SAFETEA-LU Provisions Require Assessment of Mitigation to Protect Public Health from Adverse Health Effects of Motor Vehicle Emissions

Statement of John Balbus, MD, MPH

Director, Health Program
Environmental Defense
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The 2005 federal transportation law, SAFETEA-LU, requires metropolitan transportation planners to consider ways to mitigate the adverse health impacts of transportation plans, programs, and projects. Long-standing language in section 109(h) of the federal transportation law requires that highway projects be reviewed for their adverse environmental, social and economic impacts, identifying mitigation strategies to eliminate or minimize these impacts. SAFETEA-LU now requires that not only the adverse effects of individual projects, but also the aggregate effects of all the projects in a regional plan, be discussed in long-range transportation plans. Now that MPOs must consider mitigation options on a regional level, they have a unique opportunity to include mitigation strategies that are not available to the implementing agencies on a corridor by corridor basis. These regional level mitigation strategies can provide a new win-win situation, improving transportation infrastructure and mobility while also improving, rather than degrading, the public's health.

One of the more far-reaching elements of SAFETEA-LU is the requirement that MPOs adopt regional plans that "accomplish [SAFETEA-LU's] objectives," which include: 1) improving mobility, 2) fostering economic growth and development, 3) minimizing fuel consumption, and 4) minimizing air pollution. Metropolitan planners must consider alternatives to the regional plans adopted that might better meet SAFETEA-LU's four objectives. Achieving the fourth goal, the minimization of air pollution, will clearly convey public health benefits as outlined below. In addition, alternatives that seek to achieve all four goals by shifting transportation modes from personal vehicle use to mass transit or active transit have the potential to provide significant public health benefits through increasing physical activity within the region.

A substantial body of peer-reviewed scientific studies documents the deleterious health impacts of mobile source fine particulate matter and air toxics emissions, especially on persons living or attending school near major roadways. Additionally, there is growing scientific evidence that transportation planning decisions affect physical activity rates. Lowered physical activity levels are associated with serious health problems, ranging from obesity and diabetes to cancer, stroke and cardiovascular disease. I have summarized some of the extensive literature pertinent to mitigation of transportation health impacts below.

Epidemiologic Studies Show That Particulate Matter Pollution Has Significant Adverse Health Effects.

Motor vehicles are a substantial source of ambient air pollutants, including fine particulate matter ("$PM_{2.5}$")[1] and toxic air pollutants.[2] $PM_{2.5}$ is a mixture of chemicals and metals

[1] $PM_{2.5}$ refers to particulate matter less than 2.5 microns.

[2] See, e.g., Brauer, et al., *Estimating Long-Term Average Particulate Air Pollution Concentrations: Application of Traffic Indicators and Geographic Information Systems*, 14(2) Epidemiology 228 (2003) (a study conducted in three

that may be composed of liquids, solids, or both. Constituents of $PM_{2.5}$ may include acidic liquids, like sulfuric and nitric acids, organic chemicals including many of the air toxics, and tiny pieces of carbon soot. Because of its small size, $PM_{2.5}$ penetrates deeper into lung tissue than coarse particulate matter ($PM_{10-2.5}$), even passing into the bloodstream. The most recent EPA Criteria Document for Particulate Matter summarizes a substantial number of peer-reviewed, scientific studies that find a clear correlation between fine particulate matter and numerous health effects, including increased risk of fatal heart attacks, strokes, respiratory disease, and cancer.[3]

Pope and Dockery (2006) reviewed the recent epidemiological literature regarding the health effects of particulate matter pollution. Pope and Dockery found that there has been a substantial amount of research corroborating previous findings that particulate air pollution is harmful to human health.[4] Such research includes studies of both short-term and long-term health effects of $PM_{2.5}$, as well as studies about the mechanisms by which $PM_{2.5}$ could cause morbidity and mortality.

Studies relating short-term changes in air pollution exposure to mortality counts indicate that increased exposure to PM pollution correlates with higher mortality rates in the short term.[4] The largest American study on the subject to date, the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), examined air pollution's health effects in 20 and 90 US cities. In both the 20-city and 90-city analyses, the researchers found an average increase of 0.5% in overall mortality per 10 $\mu g/m^3$ increase in $PM_{2.5}$ levels measured the day before death, leading the authors to conclude there is "consistent evidence that the levels of fine particulate matter in the air are associated with the risk of death."[5]

A similar research effort, the Air Pollution and Health: A European Approach (APHEA) project, included multicity modeling studies of 30 regions across Western, Southern, and Eastern Europe.[6] In this study, a 50 to 60 $\mu g/m^3$ increase in PM_{10} was associated with a 0.4% increase in total deaths and a 0.5% increase in both cardiovascular and respiratory deaths,[6] a finding which is consistent with the NMMAPS results.

Other recent studies have strengthened the evidence for long-term, chronic adverse health effects of $PM_{2.5}$ pollution.[4] These recent studies include original analyses and re-analyses of datasets collected by Harvard University (the "Harvard Six Cities" study) and by the American Cancer Society (the "ACS" study). The Harvard Six Cities study reported on a 14- to 16-year prospective follow-up of a cohort of >8000 adults living in six U.S. cities, representing a wide

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sites in Europe demonstrated that most of the variability in annual average concentrations of fine particulate matter was explained by vehicular traffic); Environmental Protection Agency, *Technical Support Document: Control of Emissions of Hazardous Air Pollutants from Motor Vehicles and Motor Vehicle Fuels*, EPA420-R-00-023, Table IV, A-1, p. 81 (2000) (EPA study estimating that motor vehicles accounted for 48% of the national total of benzene emissions, 43% of 1, 3-butadiene, 29% of acetaldehyde, and 24% of formaldehyde). Because air monitors generally do not collect data near roadways, these studies downplay the significance of human exposures to mobile source emissions near roadways.

[3] Environmental Protection Agency, *Air Quality Criteria for Particulate Matter* (EPA/600/P-99/002aF, EPA/600/P-99/002bF)(2004)

[4] Pope & Dockery, Health Effects of Fine Particulate Air Pollution: Lines that Connect, 56 Journal of the Air & Waste Management Association 709 (2006).

[5] Samet, et al., Fine Particulate Air Pollution and Mortality in 20 U.S. Cities, 343 N. Engl. J. Med. 1742 (2000).

[6] Samoli, et al., Estimating the Exposure-Response Relationships between Particulate Matter and Mortality within the APHEA Multicity Project, 113 (1) Environ. Health Perspect. 88 (2005).

range of pollution exposure.[4, 7] The ACS study was also a cohort study, and followed >500,000 adults who lived in <150 metropolitan areas from 1982 through 1989.[4, 8] The ACS study linked individual risk factor data from the ACS, Cancer Prevention Study II with national ambient air pollution data.[4, 8] According to Pope and Dockery, both the Harvard Six Cities and ACS studies provide "compelling evidence" of mortality effects from long-term fine particulate air pollution.[8] Reanalysis of these data affirmed the quality of the datasets and reproduced and validated the original results.[4, 9, 10]

Research on the health effects of air pollution in both the short- and long-term has revealed a more or less linear concentration-response curve for PM and mortality rates within the ranges studied.[4] The lack of a clear threshold below which PM pollution is safe suggests that even modest increases in exposure, even below current standards, may be associated with serious health consequences.

In sum, a large and growing body of epidemiologic evidence indicates that there is an association between PM pollution and adverse health effects. This association is likely to persist down to relatively modest levels of PM pollution.[4] Furthermore, additional laboratory experience has increased confidence that the PM-related cardiopulmonary health effects observed in epidemiology are indeed biologically plausible.[4]

Scientific Evidence for Adverse Health Effects from Mobile Source Air Toxics.

A growing body of scientific studies has emerged showing a strong correlation between exposure to mobile source air toxics and a variety of health impacts.

In December 2000, EPA evaluated mobile source emissions[11] and in March 2001 designated 21 chemicals as mobile source air toxics ("MSATs").[12] EPA selected these chemicals based on a scientific consensus that exposure to the chemicals pose serious threats to health, as reflected by their inclusion in EPA's Integrated Risk Information System ("IRIS") database.[13] To be listed in the IRIS database, a chemical must either be a known, probable, or possible carcinogen or cause significant non-cancer health effects, such as reproductive toxicity or neurotoxicity.

The chemical composition of the MSATs varies widely—ranging from metals to small organic compounds to dioxins—and their health impacts vary as well. Although significant information is available about the health effects of individual MSATs, less is known about the role they play compared to particulate matter, in part because of the difficulty of separately measuring the impact of each component of the toxic soup. A growing body of peer-reviewed scientific literature has identified serious health effects from short-term and long-term exposure to MSATs. Six of the MSATs come primarily from mobile sources (other MSATs have

[7] Dockery, et al., An Association between Air Pollution and Mortality in Six U.S. Cities, 329 N. Engl. J. Med. 1753 (1993).
[8] Pope, et al., Particulate Air Pollution as a Predictor of Mortality in a Prospective Study or U.S. Adults, 151 Am. J. Respir. Crit. Care. Med. 669 (1995).
[9] Krewski, et al., Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report. Health Effects Institute, Cambridge MA (2000).
[10] Krewski, et al., Validation of the Harvard Six Cities Study of Particulate Air Pollution and Mortality, 350 N. Engl. J. Med. 198 (2004).
[11] Environmental Protection Agency, Technical Support Document: Control of Emissions of Hazardous Air Pollutants from Motor Vehicles and Motor Vehicle Fuels, EPA420-R-00-023 (December 2000).
[12] 66 Fed.Reg. 17,229-73 (Mar. 2001), citing *Technical Support Document*, see *id*. n.7.
[13] Technical Support Document, at 36.

significant non-mobile sources). All six have extensive toxicologic data and many have substantial epidemiologic data documenting their health risks.

▪ *Acetaldehyde* is a probable human carcinogen based on studies in which rats and hamsters exposed to acetaldehyde formed nasal and laryngeal tumors, respectively.[14] It is also a potential developmental toxicant.[15] Further, exposure to acetaldehyde leads to irritation of the eyes, skin, and respiratory tract, indicating that it may contribute to worsening of health in people with asthma and other lung diseases.[16]

▪ *Acrolein* is a possible human carcinogen and a potent eye and respiratory-tract irritant. Animals chronically exposed to acrolein develop inflammation of the lungs and nasal passages.[17]

▪ *Benzene* is a known human carcinogen with extensive epidemiologic and toxicologic evidence that it causes leukemia.[18] In addition, benzene is toxic to bone marrow and blood cells, leading to decreased numbers of white and red blood cells.[19]

▪ *1, 3-butadiene* is a known human carcinogen based on epidemiologic evidence. It also causes reproductive and developmental toxicity in animals exposed to long-term, low-level doses.[20]

▪ *Diesel particulate matter ("diesel PM") and diesel exhaust organic gases ("diesel EOG")* is a probable human carcinogen. There are several occupational epidemiologic studies associating diesel PM and EOG exposure with lung cancer, and EPA has estimated a range of cancer risk from a specific level of exposure.[21] The California Office of Environmental Health Hazard Assessment (OEHHA) conducted an independent review and determined a quantitative estimate of cancer risk that falls within the range of EPA estimates.[22]

Diesel PM and EOG also cause respiratory irritation and inflammation.[23] Further, a growing body of laboratory studies shows that exposure to diesel PM worsens allergic responses, leading scientists to speculate that it may have a role in initiating allergic diseases, including asthma.[24] Lastly, diesel PM constitutes a significant portion of ambient fine particulate matter,

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[14] Environmental Protection Agency, *Integrated Risk Information System*, available at http://www.epa.gov/iris/subst/0419.htm#quaoral

[15] Environmental Protection Agency, *Health Assessment Document for Acetaldehyde*, EPA/600/8-86-015A (1987).

[16] Environmental Protection Agency, *Technical Support Document: Control of Emissions of Hazardous Air Pollutants from Motor Vehicles and Motor Vehicle Fuels*, EPA 420-R-00-023 (2000).

[17] Agency for Toxic Substance and Disease Registry, *Toxicological Profile for Acrolein* (1990), available at www.atsdr.cdc.gov/toxprofiles/tp124.html

[18] Environmental Protection Agency, *Carcinogenic Effects of Benzene: An Update* (1998).

[19] Aksoy, *Hematotoxicity, Leukemogenicity and Carcinogenicity of Chronic Exposure to Benzene*, in E. Arinc, J.B. Schenkman, & E. Hodgson, eds., Molecular Aspects of Monooxygenases and Bioactivation of Toxic Compounds pp. 415-34 (1991); Goldstein, *Benzene Toxicity*, 3 Occupational Medicine: State of the Art Reviews 541 (1998); Rothman, et al., *Hematotoxicity Among Chinese Workers Heavily Exposed to Benzene*, 29 Am. J. Ind. Med. 236 (1996).

[20] Environmental Protection Agency, *Health Risk Assessment of 1, 3-Butadiene*, EPA/600/P-8/001A (1998).

[21] See Environmental Protection Agency, *Health Assessment Document for Diesel Engine Exhaust*, EPA/600/8-90/057F (2002), available at http://cfpub2.epa.gov/ncea/cfm/recordisplay.cfm?deid=29060&CFID=474991&CFTOKEN=43362109

[22] California Office of Environmental Health Hazard Assessment, *Proposed Identification of Diesel Exhaust As a Toxic Air Contaminant: Health Risk Assessment for Diesel Exhaust* (1998), available at ftp://ftp.arb.ca.gov/carbis/regact/diesltac/partb.pdf

[23] Pandya, et al., *Diesel Exhaust and Asthma: Hypotheses and Molecular Mechanisms of Action*, 110(Supp. 1) Environ. Health Perspect. 103 (2002).

[24] See, e.g., Nel, et al., Enhancement of Allergic Inflammation by the Interaction Between Diesel Exhaust Particles and the Immune System, 102(4 pt 1) J. Allergy Clin. Immunol. 539 (1998); Diaz-Sanchez, et al., Diesel Exhaust Particles Directly Induce Activated Mast Cells to Degranulate and Increase Histamine Levels and Symptom

which is associated with both acute and chronic cardiovascular toxicity and premature death. Recent reviews suggest that the combination of fine soot, acids, and other toxic chemicals associated with diesel PM leads to significant toxicity.[25]

▪ *Formaldehyde* is a potent eye and respiratory tract irritant that triggers asthma attacks and causes asthma-like symptoms in people without asthma.[26] EPA has classified formaldehyde as a probable human carcinogen, based on animal and human studies showing mainly nasal and upper respiratory cancers with exposure.[27]

While EPA's review of mobile source emissions focused on identifying the potential hazards associated with MSATs, the Multiple Air Toxics Exposure Study, conducted by the South Coast Air Quality Management District, provides pioneering, yet rigorous insight into the magnitude of cancer risk associated with MSATs.[28] This landmark study, which relied on extensive monitoring data collected in and around the Los Angeles area, concluded that the overall cancer risk from air toxics to residents in the area was 1400 per one million, well over the 1:1,000,000 or 10:1,000,000 risk levels normally used by regulatory agencies. Using the estimate of diesel exhaust cancer risk from the California Office of Environmental Health Hazard Assessment, the MATES-II study found that 71% of overall cancer risk from air toxics in this area resulted from exposure to diesel exhaust emissions, 8% from 1, 3-butadiene, and 7% from benzene. Modeled exposure data for air toxics based on EPA's National Air Toxics Assessment reveal similar levels of cancer risk and demonstrate that diesel exhaust is the dominant source of that risk. For example, mobile sources contribute 96% of the cancer risk resulting from exposure to air toxics in Montgomery County, Maryland, with diesel emissions responsible for 85% of the risk.[29]

There is also a strong body of indirect scientific evidence that exposure to mobile source air toxics has a substantial negative effect on human health. Most epidemiologic studies rely on exposure data for criteria air pollutants, such as $PM_{2.5}$ and NO_x, because the data for those pollutants are much more widely available. Because concentrations of air toxics from mobile sources are highly correlated with these criteria pollutants, it may be difficult to separate health effects correlated with exposure to these criteria pollutants from those correlated with exposure to mobile source air toxics. This is particularly true of health effects known to be associated with particular air toxics, such as cancer and respiratory irritation.[30] Thus, to the extent epidemiologic

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Severity, 106(6) J. Allergy Clin. Immunol. 1140 (2000); Diaz-Sanchez, et al., Diesel Fumes and the Rising Prevalence of Atopy: An Urban Legend?, 3(2) Curr. Allergy Asthma Rep. 146 (2003).

[25] Diesel Epidemiology Working Group, *Part I: Report of the HEI Diesel Epidemiology Working Group* in Research Directions to Improve Estimates of Human Exposure and Risk from Diesel Exhaust (2002).

[26] Agency for Toxic Substance and Disease Registry, *Toxicological Profile for Formaldehyde*, available at www.atsdr.cdc.gov/toxprofiles/tp111.html

[27] Enivronmental Protection Agency, *Integrated Risk Information System*, available at http://www.epa.gov/iris/subst/0419.htm#quaoral

[28] SCAQMD, *Multiple Air Toxics Exposure Study: MATES II* (2000), available at http://www.aqmd.gov/matesiidf/matestoc.htm

[29] Green Media Toolshed Scorecard (2006), available at http://www.scorecard.org/env-releases/hap/source-chemicals.tcl?geo_area_id=24031&geo_area_type=fips_county_code

[30] In this respect, motor vehicle emissions are similar to tobacco smoke, which is also a mixture of toxic gases and fine particulate matter. In each case, it is difficult to attribute specific toxicity to specific constituents. Delfino, *Epidemiologic Evidence for Asthma and Exposure to Air Toxics: Linkages Between Occupational, Indoor, and Community Air Pollution Research*, 110(Supp. 4) Environ. Health Perspect. 573, 586 (2002). The extensive epidemiologic literature on indoor environmental tobacco smoke does not rely on measurements of individual constituent chemicals within tobacco smoke, but instead uses substitute measures of exposure. Studies of the health effects of motor vehicle emissions do the same thing.

studies use specific measurements of one or two constituents as proxies for the entire mixture of motor vehicle exhaust, the health effects correlated with these proxies may be correlated in part with the unmeasured air toxics.

A Large Body of Scientific Literature Demonstrates Adverse Health Effects from Exposures to Roadway Traffic

a. Correlation Between Asthma And Attending School Near A Major Roadway.

Two studies specifically investigated the effects of motor vehicle emissions on children attending schools near major roadways. The first study assessed 2509 children from 24 schools located within 400 meters of a major roadway in the Netherlands. The study separately measured truck and car traffic, measured concentrations of $PM_{2.5}$, NO_2, and benzene on the school grounds, and took into account other factors that could cause allergic or respiratory problems, such as parental smoking. The study found that children going to school near roadways with heavy truck traffic were more likely to have allergies to outdoor pollens and to have hyper-reactive airways, and that sensitization to asthma was correlated with $PM_{2.5}$ levels.[31]

The second study looked at 1019 children at 10 school sites in the San Francisco Bay Area. Five schools were located far from or upwind from major freeways, and five were located downwind and near freeways, with traffic loads ranging from 130,000 vehicles per day (i.e., less than the current traffic loads on US 95) to 230,000 vehicles per day, approximately the predicted traffic load on US 95. The study concluded that children attending schools with higher exposure to motor vehicle emissions had an increased risk of being diagnosed with asthma. Notably, significantly higher concentrations of black carbon (a measure of diesel PM and EOG) were measured at the schools downwind from the highways, and concentrations of $PM_{2.5}$ measured at the school located closest to a major freeway were 25% higher than measured at regional air quality monitoring stations (i.e., 15 $\mu g/m^3$ compared to 12 $\mu g.m^3$).[32]

b. Correlation Between Respiratory Disease And Living Near A Major Roadway.

Many studies have found a strong correlation between living near roads with high traffic and asthma. Not only do these studies show that exposure to mobile source emissions may trigger asthma attacks, a growing body of laboratory and epidemiologic literature suggests that mobile source emissions, especially diesel emissions, may play a role in initially causing asthma.

Studies have found a variety of asthma-related health effects correlated with exposure to motor vehicle pollution. One epidemiologic study of 16-year old Hispanic children living in areas of East Los Angeles with very high traffic density assessed the role of air toxics in worsening respiratory function. The study, which separately measured the effect of specific air toxics and criteria air pollutants, found positive correlations between asthma symptoms and air toxics, including benzene, acetaldehyde, diesel exhaust, and formaldehyde.[33]

Other studies show a strong correlation between exposure to mobile source emissions and asthma. A recent study from California showed that children living nearer freeways and with

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[31] Janssen, et al., The Relationship Between Air Pollution from Heavy Traffic and Allergic Sensitization, Bronchial Hyperresponsiveness, and Respiratory Symptoms in Dutch Schoolchildren, 111(12) Environ. Health Perspect. 1512 (2003).

[32] Kim, et al., *Traffic-Related Air Pollution Near Busy Roads: The East Bay Children's Respiratory Health Study*, 170(5) Am. J. Respir. Crit. Care Med. 520 (2004).

[33] Delfino, et al., Asthma Symptoms in Hispanic Children and Daily Ambient Exposures to Toxic and Criteria Air Pollutants, 111(4) Environ. Health Perspect. 647 (2003).

higher modeled exposures from freeway mobile source emissions had a higher risk of being diagnosed with asthma as well as higher medication use and wheezing.[34] One recent study showed higher rates of asthma in people exposed to mobile source emissions.[35] Another study, which followed a group of 3730 children from birth to two years of age and assessed each child's individual exposure to fine particulates and certain constituents of diesel exhaust, found significant correlations between exposure to motor vehicle emissions and upper respiratory infections.[36] A study involving 1068 Dutch schoolchildren found that children, especially girls, were more likely to be diagnosed with asthma and have respiratory symptoms if they lived within 100 meters of a major highway or if they had high exposure to truck traffic. The Dutch study also found a correlation between black carbon levels in school classrooms and respiratory symptoms in children living within 300 meters of a major roadway.[37] A study of children in Taiwan found that physician-diagnosed asthma was associated with traffic-related pollution.[38]

One hypothesis explaining the correlation between exposure to mobile source emissions and asthma is that diesel exhaust increases the risk of developing allergic disease. This hypothesis is supported by epidemiologic studies showing increased rates of allergic sensitization in children with higher exposure to mobile source emissions, especially truck traffic-related pollutants,[39] as well as by a growing body of laboratory evidence showing that components of diesel exhaust augment allergic responses to pollens and other allergens.[40]

Epidemiologic studies have consistently shown that people with higher exposures to roadway air pollutants have more hospitalizations for asthma, more respiratory symptoms, and poorer lung function. A review of 20 studies published between 1993 and 2000, found all but one showed that higher exposures to roadway pollutants, especially heavy-truck exhaust, were correlated with worsened asthma, decreased lung function, and more symptoms of asthma.[41] Subsequent studies have confirmed this correlation. For example, a 2001 study showed that exposure to moderate traffic pollution was associated with increased inflammatory markers and decreased lung function in children.[42] A study in Roxbury, Massachusetts, found that exposure

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[34] Gauderman, et al., *Childhood asthma and exposure to traffic and nitrogen dioxide*, 16(6) Epidemiology 737 (2005).

[35] Kim, et al., *Traffic-Related Air Pollution Near Busy Roads: The East Bay Children's Respiratory Health Study*, 170(5) Am. J. Respir. Crit. Care Med. 520 (2004).

[36] Brauer, et al., Air Pollution from Traffic and the Development of Respiratory Infections and Asthmatic and Allergic Symptoms in Children, 166(8) Am. J. Respir. Crit. Care Med. 1092 (2002).

[37] van Vliet, et al., *Motor Vehicle Exhaust and Chronic Respiratory Symptoms in Children Living Near Freeways*, 74(2) Environ. Res. 122 (1997).

[38] Guo, et al., Climate, Traffic-Related Air Pollutants, and Asthma Prevalence in Middle-School Children in Taiwan, 107(12) Environ. Health Perspect. 1001 (1999).

[39] Brauer, et al., Air Pollution from Traffic and the Development of Respiratory Infections and Asthmatic and Allergic Symptoms in Children. 166(8) Am. J. Respir. Crit. Care Med. 1092 (2002); Janssen, et al., The Relationship Between Air Pollution from Heavy Traffic and Allergic Sensitization, Bronchial Hyperresponsiveness, and Respiratory Symptoms in Dutch Schoolchildren, 111(12) Environ. Health Perspect. 1512 (2003); Wyler, et al., Exposure to Motor Vehicle Traffic and Allergic Sensitization, 11(4) Epidemiology 450 (2000).

[40] Nel, et al., *Enhancement of Allergic Inflammation by the Interaction Between Diesel Exhaust Particles and the Immune System*, 102(4 pt 1) J. Allergy Clin. Immunol. 539 (1998); Diaz-Sanchez, et al., *Diesel Exhaust Particles Directly Induce Activated Mast Cells to Degranulate and Increase Histamine Levels and Symptom Severity*, 106(6) J. Allergy Clin. Immunol. 1140 (2000); Diaz-Sanchez, et al., *Diesel Fumes and the Rising Prevalence of Atopy: An Urban Legend?*, 3(2) Curr. Allergy Asthma Rep. 146 (2003).

[41] Delfino, *Epidemiologic Evidence for Asthma and Exposure to Air Toxics: Linkages between Occupational, Indoor, and Community Air Pollution Research*, Environ. 110(Supp. 4) Environ. Health Perspect. 573 (2002).

[42] Steerenberg, et al., Traffic-Related Air Pollution Affects Peak Expiratory Flow, Exhaled Nitric Oxide, and Inflammatory Nasal Markers, 56(2) Arch. Environ. Health 167 (2001).

to fine particulate matter and polycyclic aromatic hydrocarbons (a constituent of diesel emissions) was associated with asthma hospitalizations.[43] A study from Nottingham, United Kingdom, concluded that living within 90 meters of main roads correlated with an increased risk of wheezing illness in children age 4-11.[44] A study in Munich, Germany, demonstrated that the mobile source emissions of particulate matter and nitrogen dioxide were associated with symptoms such as dry cough at night and cough without infection in children ages 1 and 2.[45] A study in East and West Germany found that during a time period of declining pollution in East Germany (1991-2000), improvements in lung function seen in 5-7 year old children over that time were diminished in children living within 50 meters of a busy roadway.[46] A study in Buffalo, New York, showed that the risk of asthma hospitalization increased with exposure to motor-vehicle emissions.[47] A study in Southeast Toronto demonstrated that the risk of hospital admission for asthma, bronchitis, chronic obstructive pulmonary disease (i.e., emphysema and chronic bronchitis), pneumonia, and upper respiratory tract infection increased with increased exposure to $PM_{2.5}$.[48] A 2002 study in 14 cities also associated increased hospital admissions for chronic obstructive pulmonary disease, heart disease, and pneumonia with particulate matter from motor vehicles.[49] A 2005 study from Germany found 55 year old women living near roadways had a higher risk of developing COPD and having decreased lung function.[50]

c. Association Between Lung Cancer And Living Near A Roadway.

Two studies of individuals living near roadways show a correlation between traffic density and lung cancer. A 2003 study found excess lung cancer risks associated with living near roads.[51] A study in Stockholm found a 40% increase in lung cancer risk for the highest group of average traffic-related NO_2 exposure.[52] Because NO_2 generally is not associated with lung cancer, it is likely the correlation reflects exposure to carcinogenic motor vehicle emissions, such as diesel particulates and other air toxics.

d. Association Between Adverse Reproductive Effects And Exposure To Motor Vehicle Pollutants.

One study demonstrated that long-term exposure to motor vehicle pollutants are correlated with low birth weight and pre-term birth.[53]

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[43] Levy, et al., Fine Particulate Matter and Polycyclic Aromatic Hydrocarbon Concentration Patterns in Roxbury, Massachusetts: A Community-Based GIS Analysis, Environ. 109(4) Environ. Health Perspect. 341 (2001).

[44] Venn, et al., *Living Near a Main Road and the Risk of Wheezing Illness in Children*, 164(12) Am. J. Respir. Crit. Care Med. 2177 (2001).

[45] Gehring, et al., Traffic-Related Air Pollution and Respiratory Health During the First 2 Years of Life, 19(4) Eur. Respir. J. 690 (2002).

[46] Sugiri et al., The influence of large-scale airborne particle decline and traffic-related exposure on children's lung function, 144(2) Environmental Health Perspectives 282 (2006).

[47] Lin, et al., Childhood Asthma Hospitalization and Residential Exposure to State Route Traffic, 88(2) Environ. Res. 73 (2002).

[48] Buckeridge, et al., Effect of Motor Vehicle Emissions on Respiratory Health in an Urban Area, 110(3) Environ. Health Perspect. 293 (2002).

[49] Janssen, et al., *Air Conditioning and Source-Specific Particles as Modifiers of the Effect of PM_{10} on Hospital Admissions for Heart and Lung Disease*, 110 Environ. Health Perspect. 43 (2002).

[50] Schikowski, et al. *Long-term air pollution exposure and living close to busy roads are associated with COPD in women*. 6(1) Respiratory Research 152 (2005).

[51] Nafstad, et al., Lung Cancer and Air Pollution: A 27 Year Follow up of 16,209 Norwegian Men, 58(12) Thorax 1071 (2003).

[52] Nyberg, et al., *Urban Air Pollution and Lung Cancer in Stockholm*, 11(5) Epidemiology 487 (2000).

[53] Wilhelm and Ritz, Residential Proximity to Traffic and Adverse Birth Outcomes in Los Angeles County, California, 1994-1996, 111 Environ. Health Perspect. 207 (2003).

Methods Are Available to Assess Health Impacts from Fine Particulate Matter and Mobile Source Air Toxics.

Proposed metropolitan transportation plans may increase PM and MSAT emissions and the adverse impacts of health exposures related to these. The planning process should consider mitigation strategies and alternatives that might reduce these emissions, exposures, and impacts. This will be possible only if the MPO and other authorities work together to evaluate localized MSAT emissions and concentrations that can be anticipated to result in pollution hot spots close to the highway, especially in the early years of operation of a new transportation plan, when MSAT emissions can be anticipated to be at their highest levels. FHWA has recently asserted it is unable to evaluate localized emission concentrations or health effects because of uncertainties in the MOBILE 6.2 model, especially with respect to diesel particulate matter, and uncertainties surrounding the health effects of MSAT pollutants.[54] However, exposure and risk assessment tools are available to establish the degree of risk roadside populations face from exposure to fine particulate matter and air toxic emissions of motor vehicles and to determine diesel particulate emissions.

Two different methods are available to assess the risks to human health from particulate matter and mobile source air toxics. Both methods combine estimates of exposure with estimates of the "dose-response" function to produce an estimate of risk associated with that exposure.[55] One method is based on epidemiologic data that establishes how the risk of particular health effects changes with exposure to particular pollutants.[56] If epidemiologic data are insufficient (which is the case for many carcinogens), the dose-response function can be obtained from toxicologic experiments measuring the dose-response for rodents, with the results extrapolated to humans and real world exposures.

Based on epidemiologic data for fine particulate matter, EPA has estimated dose-response functions for a large number of health effects, including total mortality, hospitalizations for heart disease and lung disease, hospitalizations for asthma in children, and asthma attacks in children. For example, EPA's regulatory impact analysis for heavy duty diesel standards calculated the change in 13 health effects as a result of reductions in $PM_{2.5}$ emissions from diesel engines because of the new standard.[57] EPA's 1996 criteria document for particulate matter, which was the basis for the 1997 air quality standard for $PM_{2.5}$, assessed four endpoints (short and long-term mortality, hospital admissions for all respiratory causes, and respiratory symptoms) using concentration-response parameters derived from epidemiologic studies.[58] Similar concentration response parameters could be established for projected changes in $PM_{2.5}$ due to the construction of major highways and other transportation facilities, yielding estimates of changes in health effects for individuals in affected neighborhoods.

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[54] Intercounty Connector Final Environmental Impact Study, FHWA, Page IV-328 (2006).

[55] The "dose-response" is an estimate of the risk of a specific health effect in response to a specified dosage, or exposure, of the pollutant.

[56] Using epidemiologic data, one can calculate the "relative risk"—the increase in risk in a "real-world" human population from a measured exposure. When the relative risk is combined with the baseline frequency of the health effect (i.e., in the absence of exposure to the pollutant), one can calculate the increase in the number of cases in response to the increase in exposure.

[57] Environmental Protection Agency, *Heavy-Duty Standards/Diesel Fuel RIA* - EPA420-R-00-026 (Dec. 2000), Table VII-14, p. VII-42

[58] Environmental Protection Agency, *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information*, EPA452-R-96-013 (1996), Table VI-2, p. VI-13

Cancer risks from exposure to MSATs may be determined using a methodology similar to that used in the MATES-II study. Changes in the concentrations of the six priority MSATs may be estimated using EPA's MOBILE6.2 model.[59] For those MSATs with cancer unit risk values in EPA's IRIS database, the estimated concentrations of the individual air toxics from the construction of major highways and other transportation facilities can be combined with the cancer unit risk values to produce estimates of cancer risk from exposure to individual air toxics as well as the total risk from exposure to all toxics combined.

Just because reductions in per vehicle MSAT and criteria pollutant emissions are expected over the next 25 years, transportation agencies and MPOs should not assume that there will be no significant adverse impacts on the human environment from future transportation plans, programs, and projects. Nor can the challenge of assessing exposure at the project level and associated health impacts be used to assert that there is no obligation to consider cancer risks. Such arguments would be misguided. Health risks to populations 20 to 25 years hence, especially cancer risks, will be based on exposures from many years prior to that future time horizon. Because old vehicles are not immediately removed from service, aggregate emissions from new cleaner vehicles and the pre-2007 vehicles likely will continue to increase for a decade or more before total emissions begin to decline. Thus cancer risks due to transportation plans must be modeled on emissions characteristics that will be prevalent over the course of the next two decades, not just based on the emission characteristics that may become prevalent in 20 or 25 years. Moreover, acute and chronic cardiovascular and respiratory health risks will be significant impacts for communities adjacent to a new freeway from its construction onward. It would be far more appropriate to estimate cumulative risks for the coming 20-25 year time horizon rather than assuming no risks prior to that time, and then discounting them because of expected declines in mobile source air toxic emissions.

Transportation planning decisions affect physical activity rates and public health.

Physically inactive lifestyles are one of today's major public health challenges, and comprise a well-documented risk factor for the chronic diseases that kill most Americans.[60] These diseases include coronary heart disease, stroke, some cancers, diabetes, and depression.[60, 61] A survey of peer-reviewed literature shows that land use and transportation planning affect people's choices about travel modes,[60] including their choices of whether to drive or use "active transport," which includes walking, biking, and skating.[60] Planning neighborhoods to encourage active transport would have widespread health benefits. A recent Atlanta-based study linked objectively measured physical activity with objectively measured urban form and showed compelling results.[62] Residents of the most "walkable" areas (with sidewalks along streets, houses built near stores and workplaces, etc.) of the Atlanta region were 2.4 times more likely to get the recommended 30 minutes daily of moderate physical activity than residents of the least walkable areas.[62] Furthermore, research in Asia has documented significant relationships

[59] Environmental Protection Agency, User's Guide to MOBILE6.1 and MOBILE6.2 Mobile Source Emission Factor Model, EPA420-R-03-010 (Aug. 2003), p.16

[60] Sallis et al., Active Transportation and Physical Activity: Opportunities for Collaboration on Transportation and Public Health Research, 38 Transportation Research Part A, 249 (2004).

[61] U.S. Department of Health and Human Services, Physical Activity and Health: A Report of the Surgeon General, U.S. Department of Health and Human Services: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion (1996).

[62] Frank et al., Linking Objective Physical Activity Data with Objective Measures of Urban Form, 28 (2S) American Journal of Preventive Medicine (2005).

between greater active transportation use and positive health indicators like lower body mass index, healthier blood lipid profiles, and lower blood pressure.[60]

Indeed, it is possible to plan neighborhoods so that people will use active transport more often. Experts state that communities which improved active transport conditions often experience significant increases in active transport and related reductions in vehicle travel.[63, 64] One study shows, for instance, that when neighborhoods are planned so that they are more walkable,[60] residents report taking an average of twice as many walking trips per week as people living in less walkable neighborhoods.[60] The extra trips taken in more walkable neighborhoods are estimated to add 15-30 minutes more exercise per person each week, which is roughly equivalent to these people meeting current physical activity guidelines one additional day per week.[60] Conversely, another study shows that sprawled land use patterns are correlated with increased time spent in cars, as well as a higher likelihood of sedentary, overweight and obese residents.[65]

Transportation initiatives can help achieve the health benefits associated with active transport by promoting more walkable communities and creating economic incentives such as Parking Pricing, Pay as You Drive Insurance, etc.[64] According to the Victoria Transportation Policy Institute, 5-10% of automobile trips can be reasonably shifted to active transport in a typical urban area even without implementing new market incentives to reduce automobile travel.[64, 66] If such market incentives are used, typically 10-35% of the reduced trips shift to walking and cycling.[66, 67] Promoting such a shift would not only produce benefits to public health, but also would help realize SAFETEA-LU's goals of increasing mobility and reducing fuel use and emissions.

A 5-10% modal shift to include more active transport would help reduce fuel consumption and carbon emissions due to personal automobiles by 3-7% relative to the projected 2006 baseline.[68] A 35% modal shift would produce even more striking results, reducing fuel use and carbon emissions by automobiles by approximately 23% relative to the baseline.[68] Such reductions will help achieve SAFETEA-LU's stated goals of fuel efficiency and reduced emissions.

Conclusion

[63] PBQD, Data Collection and Modeling Requirements for Assessing Transportation Impacts of Micro-Scale Design, Transportation Model Improvement Program, U.S. Department of Transportation (2000).

[64] Litman, Nonmotorized Transport Planning, Victoria Transport Policy Institute, www.vtpi.org, accessed 31 August 2006 (2006).

[65] Lopez, Russ (2004). "Urban Sprawl and Risk for Being Overweight or Obese," American Journal of Public Health, Volume 94 Issue 9, pp. 1574-1579.

[66] Mackett, How to Reduce the Number of Short Trips by Car, European Transport Conference, Center for Transport Studies, University College London (2000).

[67] Litman, Transportation Elasticities, Victoria Transport Policy Institute, www.vtpi.org, accessed 31 August 2006 (2006).

[68] Fuel consumption projections for light-duty vehicles (personal automobiles) were obtained from the Energy Information Administration Annual Energy Outlook 2006, which is available at http://www.eia.doe.gov/oiaf/aeo/index.html, and was accessed 31 August 2006. It was assumed that 66% of total automobile travel is urban travel. Reductions of 5%, 10%, and 35% were applied to the fuel consumption projections for urban light-duty vehicles. It was assumed that light-duty vehicle fuel efficiency is the same in urban and rural areas.

The scientific base for serious health impacts from traffic-related air pollution and activity-inhibiting transportation planning is strong and continues to grow. These effects include respiratory and cardiovascular illness, stroke, lung cancer, and impaired development related to both localized and regional exposures, as well as the host of serious health problems related to physical inactivity. Methods exist to allow reasonable anticipation, and therefore consideration of mitigation of these effects, both on a project by project basis and on a regional basis. The new mandates within SAFETEA-LU to consider mitigation options on a regional level provide a powerful new opportunity to effectively mitigate adverse health impacts while achieving critical mobility and infrastructure goals. It is essential that MPOs ensure that they are part of the solution to health problems in our communities, not part of the cause.