBE inhaled with RFG or from exposure to MTBE’s decomposition products and other species predicted to increase with RFG implementation;

• possible adverse health and ecological effects from contamination and persistence of MTBE in groundwater, reservoirs, and lakes; and,

• cost-effective MTBE remediation.

Findings from these studies will provide additional data to consider when comparing predicted air quality benefits to possible water quality detriment or other public health and ecological concerns. Possibly, other oxygenates (ethanol, TAME, and ETBE) should be considered and the public health and environmental risks associated with MTBE should be compared to these alternative oxygenates. However, there is limited data for the other oxygenates alone and inhaled with RFG (for e.g., inhalation studies for ethanol) and consequently, additional research is required for other oxygenates as well.

The RFG program is an important part of the national Urban Air Toxics strategy. A screening analysis of ambient monitoring data from forty urban areas throughout the nation have identified two chemicals targeted for reduction (benzene and 1,3-butadiene) by the RFG program as large contributors to the overall estimated cancer risk (USEPA, 1994). There is a need to carefully evaluate and document the reductions of mobile source air toxics through the ambient air monitoring network and the photochemical assessment monitoring stations (PAMS) that occur in areas which have adopted the winter and summertime RFG programs. This gathering and analysis of this information is a critical link in measuring the effectiveness of the RFG program as a year-round air toxics reduction strategy. It will also provide invaluable information for the development of a sound integrated urban air toxics strategy which will reduce individual exposures to ozone, carbon monoxide and hazardous air pollutants.
References


28. Personal Communication with Hari Rao, Methyl Tertiary Butyl Ether: Vapor inhalation oncogenicity study in CD 1 mice. October 1992


MTBE vapor inhalation carcinogenicity studies in F344 rats (Chun et al., 1992) and CD-1 mice (Burleigh-Flayer et al., 1992) and an oral gavage (olive oil) MTBE exposure study in Sprague-Dawley rats (Belpoggi et al., 1995) were considered for developing a preliminary cancer potency factor for this oxygenate present in Reformulated Gasoline (RFG). These experimental studies show that inhaled or ingested MTBE is carcinogenic in multiple animal species, with tumor responses seen at multiple organ sites. A qualitative description of the studies is provided, and the rationale for choosing the data set considered appropriate for potency calculation is presented. The methodology used to generate a potency estimate for MTBE is described. This potency estimate is compared with EPA's potency factors for benzene and 1,3-butadiene, which are other toxic/carcinogenic air contaminants associated with burning of gasoline, and which have different carcinogenic potentials. EPA's inhalation potency estimate for benzene is, however, derived from human data and the value is a geometric mean of four maximum likelihood point estimates (MLE) using pooled data and a one-hit model. For purposes of consistency in comparing potencies, and in evaluating relative potencies, this report derived a benzene potency factor (95% upper limit on MLE) using animal data and linearized multistage procedure. An estimate of the overall changes in carcinogenic risk resulting from burning different kinds of gasoline can be made by combining the potency factors with relevant exposure data.

Description of Chronic Studies

1. Chun et al., (1992) study: Four groups, each consisting of 50 F344 rats/sex, were exposed to MTBE vapor at 0 (control), 400, 3000, or 8000 ppm for 6 hr/day, 5 days/week, for 104 weeks except for male rats of the 3000 and 8000 ppm groups which were sacrificed at 97 and 82 weeks, respectively due to excessive mortality. The study reported increased numbers of renal tubular cell tumors and of interstitial cell adenomas (Leydig cell tumors) of the testes in male rats. The incidence of interstitial cell adenomas in male rats was 32/50, 35/50, 41/50, and 47/50 for the control, 400, 3000, and 8000 ppm groups, respectively. Testicular tumors, the study report pointed out, are a common lesion seen in F344 male rat and therefore, the increase in their incidence is difficult to evaluate. Furthermore, their frequencies were judged to be within the range reported for aged males and historical controls. However, a similar increase in incidence of interstitial cell tumors of the testes in the high dose group of male Sprague-Dawley rats orally exposed to MTBE was reported by Belpoggi et al., (1995). The evidence from these two independent studies suggests that MTBE may be a contributing factor to this carcinogenic effect.
2. Burleigh-Flayer (1992) study: Four groups, each consisting of 50 CD-1 mice/sex, were exposed for 6 hr/day, 5 days/week, for 78 weeks to MTBE vapor at 0 (control), 400, 3000, and 8000 ppm. Decreased survival, significant decrement in body weight gain at the highest exposure level were the reasons for the shortened duration of the study. In both sexes, the liver was the primary target organ for carcinogenic effects. An increased number of adenomas (10/50) was observed in the female mice from the 8000 ppm group, as compared to 2/50 from the control group. The incidence of liver tumors (adenomas and carcinomas) in the male mice was 16/49 for the 8000 ppm group and 12/49 for the control group. This increase was due to increased frequency of liver carcinomas (8/49) in the 8000 ppm group as compared to 2/49 in the control group.

3. Belpoggi et al., (1995) study: In this long-term carcinogenicity bioassay by the oral route, MTBE, dissolved in olive oil, was administered by gavage 4 days/week (Mon, Tues, Thurs, and Fridays) for 104 weeks at 0 (control), 250, 1000 mg/kg body weight doses to Sprague-Dawley rats/sex, 60 per group. The test animals were allowed to live out their lives and the last animal died at the age of 174 weeks. The study reported that MTBE gavage exposure produced a dose related increase in lymphomas and leukemia in female rats but not in male rats, and a statistically significant increased incidence of interstitial cell tumors of the testis (Leydig cell tumors) in the 1000 mg/kg group of male rats. It is of interest to note that no renal or CNS related anesthesia effects were observed even at the 1000 mg/kg high dose exposure.

Choice of Studies for Potency Calculation

Animal studies using inhalation route of exposure are more appropriate for the purpose of estimating a potency factor for MTBE, since human exposure to this oxygenate occurs primarily by inhalation. Therefore, the available inhalation studies were preferred for further consideration and analysis.

Since the potency factor is used to estimate a life-time risk of cancer in humans exposed to MTBE in ambient air, it is appropriate that the test animals be treated for at least 80% and observed for at least 90% of their average life span. The Chun et al., rat study (1992) satisfies this criterion (104 week exposure and observation). Data sets from this study were chosen for analysis. Since the CD-1 mice study duration was shortened to 78 weeks, a decision was made to use the findings of this study only in the MTBE hazard identification phase. The mouse liver tumor data were not utilized in quantitative calculations.

Note: MTBE has been detected in groundwater and so, there is a concern for human exposure to this chemical from drinking and other domestic uses of
the contaminated water. Regulatory guidelines are necessary to evaluate the nature of contamination and respond to the concerns of the public. Studies using the oral route of exposure are appropriate for purposes of estimating risk from consumption of MTBE in drinking water and for developing drinking water guidelines. Although there are uncertainties because of dosing rate and olive oil vehicle effects, the only available oral gavage study of Belpoggi et al., (1995 (104 week exposure and 174 week observation)) was utilized to calculate an oral potency factor. The oral potency factor was compared with the inhalation potency factor to determine if the estimates are consistent or substantially different.

Choice of Data Sets for Hazard Identification / Modeling

1. Rat Kidney Tumor Response Data: The usefulness of the rat kidney tumor data in calculating a cancer potency factor for MTBE has been questioned on mechanistic grounds. Whether the male rat kidney tumor response is mediated by 2u-globulin or some other protein mechanism is an unresolved issue. An Interagency Assessment of Oxygenated Fuels stated that "because evidence of an actual influence of 2u-globulin on the rat kidney tumor response is not established, the prudent public health approach is to use this tumor response for both hazard identification and quantitative estimation of cancer risk, yet we acknowledge that other views have been expressed on this subject. Users of this risk information should be cautious to recognize that the cancer potency value based on male rat kidney tumors shown later in this assessment could be substantially lower in magnitude, if 2u-globulin is having a meaningful influence (NSTC, 1997)". In view of the difficulties in evaluating the rat kidney tumor data, a decision was made, for purposes of this report, to use this tumor response only in the hazard identification phase, and not in the quantitative calculation of a cancer potency factor for MTBE.

2. Lymphoma/Leukemia Response data from Oral Study: A dose-related increase in Lymphoma/Leukemia response was noted in female Sprague-Dawley rats given MTBE by oral gavage, but not in male rats of this strain (Belpoggi et al., 1995). Also noteworthy is the lack of Lymphoma/Leukemia response in F344 rats exposed to MTBE by inhalation; this lack of response is perhaps masked by high spontaneous rate of mononuclear cell leukemia in this strain. Clearly, there is no consistency in the Lymphoma/Leukemia response to MTBE exposure. An additional uncertainty is the lack of data on the role of parent/metabolite compounds in eliciting this response, since it has been shown that formaldehyde, a metabolite of MTBE, has a leukemogenic effect. These uncertainties and the inconsistency in response limit the usefulness of the Lymphoma/Leukemia data for qualitative hazard determination only.
3. Male Rat Testicular Response Data from Inhalation and Oral Studies: A noteworthy finding is the increased incidence of interstitial cell tumors of the testis in high-dose Sprague-Dawley rats that were given MTBE by oil-gavage. This is consistent with the finding of an exposure-related increase in testicular tumors in F344 male rats that were exposed to MTBE by inhalation, although the latter strain is known for a high spontaneous rate of these tumors. That the parent compound, MTBE, and not its principal metabolite, TBA, is implicated in this type of testicular response is borne out by the lack of a tumor response in the testis of rats that were given TBA in drinking water. The consistency and specificity of the testicular response to inhaled or ingested MTBE, not clouded by metabolite issues, are the reasons for choosing male rat testicular response data from the inhalation study for mathematical modeling. For comparison, the testicular tumor data set from the oral study of Belpoggi et al., (1995) was also modeled.

Dose Adjustment and Modeling

Dose calculation: In the rat inhalation study in which MTBE exposure is reported in ppm units, ppm concentration is converted to mg/kg/day as follows:

\[
\text{Dose (mg/kg/day)} = ((\text{ppm}) \times (3.6 \text{ mg/m}^3 \text{ per ppm}) \times (\text{resp rate } 0.2667 \text{ m}^3 \text{ per day})
\]

\[
\times (\text{exp 6 hr/24hr}) \times (\text{exp 5 d/7d})) \div 0.35 \text{ kg rat body weight}
\]

\[
= (\text{ppm}) \times 0.49
\]

Assuming rat life-span to be 130 weeks, the doses in mg/kg/day for 400, 3000, 8000 ppm concentrations were adjusted for early termination of test by the factors \((104/130)^4\), \((97/130)^4\), and \((82/130)^4\), respectively. The adjusted doses 80, 456, and 621 mg/kg/day were modeled using the linearized multistage model and extra risk (global86).

The cancer potency estimate for MTBE is based on the assumption that dose-response relationships are linear and that similar mechanisms of tumor induction can occur in both rats and humans. The input to the model and the model output are shown below:
1. MTBE Male F344 Rat Testicular Data (Inhal) for Global86 Modeling

<table>
<thead>
<tr>
<th>Group</th>
<th>Adjusted Dose</th>
<th># Rats with Tumors /# Animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.0</td>
<td>32 /50</td>
</tr>
<tr>
<td>2</td>
<td>80.0</td>
<td>35 /50</td>
</tr>
<tr>
<td>3</td>
<td>456.0</td>
<td>41 /50</td>
</tr>
<tr>
<td>4</td>
<td>621.0</td>
<td>47 /50</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group</th>
<th>Predicted</th>
<th>Observed</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.653004</td>
<td>0.640000</td>
</tr>
<tr>
<td>2</td>
<td>0.683241</td>
<td>0.700000</td>
</tr>
<tr>
<td>3</td>
<td>0.824511</td>
<td>0.820000</td>
</tr>
<tr>
<td>4</td>
<td>0.939195</td>
<td>0.940000</td>
</tr>
</tbody>
</table>

CHI-SQUARE GOODNESS OF FIT STATISTIC IS .109803
P-VALUE FOR THE CHI-SQ TEST WITH 1 DEGREES OF FREEDOM IS 0.74

MAXIMUM LIKELIHOOD ESTIMATES OF DOSE COEFFICIENTS

Q( 0) = 1.05844325774
Q( 1) = 1.139586942975E-03
Q( 2) = 0.00000000000
Q( 3) = 0.00000000000
Q( 4) = 0.00000000000
Q( 5) = 0.00000000000
Q( 6) = 1.802810011958E-17

MAXIMUM VALUE OF THE LOG-LIKELIHOOD IS -98.187
CALCULATIONS ARE BASED UPON EXTRA RISK
GLOBAL 86 LOWER CONFIDENCE LIMITS ON DOSE FOR FIXED RISK

CONFIDENCE LIMIT FOR A RISK OF 1.000000E-06
THE M.L.E. OF DOSE IS 8.775114E-04
THE 95.0 PERCENT LOWER LIMIT ON DOSE IS 3.452480E-04

THE COEFFICIENTS CORRESPONDING TO THE 95.0 % BOUND ARE:
Q(0) = 0.928586913623
Q(1) = 2.896469787588E-03 (inhalation potency factor, per (mg/kg)/day)
Q(2) = 0.000000000000
Q(3) = 0.000000000000
Q(4) = 0.000000000000
Q(5) = 0.000000000000
Q(6) = 0.000000000000

2. MTBE Male Sprague-Dawley Rat Leydig Cell Tumor Data (Oral)

<table>
<thead>
<tr>
<th>Group</th>
<th>Dose (mg/kg/day)</th>
<th># Responses /# Animals (Corrected)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.000</td>
<td>2 / 26</td>
</tr>
<tr>
<td>2</td>
<td>143.000</td>
<td>2 / 25</td>
</tr>
<tr>
<td>3</td>
<td>571.000</td>
<td>11 / 52</td>
</tr>
</tbody>
</table>

* Dose adjustment factor: gavage dose * 4d/7d

<table>
<thead>
<tr>
<th>Group</th>
<th>Predicted</th>
<th>Observed</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7.692308E-02</td>
<td>7.692308E-02</td>
</tr>
<tr>
<td>2</td>
<td>8.000000E-02</td>
<td>8.000000E-02</td>
</tr>
<tr>
<td>3</td>
<td>0.343750</td>
<td>0.343750</td>
</tr>
</tbody>
</table>

CHI-SQUARE GOODNESS OF FIT STATISTIC IS 2.630751E-24
MAXIMUM LIKELIHOOD ESTIMATES OF DOSE COEFFICIENTS

Q(0) = 8.004270767348E-02
Q(1) = 3.792749827419E-06
Q(2) = 6.157958861109E-08
Q(3) = 1.289926762076E-10
Q(4) = 2.774276124762E-12
MAXIMUM VALUE OF THE LOG-LIKELIHOOD IS -34.6118876899
CALCULATIONS ARE BASED UPON EXTRA RISK
GLOBAL 86 LOWER CONFIDENCE LIMITS ON DOSE FOR FIXED RISK CONFIDENCE LIMIT FOR A RISK OF 1.000000E-06
THE M.L.E. OF DOSE IS 0.262541
THE 95.0 PERCENT LOWER LIMIT ON DOSE IS 1.134194E-03
THE COEFFICIENTS CORRESPONDING TO THE 95.0 % BOUND ARE:

\[
\begin{align*}
Q(0) &= 4.615172362548E-02 \\
Q(1) &= 8.81683736146E-04 \text{ (oral potency factor, per (mg/kg)/day)} \\
Q(2) &= 0.000000000000 \\
Q(3) &= 0.000000000000 \\
Q(4) &= 0.000000000000 \\
\end{align*}
\]

The calculated inhalation and oral potency factors for MTBE, 2.89E-03 and 8.81E-04 per (mg/kg)/day, respectively are comparable. The relatively close agreement of the calculated values across routes of exposure for male rats of different strains is impressive. The potency estimate for MTBE, 1.59E-03 per (mg/kg)/day, is the geometric mean of the two potency values calculated above. Based on this potency value, the theoretical risk associated with a continuous life-time exposure to MTBE at 1 ug/m3 (inhalation unit risk) is estimated to be 4.5E-07. A concentration of 2.2 ug/m3 MTBE is associated with one in a million risk for a life-time exposure.

EPA has calculated unit risk estimates for benzene and 1,3-butadiene as 8.3E-06 and 2.8E-04 per (ug/m3), respectively. The unit risk estimate for MTBE, 4.5E-07 per (ug/m3), indicates that, among these three constituents of gasoline, MTBE has the lowest carcinogenic potential. A comparison of the unit risk estimates shows that MTBE is 18-fold, and 622-fold less potent than benzene and 1,3-butadiene, respectively.

EPA’s potency estimate for benzene, as indicated before, is based on human data and is a geometric mean of four ML point estimates of pooled data. The potency estimates for MTBE and 1,3-butadiene are, however, derived from animal data. For purposes of consistency in potency comparison, a potency estimate for benzene was calculated using the NTP rat gavage (1985) data as shown below:

**Benzene Male Rat Squamous Cell Carcinoma NTP (1985)**

<table>
<thead>
<tr>
<th>Group</th>
<th>Dose</th>
<th># Responders /# Animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.000</td>
<td>0 / 48</td>
</tr>
<tr>
<td>2</td>
<td>14.6000</td>
<td>10 / 50</td>
</tr>
<tr>
<td>3</td>
<td>29.3000</td>
<td>8 / 50</td>
</tr>
<tr>
<td>4</td>
<td>58.5000</td>
<td>16 / 50</td>
</tr>
</tbody>
</table>

A-7
<table>
<thead>
<tr>
<th>Group</th>
<th>Predicted</th>
<th>Observed</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>.000000</td>
<td>.000000</td>
</tr>
<tr>
<td>2</td>
<td>.106207</td>
<td>.200000</td>
</tr>
<tr>
<td>3</td>
<td>.201748</td>
<td>.160000</td>
</tr>
<tr>
<td>4</td>
<td>.362303</td>
<td>.320000</td>
</tr>
</tbody>
</table>

CHI-SQUARE GOODNESS OF FIT STATISTIC IS 5.56206
P-VALUE FOR THE CHI-SQ TEST WITH 2 DEGREES
OF FREEDOM IS .6197472529E-01
MAXIMUM LIKELIHOOD ESTIMATES OF DOSE COEFFICIENTS

\[
\begin{align*}
Q(0) &= 0.00000000000000 \\
Q(1) &= 7.690458646068E-03 \\
Q(2) &= 0.00000000000000
\end{align*}
\]

CALCULATIONS ARE BASED UPON EXTRA RISK
CONFIDENCE LIMIT FOR A RISK OF 1.000000E-06
THE M.L.E. OF DOSE IS 1.300313E-04
THE 95% LOWER LIMIT ON DOSE IS 9.917850E-05
THE COEFFICIENTS CORRESPONDING TO THE 95% BOUND ARE:

\[
\begin{align*}
Q(0) &= 0.00000000000000 \\
Q(1) &= 1.008283547932E-02 \quad \text{(potency factor per (mg/kg)/day)} \\
Q(2) &= 0.00000000000000
\end{align*}
\]

The benzene potency factor of 1.008E-02 per (mg/kg)/day based on rat data is three-fold lower than EPA's oral slope factor of 2.9E-02 per (mg/kg)/day based on human data. The close agreement between these two potency factors for benzene is noteworthy. Benzene potency estimate based on animal data is still about an order of magnitude higher than MTBE potency estimate. This type of analysis provides additional support to the potency ranking of the three toxic air contaminants associated with combustion of gasoline, and confirms their potency rank order: MTBE < benzene < 1,3-butadiene.
References


Appendix B

Derivation of the Unit Risk Factor for Polycyclic Organic Matter

Polycyclic organic matter (POM) is a complex mixture of polycyclic organic compounds including several classes of hydrocarbons, substituted aromatic hydrocarbons, and heterocyclic, aromatic compounds. The first compounds recognized to be human and animal carcinogens were coal tars and coal chimney soot. The cancer was subsequently found to be related to the presence of POM. POM is emitted from different sources due to combustion such as petroleum compounds, wood and plastics. A primary carcinogen in the POM is the polycyclic aromatic hydrocarbon benzo(a)pyrene but B(a)P in itself is not sufficient to predict the carcinogenic potency of POM.

The presence of other carcinogens in the mixture also contribute to the unit risk of POM. Because the content of these products apparently can vary between POM from different sources it is necessary to determine source specific cancer unit risk estimates. Potencies of different POM for human lung cancer ranges from lows of 3E-06 for cigarette smoke to a high of 1E-03 for coke oven emissions. In other words one cancer for every million persons exposed to 3 cancers for every thousand persons exposed.

Thus while it is known that POM is carcinogenic the severity of the risk is affected by the source which is affected by the content of different carcinogenic polycyclic hydrocarbons. Because the variation is so great it is necessary to determine the potency for each source of POM.

Two approaches were considered for the RFG relative risk assessment potency estimate for POM. One followed the methodology outlined in the paper by Lewtas, 1993 Complex Mixtures of Air Pollutants: Characterizing the cancer risk of Polycyclic Organic Matter, Environmental Health Perspective (100) 211-218 and the second a methodology described by Meek, Chan and Bartlett (1994), Polycyclic Aromatic Hydrocarbons: Evaluation of risks to health from environmental Exposure in Canada, Environmental, Carcinogenic and Ecotoxicological Reviews c12(2), 443-452. The former approach uses a mouse skin painting test to infer the cancer potency of POM from differing sources while the latter measures to content of known carcinogens in the mixture and measures the risk in terms of B(a)P equivalents. Neither method is ideal but each provides a rough estimation of the cancer potency of POM from gasoline exhaust.

Mouse Skin Painting Approach

Response to the skin painting studies determined for several compounds were plotted against the human lung cancer risk for a series of compounds some of which have cancer unit risk estimates. The human lung
cancer potency for the unknown compounds was inferred from the results of
the skin painting study to be 6.0 E-5.

Estimate Based on Concentration of Known Carcinogenic PAHs

The latter paper calculated to unit risk for exhaust from diesel fuels
based on the content of B(a)P and other PAH's. the cancer risk of the other
PAHs was expressed in terms of B(a)P equivalents. Because the exhaust from
diesel vehicles is quite different from that of vehicles using conventional
gasoline the proportion of the PAHs in exhaust from conventional fuels was
used. This information was provided by EPA from a study of on-the-road
automobiles in several locations in the US. The preliminary finding from
the EPA survey indicated that the average POM emissions are 46 mg/mile
and that about 5 mg/mile is PAH's of which 0.5 µg/mile is B(a)P and 0.02
µg/mile is B(a)P equivalents. Thus the carcinogenic potency of the overall
POM is about 2 to 3 fold less than B(a)P itself or about 2.0 E-05.

Therefore the POM unit cancer risk is estimated to be between 2.0 E-05
and 6.0 E-5. Because the potency in the skin painting study may reflect the
presence of other carcinogens such as benzene and 1,3-butadiene which are
counted elsewhere in this relative risk assessment and because the study
measured cancer produced by skin painting instead of internal bioassays as
was used elsewhere in the relative risk assessment it was decided to use the
value 2.0 E-05. Because there are potentially other carcinogens present not
used in the calculation it is probable that the cancer risk for POM may be
somewhat underestimated.

References

Lewtas J. 1993. Complex mixtures of air pollutants: characterizing the cancer
risk of polycyclic organic matter. Environmental Health Perspectives 100
211-218.

Hydrocarbons: Evaluation of risks to health from environmental exposure
in Canada. Environmental Carcinogenicity and Ecotoxicity Reviews 12 (2)
443-452.