Control of Hazardous Air Pollutants from Mobile Sources

Summary and Analysis of Comments

Chapter 2
Environmental/Air Quality and Public Health Impacts

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

United States
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Agency

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2. ENVIRONMENTAL/AIR QUALITY AND PUBLIC HEALTH IMPACTS

What We Proposed:

The comments in this section correspond to Sections III through V of the preamble to the proposed rule, and Chapters 1 through 3 of the Regulatory Impact Analysis. They are therefore targeted at the environmental, air quality, and public health impacts from the proposal. A summary of the comments received, as well as our response to those comments, are located below.

For the full text of comments summarized here, please refer to the public record for this rulemaking.

2.1 Public Health Issues

2.1.1 Public Health Justification and Implications for Controls

What Commenters Said:

A number of commenters stated that mobile source air toxics pose a significant risk that justifies the proposed controls, and that EPA should do even more to reduce emissions. A summary of these comments follows.

A private citizen stated that benzene causes a significant threat to human health and if it is possible to reduce the amount of benzene being released into the environment, then it should be done. The comments address the harmful side effects of benzene and six other chemicals found in gasoline. If the levels of these chemicals in gasoline were reduced, the commenter argues that there would be a significant savings in health care.

The Northeast States for Coordinated Air Use Management (NESCAUM) commented that it believes that both onroad and nonroad mobile sources such as cars, trucks, buses, construction equipment, lawn and garden equipment, snowmobiles, and boats emit pollutants that cause cancer or other adverse health effects. The commenter further stated that it believes that mobile source air toxics clearly pose a significant public health threat in the northeastern U.S. and public exposure to toxic emissions from mobile sources is a major concern to health officials and air quality regulators in the Northeast. They cite emissions inventory and air quality monitoring and modeling data indicating that 50 and 74 percent of cancer and non-cancer risk related to breathing outdoor air results from mobile source air toxics emissions. The commenter further noted that Northeast state modeling and monitoring data indicate that ambient concentrations of acetaldehyde, benzene, 1,3-butadiene, formaldehyde, acrolein, and diesel particulate matter exceed risk screening thresholds for cancer and, in some cases, non-cancer effects throughout the region. The commenter stated that a review of emissions inventory data concluded that mobile sources dominate the primary emissions for these pollutants in all Northeast states.

NESCAUM commented that it believes that additional reductions in benzene are needed because even the simplest risk assessment predicts that exposures to benzene (directly and

indirectly from the use of mobile sources) are very high throughout the US. The commenter noted that it was stated in the RIA (p.3-48) that "...based on average census tract risks, the vast majority of the population experiences risks between one in a million ($1x10^{-6}$) and one in ten thousand ($1x10^{-4}$)". However, the commenter noted, the number of people experiencing risks above one in a hundred thousand ($1x10^{-5}$) increases from 214 million in 1999 to 240 million in 2030. NESCAUM commented that, based on the experiences of the Northeast states with monitoring, modeling, and controlling air toxics in the Northeast, it believes that the need for more reductions in MSAT emissions is evident. The commenter cited the example of monitoring data for Burlington, VT for 1999 which it stated documents that ambient air concentrations of benzene exceeded health benchmarks (10^{-6} cancer risk) by roughly a factor of 20. The commenter noted that, consequently, an urban-scale benzene modeling study was applied to the Burlington area for 1999. The commenter stated that this study demonstrated that annual ambient concentration impacts modeled in Burlington from motor vehicles over the whole domain were anywhere from 5 to 20 times the Vermont health standard (0.12 $\mu g/m^3$) for benzene. Seventy six percent of this modeled local source annual benzene impact was due to motor vehicle traffic.

NESCAUM also provided conclusions from recent studies of microenvironment exposure levels in the Northeast, and stated these analyses provide evidence of the need for substantial reductions in mobile source air toxic emissions. The commenter also stated that, beyond the risk quantified in the national-scale modeling in the proposal's Regulatory Impact Analysis, there are many more risks from exposure to MSAT that have not been quantified. The commenter stated that it believes this increases the urgency for additional MSAT reductions in the Northeast states.

STAPPA and ALAPCO noted that the proposal stated that 68 percent of our nation's benzene emissions come from mobile sources and that benzene will continue to be the key cancer risk driver into the future. The commenters stated that they acknowledge EPA's effort to stem this risk, but believe that more can and should be done and that they strongly urge EPA to maximize this opportunity to glean the greatest benzene reductions possible.

The Oregon Department of Environmental Quality (ODEQ) and Lane Regional Air Protection Agency (LRAPA) commented that the EPA National-scale Air Toxics Assessment (NATA) indicates that benzene is the most significant air toxic for cancer risk and that mobile sources are the major source of benzene. The commenters asserted that benzene concentrations in the Pacific Northwest have been among the highest in the nation. LRAPA further stated that it believes that the MSAT rule revisions are the greatest opportunity to reduce benzene to safe levels.

The New York State Department of Environmental Conservation (NYDEC) commented that in its study of concentrations and trends of benzene in ambient air over New York during 1990-2003, a 50 percent or more decline in mean annual concentrations of benzene was demonstrated. The commenter stated that it believes that this downward trend in benzene concentrations can be attributed partly to: 1) the adoption of the Reformulated Gasoline Program (RFG) in 1-hr ozone non-attainment areas; and, 2) for other non-RFG sites, improvements in vehicle emissions technology and the statewide adoption of the California Low Emissions

Vehicle (LEV) program. The commenter further stated that it believes that an examination of the information included in this rulemaking indicates this proposal will not provide any real meaningful reductions of MSATs in the New York City Metropolitan area over the next 24 years and the predicted cancer risk estimates will remain steady or actually increase as a result of this rulemaking. Similarly, NESCAUM stated that it believes that this rulemaking will only provide small reductions in MSAT risk in areas that currently use RFG and have adopted the California LEV program. NESCAUM stated that it believes that an examination of the information included in this rulemaking indicates that the predicted cancer risk estimates in these areas will remain steady or actually increase between now and 2030.

The New Jersey Department of Environmental Protection (NJDEP) commented that it believes that "all risk assessments" predict that exposures to benzene, attributed directly and indirectly to mobile sources are high. The commenter cited the Draft RIA (p.3-48) in describing the population exposed to various risk levels ($1x10^{-6}$ – $1x10^{-4}$) in 1999 and in 2030. NJDEP also summarized risk characterization data from the 1999 National Air Toxics Assessment and concerns about exposures attributable to attached garages and residence near major roads, commenting that the information supported the conclusion that emissions of hazardous air pollutants from mobile sources is a "very serious problem." The commenter also summarized its own analyses of 1996 NATA results, indicating that mobile sources are the predominant source of several air toxics in New Jersey.

The Wisconsin Department of Natural Resources (WDNR) commented that it believe that mobile source air toxics must be addressed in order to ensure that communities are as healthy as possible. The commenter also expressed concern about its impression that EPA is using benzene as a "the only surrogate" of mobile source air toxics in general.

Environmental Defense, the Natural Resources Defense Council (NRDC), U.S. Public Interest Research Group (PIRG), and the American Lung Association (ALA) commented that they believe that exposure to benzene presents a serious risk to human health. The commenters further stated that they believe that benzene is responsible for carcinogenic and non-carcinogenic health effects through all routes of exposure, and is found in considerable concentrations in communities throughout the United States. The commenters noted that in 1999, 68 percent of benzene emissions nationwide were from mobile sources, and stated that in the coming years they believe that mobile sources will continue to be a major source of benzene.

The Alliance of Automobile Manufacturers (Alliance) stated its support for EPA's initiative to reduce mobile source air toxics, and indicated that it understands the goal of a healthier environment

ExxonMobil and the American Petroleum Institute (API) commented that they view the health justification for the proposal as inadequate. ExxonMobil further stated that the discretion afforded EPA in CAA 202 (l) should only be exercised after an adequate health based justification. The commenters also asserted that cancer risk due to benzene from mobile sources could fall below a *de minimis* risk level before implementation of the fuels program, especially if the lower range, or possibly even the midpoint of the range of "equally scientifically valid" unit risk estimates is used to calculate benefits.

Letters:
Alliance of Automobile Manufacturers (Alliance) OAR-2005-0036-0881
American Petroleum Institute (API) OAR-2005-0036-0884
Environmental Defense, NRDC, U.S. PIRG, ALA OAR-2005-0036-0868
ExxonMobil Refining & Supply Company OAR-2005-0036-0772
Lane Regional Air Protection Agency (LRAPA) OAR-2005-0036-0848
New Jersey Department of Environmental Protection OAR-2005-0036-0829
New York State Department of Environmental Conservation (NYDEC) OAR-2005-0036-0722
NESCAUM (Northeast States for Coordinated Air Use Management) OAR-2005-0036-0993
Oregon Department of Environmental Quality (ODEQ) OAR-2005-0036-0987
STAPPA/ALAPCO OAR-2005-0036-0836
West Chester University of Pennsylvania Student OAR-2005-0036-0368
Wisconsin Department of Natural Resources (WDNR) OAR-2005-0036-0828

Our Response:

Section 202 (l) (2) requires EPA to adopt technology-based (indeed, technology-forcing) regulation of air toxics from motor vehicles to the maximum extent achievable, taking into consideration cost, energy, noise, safety, and lead time. See Sierra Club v. EPA, 325 F. 3d at 378 (section 202 (l) (2) is technology-forcing and so requires EPA to consider future advances in pollution control capability, among other factors). It is not a risk-based provision whereby the reasonableness of the rule is determined by evaluating whether some measure of risk reduction is achieved. Section 202 (l) (2) also states, however, that the rules for air toxics are to be promulgated under sections 202 (a) (1) (vehicles) and 211 (c) (1) (fuels). Both provisions contain certain risk-based condition precedents to exercise of the technology-based authority. This condition precedent is a showing that emissions (or, for fuels, emission products) "cause, or contribute to, air pollution which may reasonably be anticipated to endanger public health or welfare." We believe the information presented in the preamble and the RIA support such a judgment, and agree with the comments that concurred with this determination. We disagree with the comment that the health justification is inadequate to move forward with technology-based regulation.

While we agree that the mobile source air toxics increase the risk of health effects and that risks will remain after the rule is implemented, we believe the rule achieves the "greatest degree of emission reduction achievable through the application of technology which will be available, taking into consideration the motor vehicle standards established under section 202(a) of the Act, the availability and cost of the technology, and noise, energy and safety factors, and lead time" (Clean Air Act section 202(l)).

In response to the comment that cancer risk due to benzene from mobile sources could fall below a de minimis risk level before implementation of the fuels program , unlike other provisions of the Clean Air Act, section 202(l)(2) does not direct EPA to set standards to eliminate or achieve a certain level of risk. Nevertheless, EPA would like to note that in setting a

NESHAP (National Emission Standards for Hazardous Air Pollutants) for benzene in 1989 (FR 54, 177: 38044-38072; September 14, 1989), a de minimus risk level was not defined based on average population risk. Acceptability of risks in a population were based on the individual exposed to the maximum level. If average risks are below the one in a million level, a significant number of people in the population could still be judged to have "unacceptable" risks under the criteria set in this NESHAP.

We note that comments indicating that we are using benzene as the "only surrogate" of mobile source air toxics more generally are incorrect. The standards in this rule are expected to reduce emissions of a broad range of particulate and gaseous air toxics other than benzene.

We disagree that there will be no meaningful reductions of MSATs in the New York City metropolitan area or areas that currently use RFG and have adopted the California LEV program. The vehicle standards add cold temperature emission standards to existing requirements nationally, and these standards address lower temperatures than California's LEV standards, which extend to only 50 degrees Fahrenheit. As a result, substantial reductions in cold-temperature emissions are expected to accrue even in areas that have adopted the California LEV program, with concurrent reductions in risk. Furthermore, the portable fuel container emission standard will provide substantial reductions in MSAT emissions and exposures.

2.1.2 Benzene

What Commenters Said:

Based on material cited in Chapter 3 of the RIA, the New Jersey Department of Environmental Protection asserted that benzene may be even more potent than EPA's IRIS (Integrated Risk Information System) database suggests. In particular, the commenter noted the RIA's note that the cancer dose-response curve for benzene may be "supralinear" and that benzene metabolism may be saturated at 1ppm. The commenter noted the RIA's statement that health effects from occupational exposures to benzene have been seen below the 1ppm level.

API and ExxonMobil commented, with the endorsement of Marathon Petroleum Company, that they believe that in the proposal, EPA overstated the predicted health benefits of the rule, and as such, the benzene content standard for gasoline is not adequately justified. They first noted that while benzene is identified as a national risk driver under the 1999 NATA, predicted increases in the number of people exposed to higher risk levels are due to population growth, rather than increased benzene emissions.

API and ExxonMobil comment that EPA provided an unbalanced view of the science regarding benzene's health effects, and that benzene risks are equally if not more likely to be overestimates than underestimates. In particular, the commenters assert that EPA made several questionable or incorrect statements in discussing how its risk assessment could underestimate benzene risk. Specifically, they suggest that EPA's following assertions are questionable or incorrect:

- EPA's mention of the possible supralinearity of benzene's dose-response function at environmental exposure levels, noting that even if one metabolic pathway

saturates in the 1 ppm range, other processes of detoxification and clearance could compensate and reduce risk above that level of exposure;
- benzene's association with more than one leukemia subtype;
- that a transition from linear to saturable metabolism below 1 ppm could result in underestimation of risk;
- assertion that the National Cancer Institute (NCI) Chinese cohort study as not having undergone review under Integrated Risk Information System (IRIS) or consideration for establishing the benzene unit risk estimate.

They commented that EPA did not consider "reality checks," such as the absence of benzene health effects in populations exposed to higher concentrations than the general public, such as petroleum workers, for which they cited occupational epidemiology studies. In particular, they cited a meta-analysis of petroleum worker epidemiology studies in which relative risks for leukemia were near 1 (Wong and Raabe, 1995). They also cite the lack of consistency in epidemiologic studies of gas station attendants and vehicle mechanics. Accordingly, they asserted that the true risk for benzene induced disease from current environmental exposures could be zero, and cite EPA's use of such language in describing risk from diesel exhaust exposure.

Letters:
American Petroleum Institute (API) OAR-2005-0036-0884
ExxonMobil Refining & Supply Company OAR-2005-0036-0772
New Jersey Department of Environmental Protection, Division of Air Quality OAR-2005-0036-0829
Marathon Petroleum Company (MPC) OAR-2005-0036-0946

Our Response:

We disagree that the Agency provided an unbalanced view of the science regarding benzene health effects.

EPA believes that the best currently available peer-reviewed published scientific information/data sources on health effects of benzene should be used for health hazard or risk determination. EPA's preferred choice of source for this determination remains the currently available EPA IRIS values. When EPA IRIS assessments are updated, EPA uses emerging peer-reviewed scientific literature and/or other assessments in support of hazard risk determination. A brief summary of emerging science is provided below.

EPA (IRIS, 2000) concluded that "some recent evidence suggests the possibility that the low-dose curve could be supralinear since the formation of toxic metabolized plateaus above 25 ppm benzene in air. Thus, it is possible that the unit risk is underestimated if linearity is assumed at low doses." Furthermore, EPA concluded that "there is no sufficient evidence currently to reject a linear dose-response curve for benzene in the low-dose region, nor is there sufficient evidence to demonstrate that benzene is, in fact, nonlinear in its effects." In absence of this information, EPA recommended a default approach of using a model with low-dose linearity and

estimated the risk at 1 ppm ranging from $7.1x10^{-3}$ to $2.5x10^{-5}$, within which any calculated unit risk estimate would have equal scientific validity. For risks ranging from $1x10^{-4}$ to $1x10^{-7}$, the corresponding air concentrations for lifetime exposure range from 0.013 to 13.0 $\mu g/m^3$ (0.004 - 4 ppb) using the higher end of the unit risk range to 0.045 to 45.0 $\mu g/m^3$ (0.014 – 14 ppb) using the lower end of the unit risk range.

Since the revision to the IRIS assessment for benzene, additional research has been published. Commenters made note of some recent studies (e.g. Wong and Raabe, 1995; Ruston and Romaniuk, 1997; Schnatter et al, 1996; Glass et al, 2003). However, other recent studies support a supralinear dose-response relationship between exposure and cancer risk for lower exposure levels. For example, Rappaport et al. (2005) and Lin et al. (2006) characterized relationships between levels of albumin adducts of benzene metabolites in blood and the corresponding benzene exposures in benzene-exposed and control workers, after adjusting for important covariates.[1,2] The levels of reactive and hematotoxic benzene metabolites were less than proportional to benzene exposure at air concentrations in the range of 1 to 10 ppm. Another example is work by Rappaport et al., (2002), which indicates deviations from linearity beginning at approximately 1 ppm.[3] These examples would imply that linear fits of leukemia mortality among occupationally exposed workers to hundreds of ppm of benzene could possibly underestimate risks from benzene metabolites in persons exposed at lower (non-saturating) air concentrations. All of these studies will be evaluated when EPA reconsiders health risks from exposure to benzene.

The commenters also hypothesize that additional mechanisms of metabolism may mitigate risk, even if enzymes saturate at around 1 ppm. While it is always possible some unidentified metabolic pathway could impact risk, we are aware of no existing data to either refute or support this hypothesis. EPA will continue to monitor research regarding low-dose metabolism of benzene in humans.

As for the comments on the possible association of benzene with more than one leukemia subtype, it should be recognized that while most of the epidemiological studies are generally limited by confounding chemical exposures, and methodological problems, the overwhelming evidence is consistent for excess risk of leukemia across studies. It should be recognized that earlier studies are limited by a lack of information on leukemia cell types other than AML, because leukemia used to be considered a single diagnostic category for epidemiological investigation, partly because of historical nomenclature, small number of deaths by cell type, and unavailability of cell-type specific rates for comparison.

The commenters selectively cite certain epidemiological publications referring to "[e]vidence of uncertainties include inconsistent results from epidemiological studies (Ruston and Romaniuk, 1997; Schnatter et al, 1996; Glass et al, 2003),[4,5,6] "relative risk near unity" (Wong and Raabe, 1995)[7] and "Studies on CLL and benzene show equivocal results as some recent reviews have revealed" (Schnatter et al., 2005; Linet et al, 1996).[8,9] Inconsistencies across the epidemiological studies are to be expected given the uneven power and quality of the studies with respect to their ability to provide meaningful information on the existence of cancer risks from benzene exposure. The more informative studies show increased risks of leukemia. In contrast with the commenter's view that EPA did not consider "reality checks," such as the

absence of benzene health effects in occupationally exposed populations and the comment that the results of epidemiologic studies of leukemia in some worker groups are inconsistent, on the contrary, the preponderance of evidence based on epidemiological studies and case reports clearly indicates a causal relationship between occupational exposure to benzene including benzene-containing solvents and the occurrence of acute nonlymphocytic leukemia (ANLL), particularly, the myeloid cell type (acute myelogenous leukemia, AML) (Rinsky et al, 1987, 2002; Yin et al, 1996; Hayes et al, 1997).[10,11,12,13] Several of the studies also provide suggestive evidence of association between benzene exposure and non-Hodgkin's lymphoma (NHL) and multiple myeloma (Hayes et al, 1997; Rinsky et al, 1987)' and to some extent chronic nonlymphocytic leukemia (CNLL) as well as chronic lymphocytic leukemia (CLL) (Vighani and Saita, 1964; Aksoy, 1976, 1977; Infante et al, 1977; Rinsky, 1981, 1987).'[14,15,16,17,18] Although the Pliofilm study (Rinsky et al, 1987) provided estimates of leukemia risk at high levels of benzene exposure, The NCI-CAPM (National Cancer Institute/Chinese Academy of Preventative Medicine) study (Hayes et al, 1997, 2001) extended estimates of risk to lower levels of exposure below 10 ppm.'[19] In spite of the recognition that the NCI-CAPM study and all other retrospective investigations have limitations, the criticisms raised by Wong (1999) and Budinsky et al (1999) do not negate the findings that significantly elevated risks for lymphohematopoietic disorders occurred at substantially lower levels of benzene exposure than in the study of Rinsky et al (1987).[20,21]

Rushton and Romaniuk (1997) investigated the risk of leukemia in workers in the petroleum distribution industry who are exposed to low levels of benzene. Although they reported no significant increase in the overall risk of all leukemia with higher cumulative exposure or with intensity of exposure, the authors also noted that the risk was consistently doubled in subjects employed in the industry for more than 10 years. There is a suggestion of a relation between exposure to benzene and myeloid leukemia, in particular for acute myeloid and monocytic leukemia. Risk was increased to an odds ratio (OR) of 2.8 for a cumulative exposure between 4.5 and 45 ppm-years compared with 0.5 ppm-years. The Glass et al. (2005) study suggests that benzene exposure is associated with a spectrum of hematologic neoplasms and related disorders in humans.[22] Risks for these conditions are elevated at average benzene-exposure levels of less than 10 ppm. The Glass et al. (2003) study found an excess risk of leukemia associated with cumulative benzene exposures and benzene-exposure intensities that were considerably lower than reported in previous studies.[23] They also concluded that no evidence was found of a threshold cumulative exposure below which there was no risk. A recent review of the literature of nine cohort and 13-case control studies on benzene exposure and leukemia subtypes (Schnatter et al., 2005) concluded that high and significant acute myeloid leukemia risks with positive dose-response relationships were identified across study designs. Risks for CLL tended to show elevations in nested case-control studies, with possible dose-response relationships in at least two of the three studies. However, data on chronic myeloid leukemia and acute lymphocytic leukemia are sparse and inconclusive.

Regarding the comments that a description of the risk of benzene should contain a note that the risks could actually be zero, no such language is found in EPA's IRIS summary or supporting documents for benzene, and making such a statement would be highly speculative as well as unsupported by the preponderance of published literature. In contrast to compounds for which the human carcinogenicity is not known with certainty, benzene is known to be

carcinogenic to humans, and environmental exposure levels are not so far below the occupational exposure levels exhibiting increased cancer risks that there is any expectation of zero cancer risk given the genotoxic properties of benzene.

For these reasons, EPA disagrees with API and others that EPA overstates the health benefits related to benzene control. However, EPA acknowledges using the upper end of the maximum likelihood range in assessing cancer risk attributable to benzene exposure. This is consistent with the way benzene risk is modeled in the National Air Toxics Assessment. It is important to note that the rule is not justified based on some (asserted) level of risk, as EPA again notes that it is reasonably interpreting section 202(l)(2) as requiring standards which are technology-based (taking into consideration cost, energy, safety and other enumerated factors, along with technical feasibility), not risk-based.

What Commenters Said:

ExxonMobil commented that because ambient concentrations of benzene lie below the reference concentration (RfC) of 30 $\mu g/m^3$, no non-cancer health effects should be discussed as possible health effects of environmental benzene exposures.

Letters:
ExxonMobil Refining & Supply Company OAR-2005-0036-0772

Our Response:

The commenter inappropriately cites only ambient concentrations of benzene as the context for discussion of non-cancer health effects. An RfC refers to a time-weighted exposure concentration, rather than an ambient concentration. As discussed in Chapter 3 of the RIA, indoor and personal breathing zone concentrations of benzene can be substantially higher than ambient concentrations. Among the studies reported in Chapter 3 are those examining indoor concentrations of benzene in homes with attached garages. In those studies, concentrations in excess of 30 ug/m^3 are not uncommon. In fact, one study from the mid-1990s reported integrated 24-hour average benzene concentrations of 364 parts per billion by volume (ppbv), which is substantially in excess of EPA's RfC.[24] Other studies of homes, particularly in Alaska, have shown 12 or 24-hour integrated concentrations in homes with attached garages over the RfC as well. While these measurements are of daily duration, the frequency with which these studies report concentrations in excess of the RfC indicates that long-term exposures above the RfC are possible.

Occupational studies of benzene concentrations discussed in Chapter 3 also indicate that among some professions, including those in the petroleum and parks management industries, work-shift averages can exceed EPA's RfC. While these occupational exposures are not within the regulatory purview of EPA, reductions in fuel benzene concentrations are likely to reduce exposures in some of these occupations, a view which is consistent with a 2002 review publication.[25]

As such, while we do not quantify the reduction in non-cancer risk that is likely to result from this rule, we believe that it is appropriate to discuss non-cancer health effects of benzene due to the observation of indoor and personal air concentrations of benzene in excess of the RfC in the literature reviewed in Chapter 3 of the RIA.

2.1.3 Other MSATs

What Commenters Said:

NESCAUM stated that it believes that other MSATs of concern in the Northeast are formaldehyde and diesel particulate matter which would also be numbered among national priorities if the risk assessment handled them properly.

NESCAUM commented that it does not support EPA's use of the URF (unit risk factor) for formaldehyde based on dose-response data from the Chemical Industry Institute of Technology Centers for Health Research (CIIT). The commenter asserted that it believes that EPA inappropriately used a cancer potency factor for formaldehyde that may substantially underestimate cancer risks. The commenter noted that EPA stated that it did not rely on the dose-response value in the Integrated Risk Information System (IRIS) because the science is not current; however, the commenter stated that it believes that by using the CIIT formaldehyde dose-response data to develop a revised cancer URF EPA has not followed the procedures set forth in the Residual Risk Report to Congress for establishing peer-reviewed consensus dose-response information. The commenter noted that the Residual Risk Report to Congress was prepared as mandated by Section 112(f) of the Clean Air Act to provide Congress and the public with a road map of the methods to be used by EPA to assess the risk associated with emissions of HAPs which remained after the implementation of the NESHAP program. The commenter stated that it believes that one of the essential considerations in risk assessment is the evaluation of the source of the data and whether it has been peer reviewed, and it cited the Residual Risk Report to Congress (p.56) to support its comments. NESCAUM summarized EPA's process for developing IRIS assessments, and asserted that EPA did not follow this process in developing the newer URF based on CIIT's analysis. The commenter stated that it believes that the use of the CIIT formaldehyde data in the analyses for this rule undermines the IRIS review process. The NYDEC also commented on the non-peer reviewed cancer risk value for formaldehyde.

Letters:
NESCAUM OAR-2005-0036-0993
New York State Department of Environmental Conservation OAR-2005-0036-0722

Our Response:

The EPA agrees that diesel particulate matter (and diesel exhaust organic gases) are among the mobile source air toxics that pose the greatest risk to human health, and states this in the preamble and RIA.

The EPA disagrees with comments from NESCAUM and NYDEC that the use of the CIIT unit risk estimate for formaldehyde is inappropriate. EPA believes that we should use the best available sources of health effects information for risk or hazard determinations. As we have stated previously, we do not rely exclusively on IRIS values. Rather, we consider all credible and readily available assessments, as noted in the Residual Risk Report to Congress. For air toxics risk assessments, we identify pertinent toxicity or dose-response values using a default hierarchy of sources, with IRIS being the preferred source, to assist us in identifying the most scientifically appropriate benchmarks for our analyses and decisions. The IRIS process contains a peer-review process, and the resulting values represent EPA consensus. When adequate toxicity information is not available in IRIS, we consult other sources in a default hierarchy that recognizes the desirability of review and consistency with EPA risk assessment guidelines. This process ensures that we have consistent and scientifically sound assessments. Furthermore, where the IRIS assessment is relatively dated and newer peer-reviewed assessments are available, we will consider the full set of such assessments in selecting the basis for the risk assessment. In the case of formaldehyde, we have determined that the cancer potency derived using the approach developed by CIIT, which has been peer reviewed by an external review panel sponsored by EPA and the Canadian government, represents an appropriate alternative to EPA's current IRIS URE for formaldehyde. Therefore, this potency represents the best available peer-reviewed science at this time. A comprehensive reassessment of cancer risk has been initiated by EPA's IRIS program. This reassessment will include modeling analyses and endpoints (e.g., lymphohematopoietic cancer) not considered in the CIIT assessment. The revised IRIS assessment will represent the best available peer-reviewed science at the time of its completion.

What Commenters Said:

The Regional Air Pollution Control Agency (RAPCA), the NYDEC, and STAPPA/ALAPCO urged U.S. EPA to investigate the impact of MSAT metals, and the possibility of their control, as part of the rulemaking process. RAPCA and STAPPA/ALAPCO urged EPA to consider the recent Health Effects Institute Research Report. NYDEC criticized the rule's treatment of metals, and urged EPA to extend a ban on manganese to all fuel.

Letters:
Regional Air Pollution Control Agency (RAPCA) OAR-2005-0036-0771
New York State Department of Environmental Conservation (NYDEC) OAR-2005-0036-0722
STAPPA/ALAPCO OAR-2005-0036-0836

Our Response:

EPA is reviewing the results of the recent HEI (Health Effects Institute) study[26] and other studies aimed at identifying the emissions of metals from mobile sources. EPA has research projects underway and is analyzing other data to improve the understanding of metal emissions from mobile sources. This information, as well as that provided by HEI and others will be used to inform any potential future action. EPA is also examining available data on tire and brake wear emissions.

Regarding manganese use as a fuel additive, EPA is currently generating the information needed to update an assessment of the potential human health risks related to having manganese in the national fuel supply. Clean Air Act section 211(c) provides the primary mechanism by which EPA would take actions necessary to minimize exposure to emissions of metals or other additives to diesel and gasoline.

What Commenters Said:

The NYDEC stated that it believes that other mobile source air toxics may be significantly contributing to the cancer risk (formaldehyde, naphthalene and other polycyclic aromatic hydrocarbon compounds) but the NATA assessment uses no cancer risk estimate for naphthalene and has not properly characterized the risk from polycyclic aromatic hydrocarbons (PAHs) emitted from mobile sources. Furthermore, the commenter argues that nitro-PAHs are among the most potent carcinogens known, yet are only briefly discussed in the rule. The commenter also presents a review of science and health concerns regarding nitro-PAHs, arguing that they can be formed in the engine or as a result of aftertreatment.

Letters:
New York State Department of Environmental Conservation (NYDEC) OAR-2005-0036-0722

Our Response:

The analyses done to support this rule do quantify potential cancer risks from naphthalene, using a dose-response value developed by California EPA. EPA's risk assessment for this pollutant is currently in progress. NYDEC provided no specific comments when it asserted that EPA had not properly characterized risk from PAHs. In addition, EPA quantifies the risks associated with fifteen other PAH compounds by grouping the PAH compounds into toxicity categories using risk values primarily from California EPA. For these risk characterizations, refer to RIA Chapter 3, section 3.2.1.2. EPA is also concerned about potential adverse health effects from PAHs and nitro-PAHs, and regulations addressing emissions from highway diesel vehicles, nonroad diesel equipment, locomotives, and commercial marine vessels will substantially reduce these emissions, particularly given the high efficiency of noble metal-based wall-flow particle traps to effectively oxidize organic species including PAHs and nitro-PAHs. EPA is also participating in research to better characterize emissions of PAHs and nitro-PAHs from diesel engines. As this work progresses, EPA will be in a better position to evaluate the need for further action.

What Commenters Said:

The American Chemistry Council Olefins Panel commented that it believes potential health risks from exposures to low levels of 1,3-butadiene in ambient air are far below what is indicated in EPA's 2002 health assessment document, and in the Office of Transportation and Air Quality's (OTAQ) proposed rule. The commenter provided a list of studies and a critique of EPA's 2002 health assessment document for 1,3-butadiene. The commenter asserted that it

believes low levels of 1,3-butadiene typically found in ambient air in fact do not present significant health risks. The basis for this conclusion is that:

1) EPA based its unit risk estimate on an upper bound estimate of the point of departure (PoD) rather than on a maximum likelihood estimate.
2) EPA multiplied its cancer potency estimate by a nonstandard adjustment factor of two.
3) EPA's dose-response assessment does not account for the role of peak exposures;
4) Recent molecular epidemiology studies do not provide any evidence of cancer hazard at current workplace exposures;

Accordingly, the commenter asserted that it believes EPA's proposed rule will be more health protective than EPA has recognized. The International Institute of Synthetic Rubber Producers (IISRP) submitted comments, stating that it has reviewed and is "in full support" of the comments submitted by the American Chemistry Council Olefins Panel.

Letters:
American Chemistry Council Olefins Panel OAR-2005-0036-0823
International Institute of Synthetic Rubber Producers (IISRP) OAR-2005-0036-0807

Our Response:

EPA does not believe that its current unit risk estimate overstates potential health risks from exposure to 1,3-butadiene in the ambient air. First, EPA's use of the upper bound estimate for the PoD for the final unit risk estimate is consistent with EPA's 2005 Guidelines for Carcinogen Risk Assessment (and with the interim draft of the guidelines which was operational at the time the 1,3-butadiene assessment was finalized). This science policy established in the new Guidelines eliminates the historical inconsistency in the treatment of human and rodent data. The policy of using an upper bound estimate is not motivated by potential low-dose computational instabilities in the models applied to rodent data, as this is not even an issue under the Guidelines' two-step approach of modeling the data in the observable range to obtain a PoD and then using linear extrapolation or a non-linear approach to estimate the unit risk or a reference value for cancer. The Guidelines underwent their own external review process, and the science policies presented therein are not generally topics for which EPA seeks external comment in its chemical-specific assessments. Furthermore, EPA's risk estimates are "upper bound" because they are based on upper bound estimates from the dose-response modeling.

A commenter also questions EPA's multiplication of its cancer potency estimate by a nonstandard adjustment factor of two. EPA's use of such an adjustment factor is not unprecedented. An adjustment factor of 2 was used in EPA's vinyl chloride assessment to account for increased early-life susceptibility. In the case of 1,3-butadiene, the primary reason for the use of an adjustment factor of 2 was to account for potential risk of breast cancer in females. Females were not part of the study population in the epidemiology study which provided the basis for the cancer potency estimate, so risks for female breast cancer could not be estimated from the human data. Yet, in the rodent studies, the mammary gland was the one concordant site exhibiting 1,3-butadiene associated tumors in both mice and rats, thus there was clearly a reason to be concerned about breast cancer risk in human females. In the external review draft, this issue was addressed qualitatively but not quantitatively, and the Science Advisory Board (SAB) recommended attempting to "quantitatively address, where possible,

differences between cancer potency for the occupationally exposed and the general population", specifying females, other lifestages, and other potentially susceptible subpopulations. Because there were no chemical-specific data on early-life susceptibility and EPA's 1,3-butadiene assessment pre-dated EPA's 2005 Supplemental Guidance for Assessing Susceptibility from Early-Life Exposures to Carcinogens, which recommends the use of default age-dependent adjustment factors in the absence of chemical-specific data for carcinogens judged to operate through a mutagenic mode of action, EPA did not quantitatively address potential increased early-life susceptibility in its 1,3-butadiene risk estimates.

A commenter also argues that EPA failed to follow the SAB's recommendation that a more appropriate model for 1,3-butadiene risk would factor out the peak-exposure component. EPA did in fact consider a peak exposure analysis; however, the data on peaks were inconsistent and did not support a quantitative analysis that factored out peaks. According to the original study authors, based on their comprehensive dose-response analyses, the relationship between 1,3-butadiene peak-years and leukemia was irregular.

Finally, EPA would like to note that a recent study extended the investigation of 1,3-butadiene exposure and leukemia among synthetic rubber industry workers.[27] The results of this study strengthen the evidence for the relationship between 1,3-butadiene exposure and lymphohematopoietic cancer. This relationship was found to persist after controlling for exposure to other toxics in this work environment.

What Commenters Said:

The Alliance stated its belief that due to uncertainties in the acrolein RfC, comparisons of concentrations to the RfC are not meaningful in drawing conclusions about its public health impacts.

Letters:
Alliance of Automobile Manufacturers (Alliance) OAR-2005-0036-0881

Our Response:

We disagree with the comment that comparisons of ambient acrolein concentrations to its RfC are not meaningful. First, we do note in the RIA, when describing the hazard quotient (HQ), that:

> "[a] value of the HQ less than one indicates that the exposure is lower than the RfC and that no adverse health effects would be expected. A value of the HQ greater than one indicates that the exposure is higher than the RfC. However, because many RfCs incorporate protective assumptions in the face of uncertainty, an HQ greater than one does not necessarily suggest a likelihood of adverse effects. Furthermore, the HQ cannot be translated to a probability that adverse effects will occur and is not likely to be proportional to risk. A HQ greater than one can best be described as indicating that a potential exists for adverse health effects."

We feel that the acrolein RfC, which is from IRIS, is sufficiently robust to allow for this level of information to be gained. Second, we note that concentrations of acrolein are such that in all years modeled, a substantial fraction of the national population is predicted to have HQs greater than one, and this holds regardless of whether the EPA RfC or California REL (Reference Exposure Level) is employed in the calculation. These factors support the identification of acrolein as an important air toxic of concern at environmental levels of exposure.

What Commenters Said:

NYDEC also commented that it is concerned that very little of the data that EPA relies upon to support its actions is verifiable. The commenter stated that it believes that rulemakings must be conducted openly, with the underlying data open to public inspection.

Letters:
New York State Department of Environmental Conservation OAR-2005-0036-0722

Our Response:

We disagree with comments that the methods and information presented in this rule are "unverifiable." All the underlying data used to support analyses in this rule are publicly available. In addition, methods and tools from the 1999 NATA, and the improvements implemented here are the best currently available for modeling exposures and risks from air toxics on a nationwide scale. Furthermore, the dose-response values used were selected using objective criteria described in the Regulatory Impact Analysis for the rule. We note that our emission inventory methods and future risk estimation techniques have been peer-reviewed and published in scholarly journals, or are "in press" and are in online prepublication versions.[28,29]

2.1.4 PM

What Commenters Said:

International Truck and Engine Corporation commented that it objects to the proposal's incomplete characterization of past findings regarding the health effects of diesel emissions. The commenter stated that EPA should refrain from making statements about the alleged health effects of "diesel exhaust" *per se*, which has no uniform or defined composition, or "diesel particulate matter and diesel exhaust organic gases," as opposed to "diesel particulate matter," which was analyzed in the 1999 National-Scale Air Toxics Assessment (NATA). The commenter stated that it believes the Agency should also clarify that the data it relied upon in reaching its prior conclusions are based solely on exposures to emissions from engines using old technology and old, high-sulfur fuel formulations. International asserted that these conclusions cannot be extended to current engines. Furthermore, the commenter stated that if the Agency intends to reiterate the 1999 NATA's "qualitative" conclusion regarding the risk of exposure to diesel particulate matter, it should at least acknowledge the weaknesses in the underlying data, as discussed in the 2002 Health Assessment Document for Diesel Engine Exhaust. The commenter urged EPA to clarify that available evidence is inadequate to determine whether diesel emissions

contribute to asthma or allergenic responses. The commenter stated that it objects to an "opaque" statement in the RIA that "[t]he RfC is not meant to say that 5 $\mu g/m^3$ provides adequate public health protection for ambient $PM_{2.5}$. In fact, there may be benefits to reducing diesel PM below 5 $\mu g/m^3$ since diesel PM is a major contributor to ambient $PM_{2.5}$." International commented that this statement unfairly targets diesel PM as a means to achieve health benefits associated with attainment of the NAAQS. International also commented that EPA should remove or clarify its statement summarizing the current Air Quality Criteria Document for Particulate Matter regarding the hypothetical link between exposure to particulate matter from gasoline and diesel engines and cancer mortality.

Letters:
International Truck and Engine Corporation (International) OAR-2005-0036-0826

Our Response:

The EPA Health Assessment Document for Diesel Engine Exhaust[30] attributes the potential carcinogenic risk from diesel emissions to whole diesel exhaust, rather than diesel PM. EPA also states the following in NATA on the potential carcinogenic risk associated with diesel exhaust:

> In this assessment, the potential risk from diesel exhaust emissions is not addressed in the same fashion that other pollutants are. This is because data are not sufficient to develop a numerical estimate of carcinogenic potency for this pollutant. However, EPA has concluded that diesel exhaust ranks with the other substances that the national-scale assessment suggests pose the greatest relative risk. First, a large number of human epidemiology studies show increased lung cancer associated with diesel exhaust. Furthermore, exposures in these epidemiology studies are in the same range as ambient exposures throughout the United States. In addition to the potential for lung cancer risk, there is a significant potential for non-cancer health effects as well, based on the contribution of diesel particulate matter to ambient levels of fine particles. Exposure to fine particles contributes to harmful respiratory and cardiovascular effects, and to premature mortality. More information on health effects associated with diesel exhaust can be found in the Health Assessment Document for Diesel Exhaust

The EPA Health Assessment Document for Diesel Engine Exhaust concludes that "long-term (chronic) inhalation exposure [to diesel exhaust] is likely to pose a lung cancer hazard to humans, as well as damage the lung in other ways depending on exposure." As stated in its Health Assessment Document, EPA concluded that available data are not sufficient to develop a confident estimate of cancer unit risk and limits EPA's ability to quantify, with confidence, the potential impact of this hazard. EPA though did develop a perspective on risk concluding "there is a reasonable potential that environmental life cancer risks from diesel exhaust may exceed 10^{-5} (one in a hundred thousand) and could be as high as 10^{-3}." EPA cannot rule out the possibility that the lower end of the risk range includes zero.

While EPA's risk assessment is based on exposure to whole diesel exhaust, in its 2000 rule EPA listed diesel particulate matter and diesel exhaust organic gases as a mobile source air toxic.[31] EPA concluded this listing was reasonable because:

1) There are several nontoxic components of diesel exhaust (e.g., water vapor, nitrogen, oxygen)
2) This listing includes the components of diesel exhaust that are likely to contribute to the cancer and noncancer hazard (with the exception of gaseous phase criteria pollutants, such as NO_x, SO_2, and CO which are subject to National Ambient Air Quality Standards)
3) The more precise listing provides Federal and State government, industry and public interest groups an ability to focus on the components of diesel exhaust that pose a potential concern for public health
4) This focus provides specific targets for emission reductions should future analysis indicate that additional controls are necessary.

In the 1999 NATA, diesel particulate matter is used as a metric for exposure to diesel particulate matter and diesel exhaust organic gases.

EPA agrees with comments that the conclusions of the diesel health assessment must be reevaluated to determine applicability to new technology engines. The EPA Health Assessment Document for Diesel Engine Exhaust states that "while EPA believes that the assessment's conclusions apply to the general use of diesel engine today, as cleaner diesel engines replace a substantial number of existing engines, the general applicability of the conclusions in this health assessment document will need to be revaluated." Language providing this clarification has been added to the Regulatory Impact Analysis. EPA and other sponsors have funded a Health Effects Institute program (Advanced Collaborative Emission Study) to characterize emissions (with the emphasis on unregulated emissions) and health effects of engines designed to meet the 2007 and 2010 standards.

In response to the comment that EPA discuss weaknesses in underlying data which form the basis of our conclusion that diesel exhaust is one of the pollutants that poses the greatest risk to human health, we would like to point out that limitations of the data are discussed in EPA's Health Assessment for Diesel exhaust, which is cited in the RIA. Readers can refer to that, but since this rule is not regulating diesel PM, such a discussion is not warranted here.

In addition, one comment above recommended that EPA remove the statement that says there may be benefits to reducing diesel PM below 5 μg/m3 since diesel PM is a major contributor to ambient $PM_{2.5}$, because it unfairly singles out diesel PM. EPA – in its recent rulemaking on $PM_{2.5}$ and also in prior rulemaking directly applicable to mobile sources – discusses and quantifies the general health benefits for reducing PM. Such benefits accrue from reduction of diesel PM as well as any other PM. The EPA Health Assessment Document for Diesel Engine Exhaust has a discussion of diesel PM and ambient PM as related to the NAAQS.

The EPA Health Assessment Document for Diesel Engine Exhaust also states that "evidence is emerging that diesel exhaust exacerbates existing allergies and asthma symptoms." These studies are discussed in the EPA Health Assessment Document for Diesel Engine Exhaust.

EPA disagrees with the comments that we should remove or clarify the statement from the PM National Ambient Air Quality Standard (NAAQS) that diesel PM and gasoline PM are important hypothesized contributors to cancer mortality associated with $PM_{2.5}$. The specific text being referred to is taken from EPA's PM Criteria Document, page 8-318:

> With regard to specific ambient fine particle constituents that may significantly contribute to the observed ambient PM-related increases in lung cancer incidence and mortality, PM components of gasoline and diesel engine exhaust represent one class of hypothesized likely important contributors. Such mobile source PM typically comprises a noticeable fraction of ambient fine particles in many urban areas, having been estimated to comprise from ~5 to 30% of ambient $PM_{2.5}$ in some U.S. urban areas (see Chapter 3). These mobile sources are reasonable candidates as contributors to ambient PM-lung cancer risks, given their being sources of known cancer-causing agents (e.g., PAHs), as are other coal-combustion and/or woodburning emission sources (at least during some seasons).[32]

What Commenters Said:

The WDNR commented that EPA should pursue research into better understanding the quantitative relationship between exposure to diesel emissions and adverse health outcomes such as cancer. The commenter asserted that diesel engine emissions may play a significant role in adverse health outcomes in communities. The WDNR suggested that EPA propose a draft cancer unit risk estimate for diesel exhaust as soon as practicable.

NESCAUM stated that it believes that the risk assessment does not acknowledge the importance of diesel particulate matter.

STAPPA and ALAPCO asserted that "diesel PM accounts for 70 percent of the risk from all air toxics," and expressed disappointment that EPA did not address diesel PM in its proposal. They comment that EPA should acknowledge the impact of diesel PM on public health and "at a minimum describe what the agency has done to reduce diesel PM and identify additional measures that can be pursued in the future."

Letters:
NESCAUM OAR-2005-0036-0993
STAPPA/ALAPCO OAR-2005-0036-0836
Wisconsin Department of Natural Resources (WDNR) OAR-2005-0036-0828

Our Response:

We agree with comments that diesel engines are important contributors to public health concerns over air toxics. We note that EPA's *Health Assessment Document for Diesel Engine Emissions* (HAD) and RIA Chapter 3 provide a comprehensive overview of health studies of diesel exhaust and traffic more generally. We agree that it would be useful if EPA were able to propose a cancer unit risk estimate for diesel exhaust. However, for several reasons outlined in the HAD and in other documents, we do not at present feel that available occupational

epidemiology or toxicology provide sufficient basis for quantification of cancer risk related to diesel exhaust or its constituents.[33,34]

While the modeling analysis in this rule does not specifically model diesel PM or diesel exhaust organic gases, EPA has made clear in a number of past and pending rules that it considers reduction of PM and other pollutants in diesel exhaust to be of high priority. The statutory requirements for this rule specifically call for achieving the greatest emission reductions achievable. In the case of diesel PM, EPA's recent regulations introducing ultra-low sulfur diesel (ULSD) and strict limits on PM emissions from on-highway and non-road diesel engines constitute the greatest emission reductions currently achievable, and no further emission reductions from the diesel engines covered by these rules are considered feasible at this time. We note that EPA has recently proposed strict emission limits on emissions from diesel engines powering locomotives and marine vessels. We also note that EPA has numerous voluntary programs dedicated to reducing air pollution from diesel vehicles, including the National Clean Diesel Campaign (www.epa.gov/cleandiesel) and the Smartway Transport Partnership (www.epa.gov/smartway). The impact of other EPA actions are discussed in Section IV of the preamble and Chapter 2 of the Regulatory Impact Analysis.

What Commenters Said:

The NJDEP cited recent research from California indicating that mobile sources may be the largest source of ultrafine particles, and that these particles may have greater potential for adverse health impacts than $PM_{2.5}$ and PM_{10}. The commenter asserted that currently employed emission control strategies to reduce particle mass may not result in corresponding reductions in ultrafine particle count. The commenter further stated that it believes that EPA should take steps to reduce particle counts when considering emission control strategies and emission standards aimed at reducing particle mass. It further called for greater research into ultrafine particle emissions and control measures.

Sensors, Inc. cited information indicating that ultrafine particles are more important than "just the PM measurements."

Letters:
New Jersey Department of Environmental Protection, Division of Air Quality (NJDEP) OAR-2005-0036-0829
Sensors, Inc. (SEMTECH) OAR-2005-0036-0958

Our Response:

We agree that mobile sources are a major contributor to ambient concentrations of ultrafine particles. Work cited in EPA's heavy-duty diesel rulemaking (66 FR 5048, January 18, 2001) shows that the EPA diesel PM standards will effectively control ultrafine particles by a factor of 10 by oxidizing the volatile organic compound precursors and by an additional factor of 10 by reducing diesel fuel sulfur. Work since then continues to show that ultrafine PM is effectively controlled by the EPA diesel PM standards, particularly when viewed across

representative driving cycles.[35] The recent rulemaking for the $PM_{2.5}$ National Ambient Air Quality Standard did not set a PM standard for ultrafine PM but did tighten the 24-hour $PM_{2.5}$ standard and reaffirmed the annual $PM_{2.5}$ standard and somewhat tightened the criteria for spatial averaging. EPA is actively engaged in emissions characterization work for both diesel and gasoline PM to make sure EPA has the latest information on exhaust PM including ultrafine PM. This information will allow EPA to determine what additional PM controls are needed and move to implement them.

What Commenters Said:

NJDEP and STAPPA/ALAPCO submitted comments that EPA needs to follow through on its observation in the proposal that "gasoline exhaust is a significant source of particulate matter, contributing to the health effects observed for ambient PM," and to continue its work "to improve the understanding of PM emissions from gasoline engines, including the potential range of emissions and factors that influence emissions."

The Regional Air Pollution Control Agency (RAPCA) commented that it recommends expanded or more stringent requirements in advancing scientific understanding of PM emissions from gasoline engines.

Letters:
New Jersey Department of Environmental Protection, Division of Air Quality (NJDEP) OAR-2005-0036-0829
Regional Air Pollution Control Agency (RAPCA) OAR-2005-0036-0771
STAPPA/ALAPCO OAR-2005-0036-0836

Our Response:

We agree with comments that EPA needs to follow through on concerns regarding the contribution of gasoline exhaust to particulate matter. We note that the current rule is expected to result in substantial cold temperature emission reductions of both direct and secondary PM from new gasoline vehicles. We also note that we continue to lead a multi-sponsor research program dedicated to characterizing PM emissions from a representative sample of light-duty gasoline vehicles.

2.1.5 General Issues

What Commenters Said:

NESCAUM commented that it believes that the risk assessment should not use national average exposures to represent the risk of exposure to MSAT (RIA p.3-46). The commenter stated that it believes that the risk reduction estimated in Section 3.2 of the proposal RIA (from 2.3×10^{-5} to 1.7×10^{-5}) is essentially insignificant, and that both risks round to 2×10^{-5}.

Letters:

NESCAUM OAR-2005-0036-0993

Our Response:

We disagree that national average exposures do not provide useful information. EPA uses a national average exposure to represent the impacts of air toxics on a national basis only in reporting summary statistics and trends over time in air quality, exposure, and risk. However, other statistical summaries feature prominently in our analysis. Throughout Section 3.2 of the RIA, we make use of county-level maps in multiple years to express the effects of the rule in different parts of the nation. Further, we have also calculated the population fractions exposed to different levels of risk. We note that underlying the exposure model HAPEM6 is an assumption of geographically-defined differences in ambient concentrations resulting from proximity to major roads.

Furthermore, we note that Table 3.2-15 of the RIA presents information indicating that the greatest reductions in exposure and risk accruing from this rule occur among individuals experiencing the highest levels of risk.

We disagree that rounding risk reductions eliminates their importance. Modeling done to support the final rule shows a 26% reduction in total cancer risk from MSATs from all sources between 1999 and 2030, with controls in place, and a 40% reduction in benzene from all sources.

What Commenters Said:

NESCAUM commented that it believes that EPA has not adequately considered in this proposed regulation the episodic, high-end exposures to respiratory irritants emitted from mobile sources or the cumulative impact of exposure to multiple respiratory irritants such as acetaldehyde, acrolein, formaldehyde, and diesel particulate.

Letters:
NESCAUM OAR-2005-0036-0993

Our Response:

EPA is currently limited in its ability to assess health impacts of episodic, high-end exposures to some pollutants, because of the lack of dose-response assessments for acute exposures to air toxics. Thus, EPA is developing acute reference concentrations for compounds that will be used to identify areas of potential public health risk from episodic, high exposures. The commenter is incorrect that we have not considered the cumulative impact of multiple respiratory irritants. We include an assessment of the cumulative respiratory hazard index in this rulemaking (see RIA Section 3.2.1.2.2. Exposure and Risk Trends for Air Toxics). We also note that the motor vehicle emission controls in this rule will also reduce primary emissions and secondary formation of aldehydes. As noted below, EPA's diesel emission rules for onroad and nonroad engines have made substantial contributions to reducing future diesel PM emissions.

What Commenters Said:

NYDEC claimed that while risk estimates in NATA and tools like it are based on toxicity estimates for individual chemical compounds, that the synergistic effects of the "complex mixture of MSATs" are unknown. It asserted that exposures to mixtures of MSATs may result in "a greater risk" (greater than additive toxicity), emphasizing that sensitive subpopulations such as children may be of particular concern, particularly when they live or attend school near roadways.

Letters:
New York State Department of Environmental Conservation (NYDEC) OAR-2005-0036-0772

Our response:

We agree that the synergistic effects (i.e., greater than additive effects) of the "complex mixture of MSATs" are not well known. We also note that antagonistic effects (i.e. less than additive) of mixture toxicities are also poorly understood. We note that we base our health conclusions as to the effects of individual MSATs on information in EPA's IRIS, and other sources where applicable. We consider toxicity of mixtures to be an area of long-term interest to EPA. We also agree that subpopulations such as children may have differential susceptibility to MSATs, both singly and in combination. Lastly, we note that the exposures of those living or otherwise spending significant quantities of time near major roadways may be elevated. Chapter 3 of the RIA discusses these concerns in greater detail.

What Commenters Said:

The NJDEP and NESCAUM commented that EPA should better assess the risks to children. This comment is in response to EPA's Supplemental Guidance for Assessing Early-Life Exposure to Carcinogens, issued by the National Center for Environmental Assessment.

Letters:
New Jersey Department of Environmental Protection (NJDEP) OAR-2005-0036-0829

Our Response:

Regarding the need to better assess health risks in children, in response to EPA's recent Supplemental Guidance for Assessing Early Life Exposure to Carcinogens,[36] EPA has not yet determined which pollutants meet the criteria for making adjustments to risks in order to better reflect risks in children. This will be done as part of the IRIS process.

What Commenters Said:

The NYDEC commented that frequently in this rulemaking, EPA claims that additional regulation cannot be undertaken because the Agency lacks sufficient or appropriate data. It asserted, however, EPA has not made sufficient efforts to obtain data. Aside from EPA's own research, NYDEC claimed that EPA has not availed itself of a number of resources, such as the

Health Effects Institute (HEI), Coordinating Research Council (CRC), and states, municipalities and their associations. EPA has also not utilized information that is (or should be) available to the Agency through reporting under Sections 202(a)(4) and 206(a)(3) of the Clean Air Act.

Letters:
New York State Department of Environmental Conservation (NYDEC) OAR-2005-0036-0772

Our Response:

We disagree with comments that EPA has not made sufficient efforts to obtain data relevant to this rulemaking. As noted in section I.B of the preamble of the proposal for this rule, EPA has devoted substantial resources to the Technical Analysis Plan to which we committed under the 2001 rule. Second, Chapter 3 of the RIA presents a comprehensive review of scholarly exposure and health studies of "near roadway exposure." Third, we have incorporated many of the findings of these studies into our analysis tools, including the exposure model HAPEM6. Fourth, since the 2001 rule, EPA staff (as well as others) have published a number of articles in scholarly journals that on many subjects including emissions characterization and exposure projects reflect our efforts to better characterize air pollution gradients near major roadways. We note that in performing this work, we worked closely with numerous states and metropolitan planning organizations in several regions, or obtained local transportation data directly from state or local governments. Fourth, we note that EPA's Office of Research and Development has undertaken a major initiative related to exposures occurring near roadways. Included in this effort:

- Analysis of near-roadway epidemiology studies – EPA and external researchers will assess consistencies and inconsistencies in near road epidemiological study results, including the metrics used to assign exposures for near road populations. A number of the key epidemiological studies will be re-analyzed using common exposure metrics to better estimate potential risks for populations living near roads. This work is being conducted within EPA's National Health and Environmental Effects Research Laboratory (NHEERL).
- Monitoring studies of near-roadway pollution gradients – EPA is leading a consortium of organizations, including the Federal Highway Administration, to conduct near road monitoring assessments to better evaluate the relationship of traffic operating characteristics with near road air pollution. Studies will be conducted in a minimum of three cities in the U.S. using consistent monitoring methods to assess potential geographic influences on near road air quality. This work is being led by of EPA's National Risk Management Research Laboratory (NRMRL).
- Evaluation of existing emissions and dispersion models – Data from the field studies will be used to evaluate the response and relationship of existing emissions and dispersion models. In addition, EPA researchers will be conducting wind tunnel experiments of several common roadway configurations to determine how pollutants disperse under these conditions and how existing dispersion models handle these configurations. The roadway configurations include at-grade roadways, depressed roads, elevated roads, and at-grade roads with vegetation or noise barriers. This work is being run through the National Oceanic and Atmospheric Administration's Air Research Laboratory and EPA's National Exposure Research Laboratory (NERL).

- Characterization of infiltration of pollutants into schools – As part of the research consortium investigating near road concentration gradients, an assessment is being conducted on how these emissions infiltrate into the indoor air of schools located near major roads. This project will assess how the pollutants infiltrate, and what mitigation techniques are available to improve indoor air quality in these schools. This research is being lead by NRMRL.
- Assessment of mitigation measures – As described, wind tunnel tests will be conducted to determine if vegetation and/or noise barriers may mitigate air pollution levels in close proximity to roadways. In addition, the effects of noise barriers and vegetation will attempt to be analyzed in the field concentration gradient measurement studies.
- Health effects of near-roadway emissions – As described, re-analyses of previous epidemiological studies will provide enhanced information on the effects of traffic emissions on public health for near road populations. EPA researchers will also be determining the toxicity of PM samples collected near and far from major roadways as part of the concentration gradient measurements. This work is being conducted between NHEERL and NERL. EPA is also supporting health effects studies on traffic emissions as part of the new PM Center grants through the National Center for Environmental Research (NCER).

We also note that EPA is an active sponsor of HEI, and works closely with HEI in their research. HEI has conducted a large number of projects related to mobile source emissions (including PM, benzene, 1,3-butadiene, NO_2, and other compounds) that have been widely accepted in the scientific community and used extensively by EPA in its regulatory programs. Also, for a number of years, the automobile manufacturers submitted annual reports on their emissions characterization work related to Section 202(a)(4). This and other emissions characterization work, which taken together is actually very extensive, including that conducted by EPA and the CRC, have been used in structuring the emission models for air toxics, MOBILE 6.2.

We also note our close involvement with CRC in co-sponsoring recent joint research, including CRC's E-55/59 emissions study of heavy-duty diesel trucks and the EPA-led emission study of light duty gasoline vehicles in the Kansas City Metropolitan Area, a multi-million dollar program testing 500 gasoline vehicles for PM and other emissions, including detailed speciation of PM and VOC emissions in a subsample of vehicles.

All of these emissions characterization and health projects conducted in the past several years means that there actually was sufficient high caliber data which EPA could use in making decisions about its mobile source air toxics regulations.

What Commenters Said:

The Alliance stated that MSAT inventories are decreasing with concurrent ambient reductions. The commenter presents an analysis of air toxic emissions across model years and concentrations in several urban areas. The commenter stated two major points: 1. Air toxic emissions are decreasing in conjunction with cleaner vehicle technology; and 2. Ambient air toxic concentrations generally fall below the EPA defined reference concentrations (RfC). To

support its comments, the commenter showed figures which it stated shows reductions in various MSAT emission factors (mg/mile), based on the model year of vehicles ranging from the 1970's to 2005. The Alliance further commented that it believes that MSAT reductions will occur after 2005 due to the Tier 2 standards phase-in continuing through 2009 Model Year. The commenter presented an analysis of toxic emission factors from the calendar year 2004 light-duty gasoline vehicle fleet in comparison with a fully phased-in Tier 2 fleet in calendar year 2040, which resulted in a reduction of greater than 70% for benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and acrolein. The commenter noted that its MOBILE6.2 analysis does not factor in I/M benefits or changes in gasoline benzene content. The commenter stated that it believes that greater reductions in MSATs may be evident if more representative fleet characteristics are modeled. It also presented charts depicting trends in ambient toxics over time, showing downward trends in concentrations of individual air toxics over time.

The commenter also presented analyses of trends in ambient concentrations from all monitoring data across the U.S. for several mobile source air toxics. They present information to indicate that between 1994 and 2004, ambient concentrations of benzene and 1,3-butadiene underwent significant decline. They also state that although a general trend in concentrations of formaldehyde and acetaldehyde is present, a "clear trend" could not be detected due to atmospheric chemistry and the contribution of biogenic sources to direct and secondary aldehyde formation. The do not present analyses of acrolein data because "EPA contractors concluded the acrolein data are not reliable." They note that with the exception of acrolein, ambient concentrations of ambient MSATs are below the relevant reference concentration (RfC).

The commenter also notes that a fact sheet accompanying EPA's National Air Toxics Assessment (NATA) indicated that the risk of contracting cancer of any type is one in three, while Table 3.2-6 of the proposal's RIA indicates that for 1999, the risk of cancer from on-road vehicles is estimated at 3 in 100,000. They also note that by 2020, existing control programs will make it so that only benzene exceeds the one in one million level of risk.

Letters:
Alliance of Automobile Manufacturers (Alliance) OAR-2005-0036-0881

Our Response:

We agree with comments that air toxic emission rates and ambient concentrations of most air toxics underwent significant declines in the 1990s, and we discuss these data in the rule. Also, EPA's future year inventories account for phase-in of Tier 2 vehicles, low sulfur standards and other changes in fuels, inspection and maintenance (IM) benefits, and other factors. We also agree that for most air toxics, ambient concentrations are well below the RfC, indicating that noncancer health effects from most individual air toxics are highly unlikely at ambient concentrations.

We note that while most MSATs have ambient concentrations below their respective reference concentrations, in the RIA we consider the totality of exposure from multiple pathways. We note that among the studies discussed in Chapter 3 of the RIA, a number of studies report concentrations of benzene in indoor air and other locations that are in excess of the

RfC for benzene, generally in studies examining the influence of attached garages on benzene in air. We expect the standards in this rule will substantially reduce exposures through this pathway, as well through reducing concentrations in ambient air.

We note that the risk of dying of any cancer over a lifetime results from multiple factors, including genetic, behavioral, and environmental factors, and that section 202(l)(2) is not a risk-based standard. The standards in this rule make substantial reductions in the emissions of mobile source air toxics, and we expect future cancer risks to decline as a result.

What Commenters Said:

Sensors, Inc. cited a 2006 presentation by Jean-Paul Morin of the French National Institute of Health for Health and Medical Research, in which high emission ratios of nitrogen dioxide to total oxides of nitrogen (NO_2/NOx) were associated with increased oxidative stress. The commenter urged EPA to begin examining this issue in greater detail.

Letters:
Sensors, Inc. (SEMTECH) OAR-2005-0036-0958

Our Response:

We agree with the comment regarding the importance of examining the public health consequences of the fraction of NOx emitted as NO_2. At present, EPA is in process of revising its air quality criteria document for NO_2 to account for recent studies of the species.

2.2 National-Scale Modeling

The following comments refer to the modeling approach employed in this rule for quantifying air quality, exposure, and risk changes associated with the rule.

What Commenters Said:

The NYDEC asserted that it believes that while limited, the 1999 National Air Toxics Assessment provided data that is useful in identifying air toxics of greatest concern, and the use of similar tools in this rule is important.

API commented that the air quality modeling performed for this rule is not sufficiently robust for regulatory purposes. The commenter cited text from the National Air Toxics Assessment, indicating that the "NATA assessment should not be used as the basis for developing risk assessment plans or regulations to control specific sources or pollutants." As EPA's national-scale modeling employs tools similar to those in NATA, API asserted that the limitations of the 1999 NATA are applicable for this rule. API asserted that the 1999 NATA did not undergo independent peer review. API cited an evaluation of the 1996 NATA Assessment System for Population Exposure Nationwide (ASPEN) modeling and claimed that the best correlation (Pearson's r) between modeled and monitored results found was 0.57, applied to the

Northeast. It notes the evaluation found lower correlations in other regions. On these bases, API asserted that the ASPEN is not sufficiently robust for use in regulation.

API and Marathon Petroleum Company LLC (MPC) commented that the proposal relies on 1999 NATA data, and that this source is out of date. The commenters assert that the 1999 NATA does not include the emission reduction benefits of EPA regulations that are already in place, including Phase 2 reformulated gasoline (RFG), the 2001 MSAT rule, and fuel desulfurization. API and MPC comment that the NATA should be updated with current data prior to any regulation.

Letters:
Alliance of Automobile Manufacturers (Alliance) OAR-2005-0036-0881
Marathon Petroleum Company (MPC) OAR-2005-0036-0946
New York State Department of Environmental Conservation (NYDEC) OAR-2005-0036-0772

Our Response:

We agree with NYDEC that the analytical approach employed in the 1999 NATA provides useful information for identifying toxics of greatest concern, and that the use of NATA-like tools in this rule provide important information

While the caveats that apply to the 1999 NATA do state that it should not be used as the basis for risk assessment plans or regulations to control specific sources or pollutants, the national-scale assessment results presented in the preamble and RIA of this rule are used only to provide a perspective on risk, and were not used as the basis for any regulatory decision. We would also like to note that, in this rule, we employed ASPEN and HAPEM6 to future years with appropriate emission inventories for each year. This more extensive analysis provides more information than a single year "snapshot," such as NATA. We note that the methodologies employed for this rule underwent peer review in a scholarly journal and are in press as of the publication of this rule.[37] Finally, we note that comparisons of modeled air toxic concentrations to monitor data show good agreement for benzene and acetaldehyde, but suggest that ASPEN could be underpredicting for other air toxics. These comparisons are discussed in Section 3.2.1.3 of the RIA.

We disagree with the comment that NATA 1999's lack of accounting for more recent regulations and emission changes makes the national scale modeling results in this rule outdated. As described in the RIA, we modeled emissions, air quality, exposure and risk for 1999 and a range of future years, accounting for the impacts of current and planned future programs.

What Commenters Said:

NESCAUM noted that as part of its March 2001 mobile source air toxics rulemaking, EPA identified nonroad engine emission factors as a critical area of research and committed to data collection as part of a technical analysis plan. NESCAUM noted that EPA has since completed a number of nonroad gasoline engine emission test programs, and that these data have

not been fully analyzed and incorporated into EPA's emission inventory tools. The commenter exhorted EPA to update NONROAD and NMIM to incorporate these data, and complete any needed emission testing programs and data analyses.

Letters:
NESCAUM OAR-2005-0036-0993

Our Response:

We agree with the comment on the need to incorporate emission test program data from nonroad engines into the NONROAD and NMIM (National Mobile Inventory Model) models. Section 2.3 of the Regulatory Impact Analysis discusses recent nonroad emission test programs and plans to integrate data from these programs into the NMIM model.

EPA remains committed to increasing the available emission data from nonroad engines through on-going efforts to fund testing and to leverage testing for engine emission data (criteria pollutant and MSAT) in both gasoline and diesel nonroad equipment types. In 2006, EPA initiated a nonroad pilot program to survey the population, activity, and emissions of construction-type engines with on-board testing equipment. We continue to work with industry sponsored trade and research groups, like Manufacturers of Emission Controls Association, to test the effect of various emission control devices on engines used in several classes of nonroad equipment. In-house, EPA has been testing the safety and level of emission control from various configurations of small SI engine (lawn and garden, primarily) aftertreatment control equipment. When appropriate, we will use available emission testing data to update our emission models.

2.3 Near-Road, Attached Garages, and Other Microenvironmental Exposure

2.3.1 Adequacy of Air Quality, Exposure and Risk Analysis

The following set of comments refer to methods EPA undertook in its analysis of air quality, exposure, and risk from air toxics. The comments address the adequacy of EPA's analytical approach, and highlight information regarding concentration patterns near major roadways or in vehicles.

What Commenters Said:

The NJDEP and NESCAUM noted that the primary analysis in the RIA accompanying this rule is based on a national-scale dispersion modeling study, which may be sufficient to establish that mobile source air toxics are a serious national problem, but fails to address higher exposures experienced by people living in urban centers, in homes with attached garages, and the elevated exposures of people traveling in their cars, and higher exposures experienced by persons living within 200 meters of roadways. NJDEP cited a recent publication from the RIOPA study where concentrations of some gaseous air toxics were elevated near major roadways. The commenters stated that they believe that EPA should better assess the impact on people near roadways. The commenter stated that it believes that, in order to protect the millions of people

who live in our most densely populated urban areas, EPA should better assess the impact on people near roadways.

NESCAUM expressed concerns with results from recent studies which reveal exposures that greatly exceed ambient monitored levels of mobile source air toxics in microenvironments in the Northeast. The commenter cited the following conclusions from recent studies of microenvironment exposure levels in the Northeast: 1) levels of benzene found in pedestrian and bicyclist zones were approximately 10 times higher than typical ambient levels due to vehicle exhaust; 2) $PM_{2.5}$ levels at commuter train stations in Boston were found to peak at 1,000 micrograms per cubic meter - 50 to 100 times higher than ambient levels; 3) construction workers operating post-1996 model year nonroad equipment were exposed to 8-hour $PM_{2.5}$ averages as high as 600 micrograms per cubic meter; and, 4) an additional study outside of the region found that vehicle drivers are exposed to PM and benzene levels that are 10 to 16 times higher than ambient levels. The commenter stated that it believes that, in light of the public health threat posed by mobile source air toxics, a more comprehensive evaluation of toxics risk and additional control measures is needed from EPA.

NESCAUM commented that since the publication of the MSAT1 rule, EPA has conducted personal exposure and ambient air monitoring studies in homes, schools, near roadways, vehicles and inside homes with attached garages. The commenter also noted that EPA has also worked to improve existing models, such as the HAPEM. However, despite this initial work, the commenter stated that it believes that the proposed rule does not fully address the much higher exposures experienced by people living in homes with attached garages, or by people traveling in their cars. The commenter further stated that it believes that the higher exposures experienced by people living within 200 meters of roadways have not been comprehensively addressed. The commenter noted that these issues are discussed in the RIA, but stated that it believes that the full burden on the American people has not yet been quantified. The commenter cited that HAPEM6 as an example, which incorporates near-roadway exposures, was only extended to three states, Georgia, Colorado and New York. NESCAUM commented that, since an improved version of HAPEM6 has been developed, it encourages EPA to implement the model nationwide.

Environmental Defense, NRDC, U.S. PIRG, and ALA commented that because benzene is emitted primarily by mobile sources, concentrations are elevated near major roadways, which they commented that they believe is a significant health consideration. The commenters cited a statistic that in 2003 12.6 percent of U.S. housing units were within 300 feet of a major transportation source, and further noted that EPA has cited dozens of studies showing increased benzene exposure for people who spend time on or near major roadways. The commenters noted that these groups include regular commuters and highway patrol officers, people who live near major roadways, and children who go to school near major traffic sources. Additionally, the commenters noted that people who have garages attached to their homes are exposed to elevated concentrations of benzene and other air toxics.

The Alliance commented that the prevalence of “hot spots” of mobile source air toxics in urban areas is unclear. They cited the Multiple Air Toxics Exposure Study (MATES-II) study in California’s South Coast Air Basin. They noted that the study did not report differences in mass

concentrations of air toxics between microscale monitors, including those located in close proximity of freeways, and fixed-site monitors. They comment that MATES-II should be viewed as one of the most complete studies examining spatial variation in ambient concentration of air toxics.

Letters:
Alliance of Automobile Manufacturers (Alliance) OAR-2005-0036-0881
Environmental Defense, NRDC, U.S. PIRG, ALA OAR-2005-0036-0868
New Jersey Department of Environmental Protection (NJDEP) OAR-2005-0036-0829
NESCAUM OAR-2005-0036-0993

Our Response:

We agree with comments that concentrations of benzene and other pollutants are elevated near major roadways. We also agree that exposures to these compounds may be elevated for people who spend considerable time traveling on or working or living near major roadways, or in proximity of other mobile sources.

We agree with the comments that HAPEM6 should be extended nationally. For this final rule, we ran HAPEM6 for the entire nation and have based subsequent risk calculations on the exposure modeling results.

We disagree with comments that our analyses fail to address exposures experienced by people living in urban centers, traveling in their cars, or living near major roadways. First, air quality, exposures, and risks in urban areas are modeled with an air quality model, ASPEN, which employs emission inputs at the census tract level of resolution. We believe that this level of detail provides sufficient representation of emission trends and resulting air quality in urban areas. Second, the HAPEM6 exposure model, and its predecessor HAPEM5, explicitly include microenvironments within vehicles, and have new approaches for calculating the time and concentration that people experience while commuting. Third, NJDEP highlighted the study in which outdoor Relationship among Indoor, Outdoor, and Personal Air (RIOPA) concentration data were shown to be elevated near major roadways. We would like to point out that the Office of Transportation and Air Quality directly funded the development of this study's geographic component, the products of which are cited in Chapter 3 of the RIA.

We agree with the comment that EPA should better assess the impact of mobile source air toxics on people living near major roadways and other mobile source-affected microenvironments. However, we disagree that we have not been sufficiently diligent in assessing the public health impacts of this phenomenon. We believe that EPA's efforts in this field have been substantial. While all risks have not been quantified, we believe that at this point, Chapter 3 of the RIA provides as much credible information regarding the nature and magnitude of these risks as is currently possible.

We agree with the comment that the MATES-II study is a valuable source of information on spatial variability of air toxics, and acknowledge the lack of significant differences between microscale and fixed ambient site monitors in that study. Three of the 14 microscale sites at

which air toxics were measured in the MATES-II study were sited to monitor mobile source toxics near roadways during a four to five week period utilizing two to three samples per week. While the study did not report elevated levels at these three microscale sites over these short monitoring periods, ambient monitoring conducted at 10 sampling sites in the South Coast Air Basin during a one-year period found concentrations of mobile source related compounds such as benzene and 1,3-butadiene were generally high throughout the South Coast Air Basin. However, taken in totality, the available scholarly literature, summarized in Chapter 3, provides unequivocal evidence that concentrations of numerous air toxics, including benzene, are elevated near major roadways.

2.3.2 Attached Garages

The comments in this section refer to the influence of residential attached (integral) garages on indoor air quality.

What Commenters Said:

The Alaska Department of Environmental Conservation (ADEC) commented that homes with attached garages predominate Alaskan housing in Anchorage and Fairbanks, noting that these homes often do not have a barrier between the garage and the living area. The commenter stated that it believes that evaporative emissions of cars and gasoline containers in the garage cause high levels of benzene to permeate a home's living area. The commenter cited evidence of high indoor benzene concentrations in Alaskan homes, noting that a 1998 from Anchorage found indoor benzene concentrations in homes with attached garages exceeding concentrations in homes without attached garages by over 60 $\mu g/m^3$. ADEC also noted that in some rural areas of Alaska, residents store fuel indoors to prevent "gelling," commenting that the portable fuel container provisions of the rule will improve indoor air quality in such homes.

The Municipality of Anchorage Department of Health and Human Services (Anchorage) noted that recent studies of homes with attached garages in Anchorage have found concentrations of benzene that are substantially higher than nationwide survey data (EPA's 1980s TEAM study) of average concentrations indicate. Anchorage also refers to results of an unpublished study that employed tracer gases to determine garage air infiltration into residential living spaces in homes with attached garages. Anchorage reports that the study found that approximately 27% of air and 90% of benzene in indoor air originated in an attached garage. They also report that study results indicate that indoor air concentrations of benzene in Anchorage are much greater than outdoor air. Anchorage also reported results of a telephone survey of householders with attached garages. From the comment: "Approximately 30% stored fuel in the garage. Though cars were parked overnight in 82% of garages, 52% contained one or more snow blowers, lawn mowers, or chainsaws. Additionally, a motorcycle, ATV or similar vehicle was parked in 23% of respondents' garages." Anchorage also cited as study from Australia in which children exposed to benzene at concentrations of 6.3 parts per billion by volume (ppbv) were eight-fold more likely to have asthma. Anchorage noted that it has released a request for proposals for a study of asthma and indoor Volatile Organic Compounds (VOCs) in Anchorage.

ADEC commented that it believes that Alaskans may be exposed to relatively greater concentrations of benzene than residents of other areas. ADEC stated its belief that given benzene's classification as a carcinogen, benzene is an important pollutant with possible public health implications. ADEC further commented that it believes that Alaskans face "a multi-faceted problem":

- winter inversions keep pollutants in the breathing space of Alaskan residents;
- winter gasoline has higher benzene levels;
- people's homes maximize exposure to their off-gassing cars in the garage.

ADEC stated that it believes that factors leading to high benzene exposure are exacerbated by Alaska's gasoline having the highest benzene content in the nation. Lastly, ADEC cited a study of 137 Anchorage homes by the Municipality of Anchorage, in which indoor benzene concentrations averaged 70.8 μg/m3 for homes with attached garages and 8.6 μg/m3 for homes without attached garages.

Letters:

Alaska Department of Environmental Conservation, Division of Air Quality (ADEC) OAR-2005-0036-0975

Municipality of Anchorage, Department of Health and Human Services (Anchorage) OAR-2005-0036-0976

Our Response:

We agree with the comments that homes in Alaska may have substantially higher indoor concentrations of benzene as a result of benzene concentrations inside residential attached garages. We note that the studies provided by Anchorage and ADEC have been useful contributions to our summary and analysis of air toxics exposure data. We also note that the greatest emission benefits from this rule, in percentage, will be realized in Alaska. See Chapter 2 of the RIA for details.

2.4 Emission Reductions

What Commenters Said:

NESCAUM acknowledged that formaldehyde emissions are expected to decline but stated that it believes that additional reductions in the emissions of other MSATs are needed. NESCAUM noted that in the RIA (p.3-43) several MSATs are flagged as "significant contributors to cancer risk," including 1,3-butadiene, acetaldehyde, naphthalene and hexavalent chromium. However, the commenter stated that it believes that the proposed rule did very little to lower emissions of these significant pollutants.

Letters:

American Petroleum Institute (API) OAR-2005-0036-0884

ExxonMobil Refining & Supply Company OAR-2005-0036-0772

Our Response:

We disagree with comments indicating that this rule does little to lower emissions of "significant pollutants" not specifically addressed in this proposal. We note that the cold temperature gasoline emission standards will reduce all VOC-based air toxics, as well as particulate matter. Chromium is a trace contaminant in mobile source emissions, and the processes leading to its emissions are not well understood. . Although engine wear, trace contamination of fuel or oil may be likely sources, existing data do no allow an apportionment of the extent to which any one process may effect emissions. In the 1999 NATA, mobile sources contributed less than 5% of the national chromium inventory and 13.4% of personal exposure concentrations. However, the emissions data underlying the mobile source inventory are very limited. Mobile source speciation fractions for Hexavalent Chromium (Cr(VI)) are based on data obtained from utility boilers and gas turbines. Given these factors, we do not consider control of mobile source Cr(VI) to be sufficiently supported by data or feasible to control at this point.

Also, contrary to several comments, we have addressed a broad range of air toxics, although the fuel standard applies to benzene only. As noted above, the motor vehicle and portable fuel container emission standards will substantially reduce emissions of many VOC species, including air toxics.

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