Commission Briefing Paper 4D-06

Documentation of Impacts of Vehicle Toxic Air Emissions on Public Health

Prepared by: Section 1909 Commission Staff
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Introduction

This paper is part of a series of briefing papers to be prepared for the National Surface Transportation Policy and Revenue Study Commission authorized in Section 1909 of SAFETEA-LU. The papers are intended to synthesize the state-of-the-practice consensus on the issues that are relevant to the Commission's charge outlined in Section 1909, and will serve as background material in developing the analyses to be presented in the final report of the Commission.

This paper presents recent findings about the health impacts of key mobile source air toxics (MSATs) -- with particular emphasis on what EPA previously identified as the "priority" air toxics: benzene, 1,3 butadiene, acrolein, acetaldehyde, formaldehyde and diesel particulate matter -- but also includes naphthalene and polycyclic organic matter (POM). On March 29, 2006, EPA proposed a change that redefines what compounds are considered MSATs and which are of the greatest concern. The emissions emphasized here largely coincide with EPA's new information.

Background and Key Findings

- Air toxics, which are also known in the Clean Air Act as "hazardous air pollutants," are pollutants known or suspected to cause cancer or other serious health effects, including effects on people's respiratory, cardiovascular, neurological, immune, reproductive, or other organ systems. They may also have developmental effects.
- EPA's National Air Toxics Analysis (NATA) for the 1999 calendar year estimates that a large fraction of the national population experiences elevated cancer risk from air toxics. EPA estimates in NATA that motor vehicles and other mobile sources were responsible for about 44% of total estimated outdoor air toxic emissions, almost 50% of the estimated cancer risk, and 74% of the estimated noncancer risk
- There are substantial uncertainties regarding the health effects of ambient levels of air toxics in general, irrespective of their sources. Benzene may be more harmful than previously thought, but the health impacts of some of the other MSAT emissions, like acetaldehyde, may not be clear, particularly at ambient levels. The Federal government is evaluating the evidence for possible increased emissions of acetaldehyde with particular fuels (ethanol) as well as its carcinogenicity.
- The assessment of MSATs is a dynamic field with a considerable number of new studies underway and a steady flow of new research results. Interest is high from environmental, health and community groups in the potential health impacts of these emissions, and how transportation may contribute to them. Concerns about the project development and perhaps the transportation planning process will not likely reduce in the short term, and will likely continue to be the basis of legal challenges to transportation projects.

- Over the next 20 to 30 years, there are likely to be very significant reductions in MSAT emissions. EPA estimates that diesel particulate matter from on-road vehicles will decline by 90 percent in this time frame due to new vehicle and fuel regulations. Other MSATs will decline significantly (by up to two-thirds) as well.
- Despite these reductions, interest in motor vehicle emissions and their potential health impacts may not abate. There is always the possibility that new health dangers related to motor vehicle emissions will be discovered or proposed including those from new technologies introduced into the market place, triggering a new set of concerns or an exacerbation of existing concerns.

Overview of Air Toxics

Air toxics, or hazardous air pollutants as they are called in the Clean Air Act, are emitted into the outdoor air from many different sources and represent a diverse group of air pollutants. They were identified in the authorizing legislation for the 1970 Clean Air Act as “pollutants which present, or may present, through inhalation or other routes of exposure, a threat of adverse human health effects (including, but not limited to, substances which are known to be, or may reasonably be anticipated to be, carcinogenic, mutagenic, teratogenic, neurotoxic, which cause reproductive dysfunction, or which are acutely or chronically toxic)”.

In its Integrated Urban Air Toxics Strategy, US EPA reviewed exposure and health data for all 188 air toxics and identified a list of 33 high-priority hazardous air pollutants, including acetaldehyde, acrolein, benzene, 1,3-butadiene, formaldehyde, and polycyclic organic matter (POM). In its recent National Air Toxics Assessment (NATA) EPA estimated, using 1999 emissions data, that as a result of exposure to these 33 air toxics from outdoor sources, 92% of the U.S. population have some increased risk for respiratory toxicity (including irritation and other adverse respiratory effects). NATA also estimates that in most of the country people have a slightly increased lifetime cancer risk from air toxics (between 1 and 25 in a million) if they were exposed to 1999 levels of these 33 pollutants over the course of their lives. Since 1989, EPA and other regulatory agencies have interpreted cancer risk levels above 1 in a million to be cause for further investigation and possible regulation. Likewise, risk levels of less than 1 in a million have generally been interpreted to be below the level of regulatory concern.

Mobile Source Air Toxics - MSATs represent a subset of air toxics that are emitted by on- and non-road vehicles. By considering pollutants that originate, at least in part, from mobile sources, and taking into account health and risk assessment information contained in the Integrated Risk Information System (IRIS), the EPA had further defined a list of 21 MSATs (see Table 1). The IRIS database was designed to provide consistent, quantitative information on human health effects that may result from exposure to various substances found in the environment for use in risk assessment. Although many of these MSATs are also emitted by non-mobile sources (mobile sources are dominant sources of exposure for only a few on the EPA list of 21 MSATs), U.S. EPA estimates in NATA that mobile sources were responsible for about 44% of total estimated outdoor air toxic emissions, almost 50% of the estimated cancer risk, and 74% of the estimated non-cancer risk (largely derived from acrolein). Seven of these pollutants are included in EPA’s list of 33 high priority hazardous air pollutants: benzene, 1,3 butadiene, acrolein, acetaldehyde, formaldehyde and diesel particulate matter and POM, and all of these, except acetaldehyde (but including naphthalene), were recently identified by EPA as “risk drivers.”

Table 1. MSATs Previously Identified by the U.S. EPA

Acetaldehyde	Lead compounds
Acrolein	Manganese compounds
Arsenic	Mercury compounds
Benzene	Methyl *tert* butyl ether
1,3-Butadiene	Naphthalene
Chromium compounds	Nickel compounds
Diesel engine exhaust	Polycyclic organic matter (POM)
Dioxins and furans	Styrene
Ethylbenzene	Toluene
Formaldehyde	Xylene
n-Hexane	

On March 29, 2006, U.S. EPA proposed to modify its approach to MSAT emissions. The Agency proposed to replace the above list of 21 MSATs with a more specific list of 91 substances (compounds and derivatives) that are listed in U.S. EPA's IRIS database as coming from mobile sources and contributing to cancer risk (see 71 FR 15813).

Regulatory Framework: A US air toxics regulatory program was authorized under the 1970 Clean Air Act and redesigned under the 1990 Clean Air Act amendments. Under this legislation, the U.S. EPA is required to characterize, prioritize and adopt regulations to reduce the impacts on public health and the environment of a list of 188 air toxics from all outdoor sources, including stationary, area, and mobile sources. The 1990 Clean Air Act amendments also contained provisions directed at air toxics from motor vehicle and their fuels. This source-specific regulatory approach is in contrast to that used for the criteria air pollutants (e.g. CO, O_3, and particulate matter) for which national ambient air quality standards (NAAQS) are established and specific requirements are placed on State DOTs and MPOs to help meet those standards.

In section 202(l), the Clean Air Act specifically requires U.S. EPA to regulate or consider regulating air toxics from motor vehicles in the form of standards for fuels, or vehicle engines. This approach of regulating fuels and vehicle emissions, including the introduction of emission control devices such as catalytic converters, has lead to substantial reductions in the emission of air toxics since enactment of the Clear Air Act. Future reductions in air toxics are expected from existing regulatory programs designed to reduce ozone and particulate matter inventories.

In addition, U.S. EPA has recently proposed additional standards to further control air toxics. In March 2006, EPA proposed a rule to reduce hazardous air pollutants from mobile sources; specifically, the rule would limit the benzene content of gasoline and reduce emissions of passenger vehicles and gas cans. The final rule is expected in February 2007. In addition, reformulated and alternative fuels (including fuels using increased amounts of ethanol) are being introduced with expectations of substantial environmental benefits, as their emissions profiles will be different from traditional fuels. These changes are resulting in decreases in the emission of some air toxics but increases in the levels of others.

MSATs and Transportation Projects - In addition to broad public health issues, concerns over health risks of air toxics have arisen during the development of transportation projects. These concerns have focused on whether transportation projects will result in increased exposure to air toxics and increased health risks. Concerns have been raised regarding the overall impact of transportation projects on ambient air levels of air toxics and about the possibility of even higher concentrations of air toxics near transportation facilities.

Under the National Environmental Policy Act (NEPA) agencies are expected to disclose environmental impacts of proposed transportation improvements. Concerns over MSATs have led to requests for analysis of these emissions on several projects nationwide, including projects on highways, transit and airports, and in a few cases to lawsuits over the alleged failure to address the impacts of these emissions. In all suits brought to date on highway projects, FHWA has prevailed. The most significant of these was a suit brought by the Sierra Club on the expansion of US 95 in Las Vegas, alleging inadequate analysis of MSAT emissions. FHWA and Nevada DOT prevailed in district court. An appeal was filed, but the suit was settled before a decision was rendered. A part of the settlement agreement was to conduct a study of the dispersion patterns and other behavioral characteristics of certain MSATs attributed to mobile sources.

In February 2006, FHWA released guidance on the types of analysis under NEPA that should be done to disclose potential MSAT impacts. Although tools and techniques for assessing project-specific health effects from MSATs are limited, FHWA guidance calls for an air toxics emissions analysis in some cases, a qualitative assessment in others, and no analysis for projects that will not likely have any impact on emissions. The FHWA guidance is similar to the approach followed by the FAA. However, air toxics challenges on transportation projects continue. A recent lawsuit filed on the Intercounty Connector in Maryland includes a challenge to the air toxic analysis conducted for the project pursuant to FHWA's guidance.

Assessing Exposure and Health Risk

Many scientists and agencies have investigated and reviewed exposure to and the health effects of air toxics. Given the complex mixtures of these compounds to which people are exposed, these studies have taken four basic forms: (1) measurement studies that attempt to measure ambient levels and personal exposure to the compounds, (2) laboratory toxicology studies in animals and cultured cells designed to test the effects (often at high levels) of single compounds, (3) occupational epidemiology studies that investigate the cancer and health outcomes in populations of workers exposed at moderate levels to one or a few air toxics, and (4) general population or community-level epidemiology studies of effects of exposure to air toxics at ambient levels. There are very few community epidemiology studies explicitly assessing the effects of specific toxic chemicals, given the expense of measurement and laboratory analysis, the difficulty of separating out effects of different correlated pollutants, and the generally low levels of air toxics to which most people are exposed.

Given these sources of data, FHWA has been funding – in partnership with US EPA and the motor vehicle (automobile, truck) and oil industries – a Special Panel of the Health Effects Institute (an organization founded in 1980 to provide impartial and independent research on motor vehicle air pollution) to review the exposure and health issues of air toxics. This work has focused on those emissions deemed most likely to be derived in significant proportion from mobile sources and for which existent data suggested that health effects may be observed at concentrations approaching ambient exposures. The Special Panel is still finalizing its report, but from a workshop conducted earlier this year, it is clear that the Panel has focused on acetaldehyde, acrolein, benzene, 1,3-butadiene, formaldehyde, naphthalene and polycyclic organic matter (POM). Diesel particulate matter was addressed previously in another effort. For each of these MSATs, the Panel is addressing three questions: To what extent are mobile sources a significant source of exposure? Does it cause human health effects? Does it cause human health effects at ambient levels of exposure? A summary of some of the Panel's initial findings, drawn from the briefings to date, follows.

Exposure and Health - Of the key MSATs reviewed by the Panel, source attribution suggests that relative to other sources of air pollution, the contribution of mobile sources to overall exposure is greatest for 1,3-butadiene, followed by benzene, then formaldehyde and acetaldehyde, and then acrolein. Mobile source contributions to overall POM exposure vary depending on the specific species; however, it is clear that mobile sources have an impact on those POM associated with particulate matter.

HEI notes that there is insufficient data with regards to the mobile source contribution to naphthalene. Further it appears likely that they provide a limited contribution to human exposures. Yet mobile sources may be a significant source of naphthalene emissions, perhaps 21 percent according to NATA.

Estimates of likely exposures to some of these compounds can be complicated by both their degradation and formation in the atmosphere. For acetaldehyde and formaldehyde, formation of these compounds occurs in the atmosphere from precursor hydrocarbons, some of which are emitted by mobile sources.

Given that substantial exposures to some air toxics can arise from non-mobile sources (e.g., indoors, smoking, food), regulatory authorities beyond those contained in the Clear Air Act or multi-media approaches may be required to significantly reduce overall human exposure to these agents.

Other organizations, including EPA, the National Toxicology Program within the National Institutes of Health, the National Institute for Occupational Safety and Health, the California Office of Environmental Health Hazard Assessment, the American Council of Governmental Industrial Hygienists, and the International Agency for Research on Cancer (IARC) maintain their own databases on the health effects of these compounds.

Benzene: There is more air monitoring data for benzene than any other MSAT considered in this report. Highest levels have been found at urban roadside and urban in-vehicle locations. Mobile sources are an important component of overall exposure to benzene. Consistent with this observation, levels of personal exposures to benzene appear to be higher but within an order of magnitude of those found in outdoor settings.

There is clear and widely accepted evidence from a variety of occupational epidemiologic studies that risks of leukemia are increased. There is evidence but less certainty concerning other cancers. Extended follow-up further confirms this association; moreover, several new studies of petroleum workers, gas and electric utility workers demonstrate increased leukemia risk at lower estimated exposures than studied previously, but there remains considerable uncertainty as to the lowest concentration that might be associated with adverse effects. Thus, benzene is classified by a number of organizations as a known human carcinogen.

1,3-Butadiene: Mobile sources are the most important contributors to 1,3-butadiene levels in ambient air in most locales. Because of its short atmospheric lifetime (it is highly reactive), 1,3-butadiene concentrations are highest near sources. However, its high reactivity results in production of other MSATs such as formaldehyde, acetaldehyde and acrolein. Several recent studies indicated that indoor concentrations may be higher than outdoor ones, an effect not accounted for by environmental tobacco smoke alone, a known source of exposure. Thus, there may be other important sources of exposure.

1,3-Butadiene is a weak carcinogen in rats, but a potent one in mice, acting in part through direct interactions with target cell DNA. Evidence suggests that these differences are based on metabolism differences, and that humans metabolize 1,3-butadiene more like rats than like mice. 1,3-butadiene is also classified as a probable human carcinogen by EPA.

Continuing updates and reanalysis of a single, albeit large, occupational study provides evidence of the carcinogenicity of 1,3-butadiene to humans at levels found in the synthetic rubber industry. While there is reasonable evidence that working in this industry is associated with elevated risk for leukemia, and that exposure to 1,3-butadiene is responsible for at least some of this increased risk, it has not been possible to completely disentangle the contribution of 1,3-butadiene from other co-exposures found in this workplace setting.

As is the case for almost all carcinogenic air pollutants, there is no direct evidence from general population epidemiology studies of health effects of 1,3-butadiene at ambient levels which are typically much lower than industrial levels. Thus, there is reliance on epidemiology studies done with workers at higher exposure levels than occur in ambient air. Cancer risk associated with 1,3-butadiene is unlikely to have a definable threshold.

Acetaldehyde: Mobile sources are a significant source of exposure to acetaldehyde. Acetaldehdye is both emitted directly and also forms in the atmosphere from photochemical reactions of various hydrocarbon and similar precursor emissions. One precursor of acetaldehyde emissions is ethanol. Concentrations tend to be lowest outdoors, with much higher concentrations (2 to 10-fold) indoors and in vehicles. Acetaldehyde is also present in many foods. Like all aldehydes, acetaldehyde is chemically reactive, causing irritation to the eye, skin and respiratory tract, and induces cellular inflammation. Although carcinogenic in rodents, the human data on the possible carcinogenicity of acetaldehyde are inadequate for hazard identification.

Data on respiratory effects are limited mainly to small clinical investigations using exposure to aerosols of acetaldehyde in asthmatic patients. Effects of exposures to multiple aldehydes, all of which can be irritants of the respiratory tract, are not known. There has been only one, limited epidemiologic study of environmental exposure to acetaldehyde. Indoor sources of acetaldehyde account for most personal exposure, and outdoor ambient levels appear to be far below levels producing irritation, suggesting that it is doubtful that acetaldehyde in outdoor air at concentrations observed in recent years has adversely affected human health.

It is possible, however, that acetaldehyde emissions will increase with current requirements for increased use of ethanol, although the exact impact on future levels is not known. The Federal government is evaluating the evidence for carcinogenicity of acetaldehyde as well as a number of other compounds. It is presently classified as a probable human carcinogen by EPA.

Acrolein: Due to the limited number of studies, the highly reactive nature of acrolein and limitations in sampling methods, the available environmental data for acrolein may not be sufficient to allow for an assessment of ambient, indoor or personal exposures. Surprisingly low concentrations were observed in tunnel studies, a finding at odds with U.S. EPA estimates of overall contributions of acrolein exposures from mobile sources. Substantial mobile source contribution to exposure may result from the formation of acrolein from 1,3-butadiene in the air. Environmental tobacco smoke is a major indoor source. Acrolein is very irritating to the respiratory tract of humans and animals. Chronic inhalation results in inflammation.

Although acrolein may damage DNA, several animal studies have not provided substantive evidence of carcinogenicity. And there are insufficient data to assess the impact of ambient exposures to acrolein on human health. However, it can be noted that measured environmental concentrations and personal exposures are only somewhat lower than concentrations shown to cause irritation.

The EPA NATA assessment concludes there is widespread exposure at levels that would be a concern due to the non-carcinogenic effects. There is no carcinogenicity classification for acrolein due to insufficient health data.

Formaldehyde: Indoor sources appear to dominate formaldehyde exposures, with concentrations 3 to 5-fold higher than outdoor concentrations. Outdoors, roadside urban levels are the highest, suggesting that mobile sources are an important source of outdoor concentrations. However, it appears that formation of formaldehyde in the summer is more important that direct vehicle emissions, as strong seasonal effects are observed. Like the other aldehydes, formaldehyde is an irritant of the eye, skin and respiratory tract of humans with sufficient exposure.

It has been recently been classified as a human carcinogen, in part because of evidence of one type of cancer at levels historically encountered in industrial settings. The underlying mechanisms of this carcinogenicity are not fully understood.

There is limited and inconclusive evidence that indoor exposures to formaldehyde increase the occurrence of asthma symptoms in children. There is no evidence regarding health effects of outdoor exposures to ambient concentrations of formaldehyde, but given the likelihood of expanded use of alternative fuels in the U.S., and probable increases in formaldehyde emissions, some attention should be paid to possible effects of increased emissions from mobile sources in the future. In this regard it is important to note that outdoor formaldehyde levels have increased

4-fold in Brazil in the past few years following expansion of the fleet of vehicles using CNG and widespread use of ethanol-based fuels.

Naphthalene: Naphthalene is the most abundant polycyclic aromatic hydrocarbon (PAH) found in air. Mobile sources are important, but not the dominant source of exposure to naphthalene. There is limited evidence to suggest higher in-vehicle or roadside concentrations of naphthalene. Indoor concentrations are typically 5-10 times higher than ambient levels, and may be derived from environmental tobacco smoke and moth repellents. However, trends towards reduction of these indoor sources may lead to the increased importance of outdoor sources as determinants of exposure.

There is evidence in rodents that exposure to naphthalene leads to inflammation and tumors of the nasal tract. However, there is no occupational data on carcinogenicity in humans and no epidemiological or other types of studies that assess the health effects of mean ambient outdoor concentrations of naphthalene exposures.

POM: Polycyclic organic matter is a term commonly used to describe a mixture of hundreds of chemicals including polycyclic aromatic hydrocarbons (PAHs), their oxygenated products and nitrogen analogs. Some POM is found in the gas phase, some in the particle phase and some in both. Different measurement studies have looked at different combinations of POM; there is no standard exposure- or health-based definition of POM. There is a lack of consistency in PAH groupings or indicator compounds for POM. Mobile sources may be significant contributors to ambient levels of POM in urban settings; however, combustion processes like wood burning, cigarette smoke, road paving and roofing tar as well as char-broiling foods may lead to substantial exposures. Diesel vehicles emit more PAHs than gasoline-fueled vehicles, and cold-starts account for up to 50% PAH emissions of diesel vehicles.

A few PAH components of POM are potent animal carcinogens; some of these are classified as human carcinogens. At high workplace exposures there is sufficient evidence for increased risk of lung cancer in coke oven workers and possibly in the asphalt industry. An association between lung cancer and use of "smoky" coal has also been observed.

Health effects have been reported in highly polluted industrial sites for respiratory, cardiovascular and immune systems, but the linkages to POM are not firm. A review of domestic and international studies on prenatal exposure to PAHs and fetal growth found adverse effects on birth weight associated with mothers' exposure to PAHs during pregnancy.

Food-derived sources of POM are likely to dominate exposures in many settings where wood and industrial fossil fuel combustion is limited. Although some POM compounds are DNA damaging agents, there is only limited information on the cancer risks associated with ambient POM exposures. Initial studies indicate that DNA in newborns may be damaged where the mothers were exposed to higher levels of PAHs in air during pregnancy. However, these results require confirmation.

Diesel Particulate Matter: Information cited here is excerpted from "Research on Diesel Exhaust and other Particles," HEI Program Summary 2003. Evidence accumulated over the past several decades suggests that exposure to airborne urban particulate matter pollution is associated with effects on the cardiovascular and respiratory systems. Some recent evidence suggests that exposure to PM may also exacerbate asthma.

The literature on the effect of diesel exhaust on cancer occurrence has been extensively investigated and reviewed. Long term studies of rats, mice, and hamsters have shown an increase in lung tumors only in rats and only at high levels of exposure. The rat studies showed that high levels of both diesel exhaust and carbon black particles caused lung tumors. Epidemiology studies of occupationally exposed workers have shown a relatively consistent, though weak, association between exposure to diesel exhaust and lung cancer. Most studies were not able to control adequately for possible confounders, however, and generally lacked sufficient data for estimating exposure across the full work experience. EPA has concluded that diesel exhaust is “likely to be carcinogenic to humans by inhalation” but that exposure-response data were too uncertain to derive a quantitative estimate of cancer unit risk. The EPA also acknowledged that it is uncertain “whether the health hazards identified from studies using emissions from older engines can be applied to present-day environmental emissions and related exposures, as some physical and chemical characteristics of the emissions from certain sources have changed over time.”

Conclusions

The assessment of MSATs is a dynamic field with a considerable number of new studies underway and a steady flow of new research results. At current levels in ambient air, EPA estimates that a large fraction of the national population experiences some elevated cancer risk from air toxics. Interest is high from environmental, health and community groups in the potential health impacts of these emissions, and how transportation may contribute to them. It is reasonable to expect such interest to continue in the short term (5 to 10 years). This will likely mean continued concerns will be raised about MSATs in the project development and perhaps the transportation planning process.

During this period, one can also anticipate a continued evolution in knowledge about the health impacts of MSATs and possibly a shifting of emphasis areas for reduction as new information is developed and made known. One new and growing area of research is the assessment of potential health effects near roadways. Recent community epidemiology studies have suggested elevated risk of certain health effects among residents near major roads, including premature mortality, respiratory symptoms, and premature birth. However, in most of these studies, the relevant chemical or physical agents are unclear.

Some quantitative potency estimates for MSATs have been derived from animal models. However, extrapolations of these animal results to humans remain troublesome and often continue to rely on default assumptions in EPA guidelines. Better understanding of the toxicokinetic pathways for MSATs (i.e. how they flow through the body and where they end up), particularly at ambient concentrations, in both animals and humans may provide clearer perspectives on the extent of similarity or dissimilarity of metabolism in laboratory animal species and humans.

Clearly there are gaps in knowledge about these emissions. Benzene may be more harmful than previously thought, but the health impacts of some of the other MSAT emissions, like acetaldehyde, may not be clear, particularly at ambient levels. Future research will likely help clarify where emphasis should be placed.

In the medium term (15 to 30 years), there is likely to be very significant reductions in the MSAT emissions of greatest concern. EPA estimates that diesel particulate matter will decline by 90 percent in this time frame due to new vehicle and fuel regulations. Other MSATs will decline significantly (by up to two-thirds) as well.

Despite these reductions, interest in motor vehicle emissions and their potential health impacts may not abate. There is always the possibility that new health dangers related to motor vehicle emissions will be discovered or proposed triggering a new set of concerns.

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