# Comments of the

# American Lung Association Clean Air Council Clean Air Task Force Earthjustice Environmental Defense Fund Natural Resources Defense Council Sierra Club

# on EPA's Proposed Revisions to the Primary National Ambient Air Quality Standards for Particulate Matter

**Docket ID # EPA-HQ-OAR-2007-0492**

**August 31, 2012**

## Table of Contents

## Summary and Introduction

The American Lung Association, Clean Air Council, Clean Air Task Force, Earthjustice, Environmental Defense Fund, Natural Resources Defense Council, and Sierra Club file these comments on EPA's proposed revisions to the National Ambient Air Quality Standards (NAAQS) for particulate matter (PM), and associated monitoring requirements and implementation issues. The list of preparers is on page 136.

Our organizations strongly support strengthening both the annual average and 24-hour standards for fine particulate matter ($PM_{2.5}$), as well as the ambient air quality standards for $PM_{10}$.

At a minimum, the following primary standards are required for EPA to satisfy its obligations under the Clean Air Act:

- an annual average $PM_{2.5}$ standard of 11 $\mu g/m^3$ or below, with elimination of the spatial averaging loophole;
- a 24-hour $PM_{2.5}$ standard of 25 $\mu g/m^3$, $99^{th}$ percentile;
- a 24-hour $PM_{10}$ standard of at least 50 $\mu g/m^3$, with continued speciation of monitoring data
- accelerated implementation of an extensive roadside monitoring program for fine particles.

The courts have determined that science and public health protection must prevail over any other consideration when EPA sets the final NAAQS for particulate matter. Anything less constitutes an abrogation of the duties and responsibilities that Congress and the American people entrusted to EPA under the Clean Air Act.

Ample scientific evidence supports adopting tighter standards to protect the health of people who are most susceptible to the serious health effects of these pollutants. More than 10,000 peer-reviewed scientific studies have been published since 1997 when EPA adopted the current annual standard. These studies validate and extend earlier epidemiologic research linking both acute and chronic fine particle pollution with serious morbidity and mortality. The newer research has also expanded our understanding of the range of health outcomes associated with PM, and has identified adverse respiratory and cardiovascular health effects at lower exposure levels than previously reported. As discussed and interpreted in the EPA's 2009 *Integrated Science Assessment for Particulate Matter*

(ISA),[1] the new evidence reinforces already strong existing studies and supports the conclusion that $PM_{2.5}$ is causally associated with numerous adverse health effects in humans, at exposure levels far below the current standard. Such a conclusion demands prompt action to protect human health.

Because of the population exposed, the complexity of the components and the wide-ranging health effects, $PM_{2.5}$ is the likely the most lethal air pollutant. Certainly, PM increases the risk of early death from heart disease, lung disease and cancer – the three leading causes of mortality in the U.S.

By EPA's own estimates in the draft Risk and Exposure Assessment, premature mortality attributable to fine particle air pollution ranges from 63,000 to 88,000 deaths each year in the United States.[2]

Earlier this year in the journal *Risk Analysis,* EPA staff published estimates that peg the annual toll from $PM_{2.5}$ at 130,000 premature deaths each year, based on 2005 air quality levels. This same analysis estimated a staggering 1.1 million life years lost among people over age 65, accounting for 7 percent of life years lost in 2005 in this population of elderly Americans. Looking at it another way, this translates into an average shortened lifespan of 8.5 months per individual affected. Further, the analysis estimated 1,800 deaths among babies and infants attributable to PM air pollution.[3]

These are preventable deaths. We have the means to control manmade air pollution to end this unnecessary toll on human life. Further, air pollution control efforts driven by more protective air quality standards for PM have the potential to diminish sickness and suffering. This analysis published in *Risk Analysis* pegged the annual morbidity impacts of $PM_{2.5}$ pollution at tens of thousands of hospital and emergency department visits for cardiac and respiratory causes and millions of asthma exacerbations, bronchitis, and other respiratory symptoms in children.[4]

Revised air quality standards have the potential to alleviate and prevent death, disease, and human suffering to an enormous degree, but only if they are set at levels that are protective of public health. Millions of Americans have pre-existing health conditions that make them particularly susceptible to harm from particulate air pollution. Nearly twenty-six million Americans have asthma, including 7.1

[1] U.S. Environmental Protection Agency. Integrated Science Assessment for Particulate Matter (Final Report). U.S. EPA, Washington, DC, 2009; EPA/600/R-08/139F.
[2] U.S. EPA. Quantitative Health Risk Assessment for Particulate Matter, Second External Review Draft, EPA-452/P-10-001 February 2010.
[3] Fann N, Lamson AD, Anenberg SC, Wesson K, Risley D, Hubbell BJ. Estimating the National Public Health Burden Associated with Exposure to Ambient $PM_{2.5}$ and Ozone. *Risk Analysis* 2012; 32: 81-95.
[4] Fann et al. 2012

million children,[5] 12.7 million have been diagnosed with COPD,[6] 74.7million have cardiovascular disease[7] and 25.8 million have diabetes.[8]

EPA's proposed $PM_{2.5}$ standards, while a step in the right direction, are insufficient to protect public health, including the health of susceptible populations, with an adequate margin of safety as required by the Clean Air Act. We are pleased to see EPA's proposal does not include a completely indefensible proposal to retain the existing annual standard. However, simply lowering the annual standard is not enough to meet the requirements of the law.

In the comments that follow, we will discuss the enormous gap in public health protection afforded by an annual standard of 13 $\mu g/m^3$, at the upper end of the proposed range, compared to the more protective 11 $\mu g/m^3$, as advocated by our organizations. We will also explain why a stronger 24-hour $PM_{2.5}$ standard is needed in addition to more protective annual standard.

Further, we urge you to adopt protective coarse particle standards that will apply nationwide, in accordance with the guidance from the World Health Organization.

We strongly favor initiation of a roadside monitoring program for PM, supplemented by modeling, to accelerate the identification and amelioration of transportation –related hotspots.

As many as 35,000 premature deaths could be prevented annually with an annual standard of 11 $\mu g/m^3$ in combination with a daily standard of 25 $\mu g/m^3$, according to a conservative analysis several of our organizations completed in 2011. This analysis looked at the relative health benefits of alternative $PM_{2.5}$ standards using the same analytical tool employed by EPA, but with more recent air quality data. Using conservative assumptions, the analysis also found that the tighter standards could prevent tens of thousands of cases or exacerbations of heart and lung disease.[9] This analysis demonstrated the powerful impact of with lowering the $PM_{2.5}$ daily standard, in conjunction with the annual average level.

We strongly urge EPA to select a standard based on science, not politics. We have reason to be concerned. EPA's *Policy Assessment* issued in April 2011 recommended standards in the range of 11-12

[5] Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System. 2011.
[6] National Center for Health Statistics. Raw Data from the National Health Interview Survey, United States, 2011. Calculations by the American Lung Association Research and Program Services Division using SPSS and SUDAAN software.
[7] National Center for Health Statistics. Raw Data from the National Health Interview Survey, United States, 2010. Calculations by the American Lung Association Research and Program Services Division using SPSS and SUDAAN software
[8] Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System. 2010.
[9] American Lung Association, Clean Air Task Force, and Earthjustice, 2011. Sick of Soot: How the EPA Can Save Lives by Cleaning Up Particle Pollution. Available at: http://earthjustice.org/sites/default/files/SickOfSoot_2011.pdf

μg/$m^3$ as most strongly supported by the science. [10] The draft proposal package forwarded by EPA to the White House contained a proposed standard of 12 μg/$m^3$. Yet, the proposal emerged from the interagency review process with a range of 12-13 μg/$m^3$. [11] Last minute changes to the proposed level were not accompanied by substantive changes in the text to the proposal. In fact, we find no justification in the preamble for an annual standard as high as 13 μg/$m^3$, other than the vague assertion that uncertainties increase at lower concentrations. Further, the final proposal completely failed to address the Policy Assessment recommendations that if 13 μg/$m^3$ was proposed, then the 24-hour standard should be strengthened as well. The apparent last minute and unsupported insertion of a less protective annual standard to the proposal appears to be political interference, not scientific consideration.

One year ago today, on the Friday before Labor Day, the President blocked the EPA's thoughtful and justified reconsideration of the ozone standards. We were appalled by that clearly political decision. We hope that, with this review, the Administration will chose instead to provide the protection the Clean Air Act requires for the people of the United States.

## Legal Background

### Overview

The Clean Air Act Amendments of 1970 first introduced the requirement to establish enforceable national ambient air quality standards ("NAAQS"). The amendments were intended to be "a drastic remedy to what was perceived as a serious and otherwise uncheckable problem of air pollution." *Union Electric Co. v. EPA*, 427 U.S. 246, 256 (1976). The 1970 amendments "carrie[d] the promise that ambient air in all parts of the country shall have no adverse effects upon any American's health." 116 Cong. Rec. 42381 (December 18, 1970).

[10] U.S. Environmental Protection Agency. Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards. U.S. EPA, Washington, DC, April 2011;EPA-452/R-11-003.

[11] Memo from Nathan J Frey, OMB, to Lydia Wegman, US EPA, re: Summary of Interagency Comments under EOs 12866 and 13563. 06/14/2012. Document ID: EPA-HQ-OAR-2007-0492-0373, available at: http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OAR-2007-0492-0373.

The NAAQS drive the Clean Air Act's requirements for controlling emissions of conventional air pollutants. Once EPA establishes a NAAQS, states and EPA identify those geographic areas that fail to meet the standards. 42 U.S.C. § 7407(d). Each state must prepare an "implementation plan" designed to control pollutant emissions in order to reduce the ambient concentrations of the NAAQS.

The Clean Air Act provides a clear process for establishing the NAAQS. The first step in establishing a NAAQS involves identifying those pollutants, the "emissions of which, in [EPA's] judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare," and "the presence of which in the ambient air results from numerous or diverse mobile or stationary sources . . . ." 42 U.S.C. § 7408(a)(1)(A) and (B). Once EPA identifies a pollutant, it must select a NAAQS that is based on air quality criteria reflecting "the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of such pollutant in the ambient air . . . ." *Id.* § 7408(a)(2).

Primary NAAQS must be set at a level "requisite to protect the public health" with "an adequate margin of safety." 42 U.S.C. § 7409(b)(1). Any standards that EPA promulgates under these provisions must be adequate to (1) protect public health and (2) provide an adequate margin of safety, in order to (3) prevent any known or anticipated health-related effects from polluted air. Further, the statute makes clear that there are significant limitations on the discretion granted to EPA in selecting a level for the NAAQS. In exercising its judgment, EPA must err on the side of protecting public health, and may not consider cost or feasibility in connection with establishing the numerical NAAQS or other important elements of the standard (e.g., form of the standard, averaging time, etc.). The D.C. Circuit summed up EPA's mandate succinctly:

> Based on these comprehensive [air quality] criteria and taking account of the 'preventative' and 'precautionary' nature of the act, the Administrator must then decide what margin of safety will protect the public health from the pollutant's adverse effects – not just known adverse effects, but those of scientific uncertainty or that 'research has not yet uncovered.' Then, and without reference to cost or technological feasibility, the Administrator must promulgate national standards that limit emissions sufficiently to establish that margin of safety.

*American Lung Ass'n v. EPA*, 134 F.3d 388, 389 (D.C. Cir. 1998); *see also Whitman v. Am. Trucking Ass'ns*, 531 U.S. 457, 464-71 (2001). Each of these requirements is discussed in more detail below.

Finally, the courts have signaled that agencies will only be afforded deference where they exercise their technical expertise. For example, decisions that appear to come from the Office of Management and Budget, and not the technical judgments of the EPA, are not entitled to judicial deference. *See, e.g., Pub. Citz. Health Research Group v. Tyson*, 796 F.2d 1479, 1505 (D.C. Cir. 1986).

**NAAQS Must Protect Public Health with an Adequate Margin of Safety**

In setting or revising a NAAQS, section 109 of the Clean Air Act requires that the EPA achieve one thing at minimum: protect public health with an adequate margin of safety. This mandate "carries the promise that ambient air in all parts of the country shall have no adverse effects upon any American's health." 116 Cong. Rec. 42381 (December 18, 1970) (remarks of Senator Muskie, floor manager of the conference agreement).[12] As a result:

> Standards must be based on a judgment of a safe air quality level and not on an estimate of how many persons will intersect given concentration levels. EPA interprets the Clean Air Act as providing citizens the opportunity to pursue their normal activities in a healthy environment.

44 Fed. Reg. 8210 (Feb. 8, 1979). Thus, as EPA has acknowledged, it cannot deny protection from air pollution's effects by claiming that the people experiencing those effects are insufficiently numerous, or that levels that are likely to cause adverse health effects occur only in areas that are infrequently visited.

Likewise, in implementing this mandate, EPA cannot deny protection against adverse health and welfare effects merely because those effects are confined to subgroups of the population or to persons especially sensitive to air pollution. It is inherent in NAAQS-setting that adverse effects are experienced by less than the entire population, and that we do not know in advance precisely which individuals will experience a given effect. As a result, opponents of protective NAAQS sometimes argue that NAAQS-setting involves evaluating "risk" and setting a level of risk that is "acceptable." But where—as here—peer-reviewed science shows that adverse effects stem from a given pollutant concentration, EPA must set NAAQS that protect against those effects with an adequate margin of safety. It cannot, under the guise of risk management, set NAAQS that allow such effects to persist. Indeed, given the scientific evidence documenting the occurrence of adverse effects year after year in numerous individuals at levels allowed by the current NAAQS, risks are by definition "significant" enough to require protection under the Act's protective and precautionary approach. *See* H. Rep. No.

[12] *See also* 116 Cong. Rec. at 32901 (September 21, 1970) (remarks of Senator Muskie) ("This bill states that all Americans in all parts of the Nation should have clean air to breathe, air that will have no adverse effects on their health."); *id*. at 33114 (September 22, 1970) (remarks of Senator Nelson) ("This bill before us is a firm congressional statement that all Americans in all parts of the Nation should have clean air to breathe, air which does not attack their health."); *id*. at 33116 (remarks of Senator Cooper) ("The committee modified the President's proposal somewhat so that the national ambient air quality standard for any pollution agent represents the level of air quality necessary to protect the health of persons."); *id*. at 42392 (December 18, 1970) (remarks of Senator Randolph) ("we have to insure the protection of the health of the citizens of this Nation, and we have to protect against environmental insults -- for when the health of the Nation is endangered, so is our welfare, and so is our economic prosperity"); *id*. at 42523 (remarks of Congressman Vanik) ("Human health and comfort has been placed in the priority in which it belongs -- first place.").

294, 95th Cong., 1st Sess., at 43-51 (1977); *Ethyl Corp. v. EPA*, 541 F.2d 1 (D.C. Cir. 1976). That is all the more true where the effects involved include highly serious ones like death and hospitalization. *See id.* at 18 ("the public health may properly be found endangered … by a lesser risk of a greater harm").

**EPA Must Err on the Side of Protecting Public Health**

Courts have properly characterized the NAAQS as "preventative in nature." *Ethyl Corp.*, 541 F.2d at 15; *see also* H. Rep. No. 294, 95th Cong., 1st Sess., at 49-51 (1977) (explaining amendments designed *inter alia* "[t]o emphasize the preventive or precautionary nature of the act, i.e., to assure that regulatory action can effectively prevent harm before it occurs"). Quite clearly, the Act's mandate requires that in considering uncertainty EPA must err on the side of caution in terms of protecting human health and welfare. As the D.C. Circuit has held, "The Act requires EPA to promulgate protective primary NAAQS even where … the pollutant's risks cannot be quantified or 'precisely identified as to nature or degree.'" *Am. Trucking Ass'ns v. EPA*, 283 F.3d 355, 369 (D.C. Cir. 2002).

**NAAQS Must Guard Against Potential Health Effects**

In keeping with the precautionary and preventative nature of NAAQS, EPA must set a standard that protects against potential health effects—not just those impacts that have been well established by science. *See Am. Trucking Ass'ns*, 283 F.3d at 369 (citing Ozone NAAQS, 62 Fed. Reg. 38857 (section 109(b)(1)'s "margin of safety requirement was intended to address uncertainties associated with inconclusive scientific and technical information ... as well as to provide a reasonable degree of protection against hazards that research has not yet identified")); *see also API v. EPA*, 684 F.3d 1342, 1352 (D.C. Cir. 2012).

In the seminal case on the NAAQS, the D.C. Circuit found that Congress "specifically directed the Administrator to allow an adequate margin of safety to protect against effects which have not yet been uncovered by research and effects whose medical significance is a matter of disagreement." *Lead Indus. Ass'n v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir. 1980). Limited data are not an excuse for failing to establish the level at which there is an absence of adverse effect. To the contrary, "Congress' directive to the Administrator to allow an 'adequate margin of safety' alone plainly refutes any suggestion that the Administrator is only authorized to set primary air quality standards which are designed to protect against health effects that are known to be clearly harmful." *Id.* at 1154-55.

In another case dealing with the "margin of safety" requirement of Clean Air Act section 109, the D.C. Circuit rejected industry's argument that EPA was required to document "proof of actual harm" as a prerequisite to regulation, instead upholding EPA's conclusion that the Act contemplates regulation where there is "a significant risk of harm." *Ethyl Corp.*, 541 F.2d at 12-13. Noting the newness of many human alterations of the environment, the court found:

> Sometimes, of course, relatively certain proof of danger or harm from such modifications can be readily found. But, more commonly, 'reasonable medical concerns' and theory long precede certainty. Yet the statute — and common sense — demand regulatory action to prevent harm, even if the regulator is less than certain that harm is otherwise inevitable.

*Id*. at 25. *Accord*, *Industrial Union Dept. v. American Petroleum Inst.*, 448 U.S. 607, 655-56 (1980) (agency need not support finding of significant risk "with anything approaching scientific certainty," but rather must have "some leeway where its findings must be made on the frontiers of scientific knowledge," and "is free to use conservative assumptions in interpreting the data," "risking error on the side of overprotection rather than underprotection").

**NAAQS Must Protect Vulnerable Subpopulations**

The NAAQS must be set at levels that are not only adequate to protect the average member of the population, but also guard against adverse effects in vulnerable subpopulations, such as children, the elderly, and people with heart and lung disease. In fact, courts have repeatedly found that if a certain level of a pollutant "adversely affects the health of these sensitive individuals, EPA must strengthen the entire national standard." *American Lung Ass'n*, 134 F.3d at 390 (citations omitted); *see also American Farm Bureau Fed'n v. EPA*, 559 F.3d 512, 524 (D.C. Cir. 2009); *Coalition of Battery Recyclers Ass'n v. EPA*, 604 F.3d 613, 618 (D.C. Cir. 2010).

The drafters of the 1970 Clean Air Act Amendments made clear that the millions of Americans subject to respiratory ailments are entitled to the protection of the NAAQS: "Included among those persons whose health should be protected by the ambient standard are particularly sensitive citizens such as bronchial asthmatics and emphysematics who in the normal course of daily activity are exposed to the ambient environment." S. Rep. No. 1196, 91st Cong., 2d Sess., at 10 (1970). As the D.C. Circuit has explained:

> In its effort to reduce air pollution, Congress defined public health broadly. NAAQS must protect not only average healthy individuals, but also "sensitive citizens" – children, for example, or people with asthma, emphysema, or other conditions rendering them particularly vulnerable to air pollution.

*American Lung Ass'n*, 134 F.3d at 390 (citations omitted); *Nat'l Envtl. Devel. Ass'n's Clean Air Project v. EPA*, 2012 WL 2948519 at *6 (D.C. Cir. July 20, 2012). Stated another way, NAAQS must "be set at a

level at which there is 'an absence of adverse effect' on these sensitive individuals." *Lead Indus. Ass'n,* 647 F.2d at 1153.

By the best estimates, 74.7 million Americans have heart disease[13]; 12.7 million have been diagnosed with Chronic Obstructive Pulmonary Disease (COPD) which includes both emphysema and chronic bronchitis;[14] 25.9 million Americans, including 7.1 million children have chronic asthma.[15] Further, according to the Centers for Disease Control (CDC), 26 million adults have diabetes and another 79 million have prediabetes[16], a condition that puts people at increased risk for adverse effects from particulate matter air pollution.[17] Considering that these disease categories alone encompass one-third of the population of the U.S. population, the public health implications are enormous. Children, the elderly, and the poor – are additional populations at increased risk from PM air pollution, according to the ISA. According to the Census Bureau, 46.2 million people live in poverty.[18] []

The standards must set at a level that protects these and other populations with an adequate margin of safety.

**EPA Cannot Consider the Economic Cost of Meeting NAAQS**

In setting or revising a NAAQS, EPA cannot consider the economic impact of the standard—only the impact on public health. Lower courts had long held that costs could not be considered in setting NAAQS, and in 2001, the Supreme Court affirmed this position. Justice Scalia, writing for a unanimous Court, found that the plain language of the statute makes clear that economic costs cannot be considered: "Were it not for the hundreds of pages of briefing respondents have submitted on the issue, one would have thought it fairly clear that this text does not permit the EPA to consider costs in setting the standards." *Whitman*, 531 U.S. at 465.

**EPA Must Give Due Deference to the Advice of CASAC**

[13] Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System, 2010.
[14] CDC. National Center for Health Statistics. National Health Interview Survey Raw Data, United States, 2011. Calculations by the American Lung Association Research and Health Education Division using SPSS and SUDAAN software.
[15] CDC. Behavioral Risk Factor Surveillance System, 2011.
16 CDC. Press Release: Number of Americans with Diabetes Rises to Nearly 26 Million; More than a third of adults estimated to have prediabetes. January 26, 2011. Accessed at http://www.cdc.gov/media/releases/2011/p0126_diabetes.html. August 31, 2012.
[17] Pearson JF, Bachreddy C Shyamprasad S, Goldfine A, Brownstein JS. Association Between Fine Particulate Matter and Diabetes Prevalence in the U.S. *Diabetes Care* 33: 2196-2201, 2010.
[18] U.S. Census Bureau. Income, Poverty, and Health Insurance Coverage in the United States: 2010. September 2011. Available at http://www.census.gov/prod/2011pubs/p60-239.pdf

The Act expressly requires EPA, in developing standards, to consider the advice of the statutorily created Clean Air Scientific Advisory Committee (CASAC) and rationally explain any important departure from CASAC's recommendations. 42 U.S.C. §§ 7409(d)(2)(B), 7407(d)(3). It is not enough for EPA merely to "disagree" with CASAC's findings. EPA must explain its reasoning for not accepting the recommendations of CASAC. Am. Farm Bureau Fed'n, 559 F.3d at 521. Even if the Act did not so require, settled principles of administrative law would require EPA to reconcile any disparity between its standards and those recommended by CASAC. Motor Vehicle Mfrs. Ass'n v. State Farm Mut. Auto. Ins. Co., 463 U.S. 29, 43 (1983).

**D.C. Circuit Remand of the 2006 PM NAAQS**

In 2009, the D.C. Circuit remanded the last revisions of the PM NAAQS adopted by EPA in 2006. *Am. Farm Bureau Fed'n*, 559 F.3d at 539. Among the various defects, the Court found that EPA had failed to justify its decision to ignore the advice of its Clean Air Scientific Advisory Committee and to divide long- and short-term studies in its evaluation of the annual and 24-hour standards. *Id.* at 521-22. The Court recognized that the annual and 24-hour standards work together to change the distribution of harmful exposures. *Id.* at 523. Thus, EPA should consider both long-term and short-term exposures of concern in determining whether the combination of standards will be adequate to protect public health with a margin of safety.

The Court also admonished EPA for failing to look at studies that showed adverse morbidity impacts on sensitive subpopulations. *Am. Farm Bureau Fed'n*, 559 F.3d at 525-26. The Court remanded the standards for "EPA to explain why it believes the NAAQS will provide, as required by the CAA, an adequate margin of safety against morbidity in children and other vulnerable subpopulations." *Id.* at 526.

Finally, the Court rejected EPA's decision not to adopt separate secondary standards to address adverse visibility impacts associated with PM pollution. *Am. Farm Bureau Fed'n*, 559 F.3d at 530. Specifically, the Court concluded: "EPA's failure to identify such a level when deciding where to set the level of air quality required by the revised secondary fine PM NAAQS is contrary to the statute and therefore unlawful." *Id.*

## EPA Review Process and CASAC Review

EPA undertook rigorous review of the new science available since the 1996 review, when the EPA staff scientists and CASAC had recommended a sharp reduction in the annual average standard for PM2.5. In the intervening years, new information has added to our understanding of adverse effects at lower concentrations. Compared to in 1997 when just two long-term cohort studies of PM effects were available to inform the setting of the current standard, there are now more than a dozen long-term cohort studies for the U.S. and other countries, and countless studies of short-term effects.

The Clean Air Scientific Advisory Committee is established under the Clean Air Act to advise EPA on the review of the NAAQS. The CASAC PM Review Panel is made up of 22 scientists including the heads of the nation's leading PM research programs. Researchers from Harvard University, New York University, University of Washington, the University of California and other leading institutions are members of the panel.

Upon request from EPA, the CASAC undertook the most thorough review and vetting of key review documents in recent history.

The review process began in June 2007 with a public workshop to consider new policy-relevant scientific evidence available since the 2006 review and to frame the issues. In March 2008, there was a subsequent workshop for invited experts and the public to review draft chapters of the ISA.

EPA prepared detailed charge letters to CASAC to ensure that the Committee provided input on the key scientific issues at each key juncture. The Committee convened in person or by teleconference on at least seven separate occasions to discuss its review and comments on the following documents:

- Draft Integrated Review Plan (IRP)
- First Draft Integrated Science Assessment (ISA)
- Draft Scope and Methods Plan for Risk and Exposure Assessment
- Second Draft ISA
- First Draft Risk and Exposure Assessment (REA)
- Preliminary draft Policy Assessment (PA)
- Second Draft Exposure and Risk Assessment
- First Draft Policy Assessment
- Second Draft Policy Assessment

All told, the Committee reviewed many thousands pages of technical documents and submitted over 200 hundred pages of detailed comments on the draft documents.

Public comment was invited and considered at each step of the review process.

The final ISA was published in December 2009. Unfortunately, following publication of the final Policy Assessment in April 2011, publication of a notice of proposed rulemaking was delayed by over a year. The American Lung Association challenged this delay in court, resulting in an injunction to propose revisions to the standards by June 2012, and a consent decree to issue final standards by December 14, 2012.

In the meantime, science marches forward and now, two and a half years have elapsed since the publication of the final ISA. The record for this rulemaking, as reflected by the ISA, REA, and PA contain ample evidence demonstrating the need for $PM_{2.5}$ standards of at least 11 $\mu g/m^3$ annual and 25 $\mu g/m^3$ daily. More recent epidemiological studies, conducted under real-world air quality conditions, reinforce the need for prompt action to strengthen the PM air quality standards, and are discussed throughout these comments.

**Current Review: CASAC Conclusion on need to strengthen standards**

Consistent with the CASAC committee in the 2006 review, the current panel has made strong recommendations on the need for more stringent annual and daily standards for $PM_{2.5}$.

> "With regard to the integration of evidence-based and risk-based considerations, CASAC concurs with EPA's conclusion that the new data strengthens the evidence available on associations previously considered in the last round of the assessment of the $PM_{2.5}$ standard. CASAC also agrees that there are significant public health consequences at the current levels of the standard that justify consideration of lowering the $PM_{2.5}$ NAAQS further."[19]

> "CASAC concludes that the levels under consideration are supported by the epidemiological and toxicological evidence, as well as by the risk and air quality information compiled in the *Integrated Science Assessment* (December 2009), *Quantitative Health Risk Assessment for Particular Matter* (June 2010) and summarized in the *Second Draft Policy Assessment.* Although there is increasing uncertainty at lower levels, there is no evidence of a threshold (i.e., a level below which there is no risk for adverse health effects). In addition, these combinations of annual/daily levels may not be adequately inclusive. It was not clear why, for example, a daily

[19] Samet J (2010b). Letter from Dr. Jonathan M. Samet, Chair, Clean Air Scientific Advisory Committee to the Honorable Lisa P. Jackson, Administrator, US EPA. CASAC Review of *Policy Assessment for the Review of the PM NAAQS – First External Review Draft* (March 2010). May 17, 2010.

standard of 30 μg/$m^3$ should only be considered in combination with an annual level of 11 μg/$m^3$. The rationale for the 24- hour/annual combinations proposed for the Administrator's consideration (and the exclusion of other combinations within the ranges contemplated) should be more clearly explained."[20]

**Interpretation of Epidemiological Studies**

The current annual average standard for $PM_{2.5}$ of 15 μg/$m^3$ was set in 1997. At that time there were two major long-term cohort studies and several dozen time-series studies indicating that exposures to $PM_{2.5}$ increased the risk of premature mortality. Since 1997, scientific understanding of the impacts of fine particles on human health has increased exponentially. The 1993 Six Cities Study and the 1995 American Cancer Society cohort study have been independently reanalyzed and validated by the Health Effects Institute, an industry-government partnership, and both studies have been extended to provide additional years of follow-up.

Following the establishment of the 1997 standards, Congress established and funded a 10-year research program administered by EPA with input and oversight by a panel of the National Academy of Sciences. Interdisciplinary PM research centers were established at five leading universities. Over $500 million in research grants were awarded over the ten years, leading to publication of thousands of scientific publications, four reports by the National Academy of Sciences, and major advances in understanding of air pollution health effects. The research findings extended our knowledge of the breadth of health impacts and potential underlying mechanisms.

The Integrated Science Assessment (ISA) prepared for this review was thoroughly vetted by the Clean Air Scientific Advisory Committee (CASAC). With encouragement and guidance from CASAC, EPA has developed a five-level hierarchy for the evaluation of evidence regarding causation. Formalization of this framework has provided an opportunity for a more systematic review of the scientific evidence.

The ISA finds that short-term exposures to fine particulate air pollution are causally related to premature mortality and to cardiovascular effects, and likely causal for respiratory effects. Long-term exposures of $PM_{2.5}$ are deemed to be causal for mortality and cardiovascular effects, likely causal for respiratory effects, and suggestive for reproductive and developmental effects and for cancer, mutagenicity and genotoxicity.[21]

[20] Samet, J (2010a). Letter from Dr, Jonathan M. Samet, Chair, Clean Air Scientific Advisory Committee to the Honorable Lisa P. Jackson, Administrator, US EPA. CASAC Review of Policy Assessment for the Review of the PM NAAQS – Second External Review Draft (June 2010). September 10, 2010.

[21] ISA Table 2-6. Summary of PM causal determinations by exposure duration and health outcome.

Studies Excluded from ISA

Commenters note that the scope of the $PM_{2.5}$ review has been narrowed to exclude a number of categories of relevant studies.

Inexplicably, EPA failed to include a detailed assessment of studies of diesel pollution in its Integrated Science Assessment. Diesel exhaust is the most visible and ubiquitous source of fine particle pollution. It is composed of a toxic stream of gases and particles such as black carbon, and is an important contributor to ambient $PM_{2.5}$ concentrations. This conspicuous omission means that the ISA is incomplete in its consideration of $PM_{2.5}$ health effects, especially those attributable to diesel fine particulate, such as cancer, mutagenicity, and other endpoints.

In June 2012, upon the advice of 24 experts from seven countries, the International Agency for Research on Cancer (IARC), a division of the World Health Organization, updated and upgraded its classification of diesel emissions as a cause of lung cancer and also noted a positive association with increased risk of bladder cancer.[22] Diesel engines are a prominent and visible source of fine particulate air pollution, commonly known as soot.

By failing to consider studies of diesel pollution in its health assessment, EPA came to the erroneous conclusion that the causal relationship between PM and cancer is merely suggestive. This conclusion does not square with the IARC finding that diesel emissions are a known human carcinogen nor with the conclusions of the extended analyses of the Six Cities and ACS cohort studies that report positive and statistically significant associations between $PM_{2.5}$ and lung cancer.

Furthermore, EPA failed to include a comprehensive discussion of the health effects of traffic-related air pollution in the PM ISA. The document references EPA's 2002 assessment of diesel pollution, but its conclusions regarding the carcinogenicity and mutagenicity of diesel do not figure into the causal findings for $PM_{2.5}$.

Because of the glaring absence of roadside monitors for PM, most traffic pollution studies rely on measures such as distance from roadway or traffic count as surrogates for exposure. Were these studies taken into account in the assessment, stronger conclusions would have been reached regarding the range of health effects associated with traffic-generated PM pollution and copollutants.

Recommendations of EPA Staff Scientists on $PM_{2.5}$ in the Current Review

[22] Benbrahim-Tallaa L, Baan RA, Grosse Y, Lauby-Secretan B,, El Ghissassi F, Bouvard V, Guha N, Loomis D, Straif K, on behalf of the International Agency for Research on Cancer Monograph Working Group. Carcinogenicity of diesel-engine and gasoline-engine exhausts and some nitroarenes. *The Lancet Oncology* 2012. 13: 663–664.

In the final Policy Assessment, EPA staff scientists conclude that consideration should be given to revising the current annual $PM_{2.5}$ standard level to a level within the range of 13 to 11 $\mu g/m^3$. **Staff further concludes that the evidence most strongly supports consideration of an alternative annual standard level in the range of 12 to 11 $\mu g/m^3$.**

> In conjunction with consideration of an annual standard in the range of 12 to 11 $\mu g/m^3$, staff concludes it is appropriate to consider retaining the current 24-hour $PM_{2.5}$ standard level at 35 $\mu g/m^3$. In conjunction with consideration of an annual standard level of 13 $\mu g/m^3$, staff concludes there is limited support to consider revising the 24-hour $PM_{2.5}$ standard level to somewhat below 35 $\mu g/m^3$, such as down to 30 $\mu g/m^3$.[23]

According to the Policy Assessment, the upper end of the range for the annual standard 13 $\mu g/m^3$, is justified if the Administrator were to place greater weight on "uncertainties." The Clean Air Act, however, directs EPA to act in a precautionary manner, and set more protective standards in the case of uncertainty.

Furthermore, the staff recommendations in the PA, and the proposal, suggest that standard at the upper end of the range should be coupled with a more stringent 24-hour standard of 30 $\mu g/m^3$.

If a tighter 24-hour standard is appropriate with an annual standard of 30 $\mu g/m^3$, it is also appropriate with an annual standard of 12 or 11 $\mu g/m^3$. That is because the annual average calculation can wash out – and thereby ignore—dangerous peak daily concentrations.

## Scientific Consensus for Stronger Standards

Widespread consensus exists in the scientific and medical community that the current air quality standards for fine particulate matter are not protective of public health. A broad spectrum of public health and medical organizations have endorsed stricter $PM_{2.5}$ standards of 11 $\mu g/m^3$ annual and 25 $\mu g/m^3$ daily, or below. In addition to the American Lung Association, these organizations include the American Heart Association, the American Academy of Pediatrics, the American Thoracic Society, the American Public Health Association, the National Association of City and County Health Officials, Trust for America's Health, and the Asthma and Allergy Foundation of America.[24]

[23] Policy Assessment at p. 2-106.

[24] White House Office of Management and Budget (OMB). Meeting Record Regarding: Review of the National Ambient Air Quality Standards for Particulate Matter. OMB, June 13, 2012. Web link: http://www.whitehouse.gov/omb/2060_meeting_06132012. [Accessed August 31, 2012].

The evidence in this current review shows the breadth of harm PM poses to respiratory health. As Rom and Samet (2006) explained in an editorial in the Journal of the American Thoracic Society, "Small Particles Have Big Effects":

> PM has now been linked to a broad range of adverse health effects, both respiratory and cardiovascular, in epidemiologic and toxicologic research. The diversity of effects may reflect the complexity of airborne PM, which is made up of a rich mixture of primary and secondary particles.[25]

The American Heart Association updated their Scientific Statement in May 2010 to review this growing evidence of PM impacts on cardiovascular health. An independent team of scientists who reviewed research from 2004 through March 2009—all well within the timeframe for this review—concluded:

> Exposure to $PM_{<2.5}$ μm in diameter ($PM_{2.5}$) over a few hours to weeks can trigger cardiovascular disease–related mortality and nonfatal events; longer-term exposure (eg, a few years) increases the risk for cardiovascular mortality to an even greater extent than exposures over a few days and reduces life expectancy within more highly exposed segments of the population by several months to a few years; reductions in PM levels are associated with decreases in cardiovascular mortality within a time frame as short as a few years; and many credible pathological mechanisms have been elucidated that lend biological plausibility to these findings. It is the opinion of the writing group that the overall evidence is consistent with a causal relationship between $PM_{2.5}$ exposure and cardiovascular morbidity and mortality.[26]

The American Academy of Pediatrics concluded in 2004 and [reaffirmed in 2009]:

> Children and infants are among the most susceptible to many of the air pollutants. In addition to associations between air pollution and respiratory symptoms, asthma exacerbations, and asthma hospitalizations, recent studies have found links between air pollution and preterm birth, infant mortality, deficits in lung growth, and possibly, development of asthma.[27]

---

[25] Rom WN and Samet JM. Small Particles with Big Effects. *Am J Respir Crit Care Med* 2006; 173: 365-369.

[26] Brook RD, Rajagopalan S, Pope CA III, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA, Peters A, Siscovick D, Smith SC Jr, Whitsel L Kaufman JD; on behalf of the American Heart Association Council on Epidemiology and Prevention, Council on the Kidney in Cardiovascular Disease, and Council on Nutrition, Physical Activity and Metabolism. Particulate matter air pollution and cardiovascular disease: an Update to the scientific statement from the American Heart Association. *Circulation.* 2010: 121: 2331-2378.

[27] American Academy of Pediatrics Committee on Environmental Health. Ambient Air Pollution: Health Hazards to Children. *Pediatrics* 2004; 114:1699-1707. Reaffirmation of this policy in 2009 can be found at http://pediatrics.aappublications.org/content/125/2/e444.short. Accessed August 24, 2012.

**Expert Elicitation on $PM_{2.5}$ Long-term Studies**

The strong scientific consensus about the causal relationship between fine particles and mortality was evident in the results of an expert elicitation sponsored by EPA. The purpose of the exercise was to formally ascertain expert judgment on the concentration-response relationship between $PM_{2.5}$ and mortality.[28]

Expert elicitation consists of structured interviews designed to elicit an expert's best estimate of the value of a particular outcome, together with her quantitative estimate of the uncertainty in that value. If the experts are well chosen to avoid bias, and the study is carefully conducted, results of expert elicitation can be helpful in characterizing leading scientists' level of confidence in a particular outcome. Here, the expert elicitation process was well-designed, rigorously executed, and of central relevance to EPA's determination in setting the health-based standard for particulate pollution.

The key findings of the expert elicitation are summarized below.

- In this study, 12 leading experts who have done research on the relationship between $PM_{2.5}$ concentrations and premature mortality were interviewed.
- The experts were asked to address the effect of a 1 microgram per cubic meter reduction in annual average $PM_{2.5}$ on annual all-cause mortality in the adult U.S. population.
- The experts were asked to consider the body of epidemiological and toxicological studies of $PM_{2.5}$ health impacts, including studies published early this year.
- The experts' best estimates of the percent reduction in deaths that would correspond to a 1 microgram per cubic meter reduction in annual $PM_{2.5}$ ranged from 0.7 to 1.6%.
- In almost all cases, their estimates were higher than those obtained in the American Cancer Society study (Pope et al. (2002)), on which EPA has relied in past benefits analyses.
- Ten of the 12 experts assigned a 90% or greater probability to the existence of a causal relationship between annual average $PM_{2.5}$ levels and all-cause mortality; one other expert estimated the probability of a causal relationship at 70%.

The Expert Elicitation gives credence to EPA's use of both the Laden (2006) and Krewski (2009) studies in the Risk Assessment.

**Growing Scientific Consensus on Ultrafine Particles**

Some leading European researchers organized an expert elicitation workshop to assess the evidence for a causal relationship between exposure to UFP and health endpoints. An expert elicitation focused

[28] Roman HA, Walker KD, Walsh TL, Conner L, Richmond HM, Hubbell BJ, Kinney PL. Expert judgment assessment of the mortality impact of changes in ambient fine particulate matter in the U.S. *Environ Sci Technol* 2008; 42: 2268-74.

on: 1) the likelihood of causal relationships with key health endpoints, and 2) the likelihood of potential causal pathways for cardiac events.[29]

Based on a systematic peer-nomination procedure, fourteen European experts (epidemiologists, toxicologists and clinicians) were selected, of whom twelve attended. They were provided with a briefing book containing key literature. After a group discussion, individual expert judgments in the form of ratings of the likelihood of causal relationships and pathways were obtained using a confidence scheme adapted from the one used by the Intergovernmental Panel on Climate Change.

The likelihood of an independent causal relationship between increased short-term UFP exposure and increased all-cause mortality, hospital admissions for cardiovascular and respiratory diseases, aggravation of asthma symptoms and lung function decrements was rated medium to high by most experts.

The likelihood for long-term UFP exposure to be causally related to all cause mortality, cardiovascular and respiratory morbidity and lung cancer was rated slightly lower, mostly medium. The experts rated the likelihood of each of the six identified possible causal pathways separately. Out of these six, the highest likelihood was rated for the pathway involving respiratory inflammation and subsequent thrombotic effects.

While EPA declines to take measures to regulate ultrafine particles in this review, the causal conclusions of the European Expert Elicitation panel are stronger than in the ISA, suggesting that action is needed to establish ambient air quality standards for ultrafines in the next review cycle.

$PM_{2.5}$ Annual Average: International Standards

Notably, international standards for airborne particulate matter are stricter than the current and proposed air quality standards in the United States. The updated guidelines adopted by the World Health Organization (WHO) in 2005 are for $PM_{2.5}$ annual standards of 10 $\mu g/m^3$ and daily standards of 25 $\mu g/m^3$.

According to the WHO update:

> "The annual average concentration of 10 $\mu g/m^3$was chosen as the long-term guideline value for $PM_{2.5}$ This represents the lower end of the range over which significant effects on survival were

[29] Knol AB, de Hartog JJ, Boogaard H, Slottje P, van der Sluijs JP, Lebret E, Cassee FR, Wardekker JA, Ayres JG, Borm PJ, Brunekreef B, Donaldson K, Forastiere F, Holgate ST, Kreyling WG, Nemery B, Pekkanen J, Stone V, Wichmann H-E and Hoek G. Expert elicitation on ultrafine particles: likelihood of health effects and causal pathways. *Particle and Fibre Toxicology* 2009, **6**:19. Web link: http://www.particleandfibretoxicology.com/content/pdf/1743-8977-6-19.pdf. [Accessed August 31, 2012]

observed in the American Cancer Society's (ACS) study. Adoption of a guideline at this level places significant weight on the long-term exposure studies that use the ACS and the Harvard Six-Cities data (Dockery et al., 1993; Pope et al., 1995; HEI, 2000, Pope et al., 2002, Jerrett, 2005). In all of these studies, robust associations were reported between long-term exposure to $PM_{2.5}$ and mortality. The historical mean $PM_{2.5}$ concentration was 18 $\mu g/m^3$ (range, 11.0–29.6 $\mu g/m^3$) in the Six- Cities study and 20 $\mu g/m^3$ (range, 9.0–33.5 $\mu g/m^3$) in the ACS study. Thresholds were not apparent in any of these studies, although the precise period(s) and pattern(s) of relevant exposure could not be ascertained. In the ACS study, statistical uncertainty in the risk estimates becomes apparent at concentrations of about 13 $\mu g/m^3$, below which the confidence bounds significantly widen since the concentrations are relatively far from the mean. According to the results of the Dockery et al. (1993) study, the risks are similar in the cities with the lowest long-term $PM_{2.5}$ concentrations (i.e. 11 and 12.5 $\mu g/m^3$). Increases in risk are apparent in the city with the next-lowest long-term $PM_{2.5}$ mean (i.e. 14.9 $\mu g/m^3$), indicating that health effects can be expected when annual mean concentrations are in the range of 11–15 $\mu g/m^3$. Therefore, an annual mean concentration of 10 $\mu g/m^3$can be considered, according to the available scientific literature, to be below the mean for most likely effects. Selecting a long-term mean $PM_{2.5}$ concentration of 10 $\mu g/m^3$also places some weight on the results of daily exposure time-series studies that examine the relationships between exposure to $PM_{2.5}$ and acute adverse health outcomes. In these studies, long-term (i.e. three- to four-year) means are reported to be in the range of 13–18 $\mu g/m^{3.}$ Although adverse effects on health cannot be entirely ruled out below these levels, the annual average WHO AQG value represents that concentration of $PM_{2.5}$ that has not only been shown to be achievable in large urban areas in highly developed countries, but also the attainment of which is expected to significantly reduce the health risks.

The WHO guidelines were established after a worldwide consultation with more than 80 leading scientists and are based on review of thousands of recent studies from all regions of the world. As such, they represent the most widely agreed and up-to-date assessment of health effects of air pollution, and recommended targets for ambient air quality.

State of California

In June 2002, the state of California, after a thorough review, vetted by the Air Quality Advisory Committee, adopted a new annual average standard for $PM_{2.5}$ of 12 $\mu g/m^3$.[30] In the intervening

[30] California Environmental Protection Agency (CalEPA) Air Resources Board (ARB). Particulate Matter – Overview. CalEPA, April 25, 2005. Web link: http://www.arb.ca.gov/research/aaqs/caaqs/pm/pm.htm. [Accessed August 31, 2012].

decade since the California standard was established, substantial new information regarding adverse health effects at lower concentrations supports setting a more protective standard.

## Form of the Annual Average Fine Particle Standard

The "form" of the standard refers to the air quality statistics EPA uses to determine whether an area meets the standards. Commenters support the proposed elimination of spatial averaging to assess compliance with the annual average standard. For most other criteria air pollutants, nonattainment is measured based on the highest reading monitor in the area.

With promulgation of the 2006 PM standards, EPA narrowed but did not eliminate the opportunity for spatial averaging. Now, EPA has proposed to close the spatial averaging loophole based on concerns that it does not provide appropriate protection of public health, and may lead to disproportionate impacts on vulnerable subpopulations within an area. In order to reduce the possibility of hotspots and the resulting environmental justice concerns, spatial averaging must be eliminated.

The effect of spatial averaging is to allow an area to meet the national standards, even if particular portions of the area are especially polluted (hotspots), so long as other portions are sufficiently clean. Thus, spatial averaging allows exposure of people to unhealthy levels of pollution at specific locales even within an area meeting the standard. In order to ensure that people in all parts of the country are *equally safe* from unhealthy air, the agency must promulgate truly *national* ambient air quality standards. Were it not to do so, and instead let areas average their way out of cleanup requirements, EPA could allow particularly polluted areas to remain so, at an unacceptable threat to public health. The Clean Air Act does not permit EPA to create sacrifice zones where the health standard can be exceeded, but rather requires standards that apply "in all parts of the country, whether inhabited or uninhabited."[31] The Act requires EPA to base standards on a safe air quality level, not on estimating how many people are exposed to various air quality levels. 44 Fed. Reg. 8210 (February 8, 1979).

The Clean Air Act and its legislative history further confirm this premise that the NAAQS must protect *all* Americans. The Act's mandate could not be plainer: it requires that primary NAAQS be set at levels which, "allowing an adequate margin of safety, are requisite to protect the public health."[32] This

[31] *Lead Industries Assn, Inc. v. EPA,* 647 F.2d 1130, 1180 (D.C. Cir. 1980).
[32] 42 U.S.C. § 7409(b)(1).

mandate "carries the promise that *ambient air in all parts of the country* shall have no adverse effects upon any American's health."[33] *See also* 44 Fed. Reg. 8210 (February 8, 1979).

Spatial averaging is in direct conflict with the Clean Air Act. Indeed attainment of the NAAQS for most criteria air pollutants is measured against the highest recorded concentration in a given area. Additionally, this provision conflicts with EPA's obligations under the President's Executive Order on Environmental Justice and under federal civil rights statutes to provide low income and minority populations with equal protection under that nation's environmental laws. Moreover, the original reasons for allowing spatial averaging were fundamentally at odds with EPA's obligations under the Clean Air Act, in that they related directly to considerations of cost and feasibility and served *no* purpose related to protecting public health.

Spatial averaging, even under present constraints, could potentially allow areas with hotspots of particulate matter concentrations to avoid nonattainment designations and cleanup requirements. This is an environmental justice concern because poor people are more likely to live near roads, depots, factories, ports, and other pollution sources.

However, merely changing the form of the standard to eliminate spatial averaging is not sufficient to address vulnerable populations and environmental justice concerns. Spatial averaging is not actually used much by state agencies. Therefore there will be little practical effect to the change in the form of the standard. EPA also needs to consider environmental justice concerns when selecting the level of the standards and determining monitoring strategies.

CASAC concurs:

> "CASAC recommends that the provisions that allow for spatial averaging across monitors be eliminated for the reasons cited in the *Policy Assessment."*[34]

The preamble acknowledges that there is a large body of new health effects studies indicating further evidence of the serious adverse health effects of fine particulates. These studies include epidemiologic, toxicological, controlled human exposure, and dosimetry analyses. Because of the serious health effects caused by PM pollution, protecting individuals from potential hotspots of the pollutant is critical. There are numerous smaller areas within cities with elevated levels of $PM_{2.5}$; the

[33] 116 Cong. Rec. 42381 (December 18, 1970) (remarks of Senator Muskie, floor manager of the conference agreement). See also id. at 32901 (September 21, 1970) (remarks of Senator Muskie) ("This bill states that all Americans in *all parts of the Nation* should have clean air to breathe, air that will have no adverse effects on their health."); id. at 33114 (September 22, 1970) (remarks of Senator Nelson) ("This bill before us is a firm congressional statement that *all Americans in all parts of the Nation* should have clean air to breathe, air which does not attack their health." (emphasis added)).

[34] CASAC letter May 17, 2012.

people living and working in these areas who are exposed on a daily basis to high levels of fine particulates deserve to be protected.

**Environmental Justice Considerations Demand that Spatial Averaging be Dropped**

EPA has had an environmental justice office for over a decade. Executive Order 12898, *Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations*, signed in 1994 by President Clinton, directs federal agencies to develop strategies to protect minority and low-income populations from environmental health concerns. The interplay between EPA's Environmental Justice requirements (Executive Order 12898) and the Clean Air Act is critical. Regarding the $PM_{2.5}$ NAAQS, complying with those requirements dictates that EPA take actions to ensure that the form of this standard and its implementation protect minority and low-income populations. The $PM_{2.5}$ hotspots that afflict many areas across the country must be addressed. It is inappropriate to develop a standard that allows spatial averaging across monitors in a certain area to downplay the importance of elevated fine particulates.

Thus, EPA concludes, and we agree, that this is further evidence that the annual $PM_{2.5}$ standard must not allow for spatial averaging across monitors. In order for EPA to meet its Environmental Justice and Clean Air Act requirements dictating that all Americans be protected from environmental health concerns, spatial averaging must be removed from the form of the annual average $PM_{2.5}$ standard.

<u>Elimination of the Spatial Averaging Loophole Does Not Excuse EPA's Obligation under the Clean Air Act to Set Standards that Explicitly Incorporate a Margin of Safety.</u>

There is a further issue with respect to the spatial averaging requirement. EPA claims in the proposal that elimination of spatial averaging will meet its legal obligation with respect to the margin of safety requirement. It reasons that the concentration-response relationships reported in some epidemiological studies are based on exposure estimate derived from an average of local monitors, while the proposed standard would be based on the highest monitor in an area thus providing a "built in" margin of safety. While this may be true for some studies, it plainly is not for many others, including most of the six cities in the Harvard study that relied on single monitors to characterize exposures.[35]

The margin of safety is a critical consideration to standard setting mandated by the Clean Air Act and common to other environmental standard setting activities. Elimination of the spatial averaging requirement does not alleviate EPA's responsibility to set standards that provide a margin of safety.

[35] Personal communication with George Allen, August 19, 2012.

A more principled way to consider the margin of safety concept would be to examine the variability in response among participants in key studies or susceptible populations and to apply a numeric safety factor to account for the increased sensitivity of sensitive subpopulations. For example, in other realms of environmental standard setting, it is common to apply a safety factor of 10 to account for the increased sensitivity of children vs. adults, and additional safety factors for other reasons.[36]

The False Notion of a Controlling Standard

The preamble states that EPA's policy preference is that the annual average standard should be the "controlling" standard. There is no basis in the Clean Air Act for such a determination. The Clean Air Act requires only that the NAAQS achieve public health protection with an adequate margin of safety. It is well-documented that both long- and short-term exposures to $PM_{2.5}$ have serious and sometimes irreversible health impacts. There is no heath protection reason to argue that one standard should be "controlling" as a matter of policy without regard to the health consequences of such a policy. To adopt such a policy ignores the obligation to provide equal protection under the law to all Americans because it would result in uneven protection from air pollution in different localities and regions of the country.

The regional inequities of such a policy are borne out by EPA's analysis in Appendix C of the Policy Assessment. This Table shows that in the Northwestern U.S., 32 percent of the population would be in nonattainment areas with a combined $PM_{2.5}$ standard of 12 $\mu g/m^3$ annual – 35 $\mu g/m^3$ daily (12/35), and 32 percent of the population in nonattainment with a combination standard of 11/35. However, with a less stringent annual standard of 13 $\mu g/m^3$, when combined with a daily standard of 30 $\mu g/m^3$, an estimated 59 percent of the population would live in nonattainment areas and be entitled to protection. The point is that lowering the annual average standard alone in these areas will not address potential peak daily concentrations of 35 $\mu g/m^3$. A similar dynamic is in effect in the Upper Midwest. However due to regional differences in sources of air pollution, meteorology, topography, and seasonal considerations, a different dynamic is seen in other regions of the country.

Appendix C in the PA clearly demonstrates that the an annual average standard of 11 or 12 $\mu g/m^3$ is not controlling in the Northwest and Upper Midwest, and that a daily standard of 30 $\mu g/m^3$ would yield additional protection even in combination with the most lenient option for the annual average standard of 13 $\mu g/m^3$. By the same token, in the Northeast and the Southeast, fewer people would be protected by a standard of 13/30 than by a standard of 11/35 or 12/35.

[36] Office of Pesticide Programs U.S. Environmental Protection Agency Determination of the Appropriate FQPA Safety Factor(s) in Tolerance Assessment. Washington, DC, February 28, 2002.

While EPA properly states that it does not rely on projections of the extent of potential nonattainment areas to select particular standards – its own analysis clearly shows the fallacy of a "controlling standard." EPA must set each standard independently at the level necessary to protect public health. It cannot rely on the annual average standard to protect against peak 24-hour concentrations. That is the job of the 24-hour average standard. There is much variation in $PM_{2.5}$ concentrations over the course of the year. As a result, peak daily concentrations can be averaged out when measured against an annual average standard. However, as we will discuss in detail below, short-term exposures to fine particle pollution at levels below the current standard can have devastating impacts on human health and no assurance is available that these spikes will be controlled by the annual average standard.[37]

CASAC agrees. The Committee noted that the approach presented in the second draft Policy Assessment to identify alternative 24-hour standard levels which focused on peak-to- mean ratios was not relevant for informing the actual level (Samet 2010a, p. 4).

When discussing the Risk Assessment, EPA argues in the preamble:

> "while the alternative 24-hour standard levels considered (when controlling) did result in additional estimated risk reductions beyond those estimated for alternative annual standards alone, these additional estimated reductions are highly variable, in part due to different rollback approaches. Conversely, the Risk Assessment recognizes that alternative annual standard levels, when controlling, resulted in more consistent risk reductions across urban study areas, thereby potentially providing a more consistent degree of public health protection (U.S. EPA, 2010a, p. 5–17)." 77 Fed Reg 38917.

It is valid that different rollback approaches rely on different assumptions and that the choice of a rollback strategy will influence the estimated risk reductions under various scenarios.

In is unclear what the value is of the "consistency" EPA is referring to. Some areas may attain an annual average standard of 11-13 $\mu g/m^3$, hence no additional emissions reductions or attainment planning would be required. However, these areas may experience short-term peak concentrations at levels that pose a risk to the residents, and resulting in unequal protection between communities. The only way to ensure "consistent" protection is to set separate daily and annual standards at levels that will protect public health on their own terms.

**EPA Must Choose a 99th Percentile Form of the 24-hour Standard for $PM_{2.5}$**

[37] If EPA were to set a 24-hour standard in the range being proposed for the annual average standard or below, in line with the health studies, then perhaps the need for a concomitant annual average standard would be obviated. However, there is no indication in the review or in the preamble that suggests EPA is considering such an approach.

A "not to be exceeded" or single exceedance form of the 24-hour standard is warranted under the Act, because these forms provide the most protection for public health. However, of the options that have been analyzed by EPA in the Policy Assessment, we recommend a 99$^{th}$ percentile form rather than the 98th percentile form chosen in the proposal. While EPA did discuss the option of a 99th percentile form, or other more protective forms of the standard, the Agency gave as its only justification that the 98$^{th}$ percentile offered "increased stability."[38] The documentation for the "increased stability" came in a chart showing the distribution of site-level variation[39], but no explanation for why such "stability" provides greater health protection for a standard that is intended to protect against inherently "unstable" episodes of harm. One could assume that if the standard were set at the 95$^{th}$ percentile, it might be even more stable, since it would eliminate more days from concern.

The 98th percentile form of the standard allows for almost a week each year of dangerously unhealthy air. If the standard is averaged over three years, then eighteen days over a three year period could have significantly elevated 24-hour $PM_{2.5}$ concentrations even in an area that meets the EPA standard. In comparison, the 99th percentile form still allows three exceedences per year, and up to 9 days over the three-year averaging period. As discussed elsewhere in these comments, even a few days each year with elevated levels of $PM_{2.5}$ will predictably result in excess morbidity, emergency room visits, hospitalizations, and deaths, allowing- such a large number of exceedences to fall within the allowed range of the standard fails to protect health.

Furthermore, with a 98th percentile form of the standard, there is no upper limit on how high pollution can rise on two percent of the days each year. For these reasons, we call on EPA to adopt a 99$^{th}$ percentile form of the 24-hour standard.

The EPA staff paper included a useful risk assessment comparing the estimated annual mortality reduction that would be achieved from the choice of the 98th vs. the 99th percentile form of the standard. 134 If the EPA chooses to finalize the current proposed combination of annual and 24- hour standards, and also chooses the 98th percentile form, the estimated annual mortality in the urban areas assessed in the EPA staff paper would be 3,700 excess deaths. This number would drop significantly, to 1,760 deaths per year, with a simple change from the 98th percentile form to the 99th (see Figure 7). Still greater reductions would be gained through lowering the daily standard and choosing the 99th percentile form.

The choice of a 99th percentile form of the standard will also send a less confusing message to the public. Many people are now using EPA's Air Quality Index. It is confusing to people to learn that their area is in overall attainment with the EPA 24-hour standard, while also learning that the concentrations

[38] 44 Fed. Reg. 38952.
[39] EPA 2011, p. 2-62.

of $PM_{2.5}$ in their area frequently exceed the EPA standard. This dissonance would be lessened by the choice of a 99th percentile form.

EPA's current explanation for selecting the 98th percentile is inadequate – it fails to comprehensively address the added risk (and loss of life) associated with selecting the 98$^{th}$ instead of the 99th percentile. This determination, as with other decisions regarding selection of an appropriate PM standard, must be based on health-related consideration and not on other considerations (such as cost or technical feasibility). EPA has failed to demonstrate that there is a rational health-based reason for rejecting the 99th percentile – not to mention a justification important enough to sacrifice thousands of additional lives every year. EPA must revisit this arbitrary decision and must select the only reasonable option, a 99th percentile standard.

## Susceptible Populations

EPA has solicited comment on studies that may be responsive to requirements under Executive Order 13045, Protection of Children from Environmental Health and Safety Risks and 12898, Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations. To that end, we are submitting a review of studies that document the risk of PM air pollution to children and to low-income and minority populations. Our purpose in doing so goes beyond satisfying an information request. NAAQS must be set at levels that will protective sensitive populations such as children and low income groups.

Other susceptible populations include pregnant women and their unborn children, the elderly, people with heart disease, lung disease, and diabetes, and people who live near heavily travelled roads and transportation depots.

In addition to the studies reviewed below, there are two 2012 publications of particular note to consideration for susceptible populations that we would like to flag for EPA's consideration:.

- A review article on the impacts of air pollution on the elderly – portions of the abstract are extracted below.[40]
    - Compared to the rest of the population, the elderly are potentially highly susceptible to the effects of outdoor air pollution due to normal and pathological aging. The purpose of the present review was to gather data on the effects on respiratory health of outdoor air pollution in the elderly, on whom data are scarce. These show statistically significant short-term and

[40] Bentayeb M, Simoni M, Baiz N, Norback D, Baldacci, S, Maio S, Viegi, G, Annesi-Maesano I. Geriatric Study in Europe on Health Effects of Air Quality in Nursing Homes GERIE Group, Adverse respiratory effects of outdoor air pollution in the elderly. *The International Journal of Tuberculosis and Lung Disease*, 2012; 16: 1149-1161.

chronic adverse effects of various outdoor air pollutants on cardiopulmonary morbidity and mortality in the elderly. When exposed to air pollution, the elderly experience more hospital admissions for asthma and chronic obstructive pulmonary disease (COPD) and higher COPD mortality than others.

- A new analysis that demonstrates a strong association between $PM_{2.5}$ exposure and diabetes prevalence in the adult U.S. population.[41]

## Risks to children

Children face substantial risks from air pollution—especially particulate matter—as growing evidence confirms. As the American Academy of Pediatrics concluded in 2004 and reaffirmed in 2009:

> Children and infants are among the most susceptible to many of the air pollutants. In addition to associations between air pollution and respiratory symptoms, asthma exacerbations, and asthma hospitalizations, recent studies have found links between air pollution and preterm birth, infant mortality, deficits in lung growth, and possibly, development of asthma.[42]

Strong studies confirm that the standards fail to protect the health of children, from the period of fetal development through adolescence, and provide support for more protective standards than EPA has proposed. Research available to EPA in this review documents those risks. In addition, newer studies add weight to the need for greater protection. We note that many of these endpoints have a stronger association with $PM_{10}$ than $PM_{2.5}$ including in cases where data on both size particles were available. These studies underline the importance for EPA to reconsider its proposed decision to leave the $PM_{10}$ standard unchanged.

### *Prenatal and Neonatal Risks*

Increasing evidence indicates that the risk to children begins before they are born. Not surprisingly, the respiratory system is developing from early in embryonic life, starting as early as 3 weeks and continuing after parturition into adolescence.[43] During this prolonged period of pre- and postneonatal development, the lungs and other developing organs and systems may face higher risk in their immaturity stemming from cell development and metabolic changes.[44] These developing systems, as

[41] Pearson JF, Bachireddy C, Shyamprasad S, Goldfine AB, Brownstein JS. Association between fine particulate matter and diabetes prevalence in the U.S. Diabetes Care 2010; 33 (10): 2196-201.
[42] American Academy of Pediatrics Committee on Environmental Health. Ambient Air Pollution: Health Hazards to Children. *Pediatrics* 2004; 114:1699-1707. Reaffirmation of this policy in 2009 can be found at http://pediatrics.aappublications.org/content/125/2/e444.short. Accessed August 24, 2012.
[43] Josi S and Kotecha S. 2007. Lung Growth and Development. *Early Human Development* 2007. 83:789-794.
[44] Šrám RJ, Bincová B, Demjmek , Bobak M. Ambient Air Pollution and Pregnancy Outcomes: A Review of the Literature. *Environmental Health Perspectives 2005;* 113 (4) 375-382.

well as the exposure of the mother, lead to several possible explanations for these risks, as discussed in the ISA.[45]

Newer research has further analyzed the risk for birth defects and low birth weight associated with particle pollution. Although Šrám et al. (2005) found uncertain evidence for birth defects, evidence is growing that some anomalies may be linked. More recently, a 2011 review by Vrijheid et al found $PM_{10}$ linked to an increased risk of atrial septal defects.[46] The preliminary results of an initial, though not full, meta-analysis of research by the International Collaboration on Air Pollution and Pregnancy Outcomes found an increase of $PM_{10}$ of 10 $\mu g/m^3$ associated with lower birth weight of between 2 and 20 grams.[47]

### *Mortality risks increased during postneonatal period*

Strong evidence warns that particulate matter exposure, especially $PM_{10,}$ increases the risk of death in infants. Glinianaia et al. (2004) in their review of research into infant deaths from particulate matter, found the strongest associations for post-neonatal mortality from respiratory causes and sudden infant death syndrome.[48] In a review of research on pregnancy outcomes, Šrám et al (2005) concluded that the evidence was "sufficient to infer a causal relationship between particulate air pollution and respiratory deaths in the postneonatal period."[49] Looking at infant deaths in Southern California, Ritz et al (2006) found risk of postneonatal death increasing by 7 to 12 percent or greater for each increase of 10 $\mu g/m^3$ in $PM_{10}$ through the first year of life. The risk of death *doubled* after these children had been exposed to at least six months of breathing elevated average levels of $PM_{10}$.[50] Woodruff et al. (2008) found added evidence of increased risk of postneonatal deaths from respiratory-related causes with an increase of $PM_{10}$ of 10 $\mu g/m^3$.[51]

### *Childhood and Adolescent Susceptibility to Particulate Matter Pollution*

As with much of their anatomy, the largest portion of a child's lungs will grow after he or she is born. Eighty percent of the air sacs found in the lungs, called alveoli, develop after birth. The lungs and

[45] U.S. EPA 2009; pp. 7-44 – 7-67.
[46] Vrijheid M, Martinez D, Manzanares S, Dadvand P, Schembari A, Rankin J, Nieuwenhuijsen M. Ambient Air Pollution and risk of Congenital Anomalies: A Systematic Review and meta-analysis. *Environmental Health Perspectives* 2011; 119 (5) 598-606.
[47] Parker JD, Rich DQ, Glinianaia SV et al. The International Collaboration on Air Pollution and Pregnancy Outcomes: Initial Results. *Environmental Health Perspectives* 2011; 119 (7): 1023-1028.
[48] Glinianaia SV, Rankin J, Bell R, Pless-Mulloli T Howel D. Does Particulate Air Pollution Contribute to Infant Death? A Systematic Review. *Environmental Health Perspectives* 2004; 112 (14): 1365-1370.
[49] Šrám et al, 2005.
[50] Ritz B, Wilhelm M, Zhao Y. Air pollution and infant death in southern California. *Pediatrics* 2006; 118:493-502.
[51] Woodruff TJ, Darro LA, Parker JD. Air Pollution and postneonatal Infant Mortality in the United States, 1999-2002. *Environmental Health Perspectives* 2008; 116 (1) 110-115.

alveoli do not fully develop until ages 18 to 22, when a child physically reaches adulthood.[52] [53] In addition, the immune system is still developing in young bodies.[54] Young children have an incomplete respiratory epithelium (a thin protective layer of tissue that lines the respiratory tract) so the same pollution exposure may cause more damage to them.[55] Children contract more respiratory infections than adults, which also seems to increase their susceptibility to air pollution.[56]

Furthermore, children's size and behavior also affect their vulnerability. A resting infant takes in roughly twice the amount of air, proportionate to their body size, as an adult. [57] Children are outside for longer periods and are usually more active when outdoors. Consequently, they inhale more polluted outdoor air than adults typically do.[58]

### *Particulate Matter Associated with Increased Risk of Underdeveloped Lungs*

This period of growth in respiratory capacity means that pollution could impact lung development. Gauderman et al., in the Southern California Children's Health study, looked at the long-term effects of particle pollution on teenagers. Tracking 1,759 children between ages 10 and 18, researchers found that those who grew up in more polluted areas faced an increased risk of having underdeveloped lungs. The average drop in lung function was 20 percent below what was expected for the child's age, similar to the impact of growing up in a home with parents who smoked.[59] Because underdeveloped lungs may never grow or recover to their full capacity, this finding indicates potentially permanent damage with long-term risks.

### *Children suffer significant respiratory effects*

As noted in the ISA, many studies link exposure to PM to new or worsening respiratory symptoms in young children. For example, Peel et al., (2005) found $PM_{10}$ associated with increased risk of upper respiratory infection for infants and children in an investigation into emergency department treatment in the extensive Study of Particles and Health in Atlanta (SOPHIA) investigation.[60] Similarly, Host et al.

---

[52] Dietert RR, Etzel RA, Chen D, et al. Workshop to Identify Critical Windows of Exposure for Children's Health: immune and respiratory systems workgroup summary. *Environmental Health Perspectives* 2000; 108 (supp 3); 483-490.

[53] Josi and Kotecha, 2007.

54 World Health Organization: The Effects of Air Pollution on Children's Health and Development: a review of the evidence. 2005; E86575. Available at http://www.euro.who.int/document/E86575.pdf . [Accessed August 28, 2012]

[55] Trasande L and Thurston GD. The role of air pollution in asthma and other pediatric morbidities. *Journal of Allergy Clinical Immunology;* 2005; 115: 689-699.

[56] WHO, 2005.

57 Trasande and Thurston, 2005

[58] American Academy of Pediatrics Committee on Environmental Health, Ambient Air Pollution: health hazards to children. *Pediatrics* 2004; 114: 1699-1707. Statement was reaffirmed in 2010.

[59] Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, Peters J. 2004. The effect of air pollution on lung development from 10 to 18 years of age. *New England Journal of Medicine* 2004; 351:1057-1067.

[60] Peel JL, Tolbert PD, Klein M, Metzger KB, Flanders WD, Todd K, Mulholland JA, Ryan PB, Frumkin H. Ambient Air Pollution and Respiratory Emergency Department Visits. *Epidemiology* 2005; 16 (1): 164-174.

(2007) found a significant association for childhood respiratory diseases for children up to age 14 with increased coarse particle fractions ($PM_{10-2.5}$), although not for $PM_{2.5}$, in their study in six French cities. [61] Pierse et al., (2006) studied 4,400 children aged 1 to 5 years and found exposure to $PM_{10}$ associated with new and returning cough not due to colds.[62]

In some studies the $PM_{2.5}$ and $PM_{10}$ exposures have had different effects or no effect on children examined, a question that researchers looking at adult exposures have explored and some opponents have cited as negating the entire body of evidence. New research into specific components of PM may further explain the challenges in assessing the impact of such complex pollutants. For example, in the investigation by Patel, et al. (2009) in Dominican and African American New York City neighborhoods, children up to 2 years of age suffered cough and wheezing associated with several $PM_{2.5}$ components in the mix, but not $PM_{2.5}$ as a whole. The association with nickel and vanadium and (in some seasons) elemental carbon held up even after accounting for potential confounders such as smoking in the home. These components were recognized as common pollutants from urban heating oil combustion and traffic pollution.[63]

Looking at the impact of cleaner, healthier air in the large study of 9 communities in Switzerland, Bayer-Oglesby et al. (2005) found strong evidence that the reduced $PM_{10}$ levels over an 11-year period benefited the 9,591 children tracked. The children suffered less from chronic cough, bronchitis, colds and other respiratory symptoms, as well as conjunctivitis, even after controlling for indoor air and other confounders.[64]

### *Increased Risk of Asthma and Allergies in Children*

Beyond the question of worsening respiratory symptoms, some studies provide evidence that PM can increase the risk of new onset asthma or allergies. Nordling et al. (2008) found that in a study of 4,089 Swedish children up to age 4, traffic-related $PM_{10}$ was linked to increased sensitization to inhaled allergens, such as pollen. [65]

---

[61] Host S, Larrieu S, Pascal L, Blanchard M, Declercq C, Fabre P, Jusot JF, Chardon B, Le Tertre A, Wagner V, Prouvost H, Lefranc A. Short-term associations between fine and coarse particles and hospital admissions for cardiorespiratory disease in six French cities. *Occupational and Environmental Medicine* 2008; 65(8): 544-551.

[62] Pierse N, Rushton L, Harris RS, Kuehni CE, Silverman M, Grigg J. Locally-generaged particulate pollution and respiratory symptoms in young children. *Thorax* 2006; 61: 216-220.

[63] Patel M, Hoepner L, Garfinkel R, Chillrud S, Reyes A, Quinn JQ, Perera F, Miller RL. Ambient Metals, Elemental Carbon, and Wheeze and Cough in New York City Children through 24 Months of Age. *American Journal of Respiratory and Critical Care Medicine* 2009; 180: 1107-1113.

[64] Bayer-Oglesby L, Grize L, Gassner M, Takken-Sahli K, Sennhauser FH, Neu U, Schindler C, Braun-Fahrländer. Decline of Ambient Air Pollution Levels and Improved Respiratory Health in Swiss Children. *Environmental Health Perspectives* 2005; 113(11) 1632-1637.

[65] Nordling E, Berglind N, Melén E, Emenius G, Halberg J, Nyberg F, Pershagen G, Svartengren M, wickman M, Bellander T. Traffic-Related Air Pollution and Childhood Respiratory Symptoms, Function and Allergies. *Epidemiology* 2008; 19 (3) 401-408.

Clearly, research has not yet resolved the question of whether air pollution—especially particulate matter--can cause new onset asthma. However, the research available in the ISA and published since then continues to add to the likelihood that the answer to that question is yes. Islam et al., (2007) found that children in the California Children's Health Study who were more likely to have developed asthma were those who lived in areas with higher $PM_{2.5}$ where their lung function was lowered. The higher $PM_{2.5}$ levels reduced the "protective effect of better lung function."[66] Morgenstern et al. (2008) found German six-year olds had an increased risk for asthmatic bronchitis or asthma diagnosis with each 1.0 $\mu g/m^3$ increase in $PM_{2.5}$.[67] Wilhelm et al. (2009) found that children living in some Los Angeles neighborhoods with higher levels of $PM_{10}$ were more likely to have doctor-diagnosed asthma, though not asthma attacks in the previous year.[68] Clark et al. (2010) assessed 37,401 children born in British Columbia in 1999 and 2000, finding a significantly increased risk of asthma diagnosis associated with higher exposures to $PM_{10}$, though not to $PM_{2.5}$.[69]

Newer studies show strong evidence as well. Penard-Morand et al. (2010) found children who had lived in one of two French communities for at least three years in areas with higher modeled $PM_{10}$ had significant higher risk of suffering from asthma, eczema, allergic rhinitis and sensitivity to pollens. Children who had lived there throughout their lives had a strong association with lifetime asthma.[70] Gehrig et al.(2010) also found positive association for $PM_{2.5}$ with a significant increase in incidence of asthma, prevalence of asthma and symptoms in a follow up with their study of a cohort of children at age 8 in the Netherlands. As with the French study, these Dutch children who lived in the same place for their entire lives had an even stronger likelihood of asthma.[71]

### *Worsening Pediatric Asthma*

As noted in the ISA, many studies have linked PM with worsened asthma. In addition, newer studies have also found that association, particularly with $PM_{10}$. In their extensive review and meta-analysis published in 2010, Weinmayr et al. (2010) concluded "clear evidence" that $PM_{10}$ triggered asthma episodes in children, though they did not include an analysis of the impact of $PM_{2.5}$ due to less

---

[66] Islan T, Gauderman WJ, Berhane K, McConnell R, Avol E, Peters JM, Gilliland FD. The Relationship Between Air Pollution, Lung Function and Asthma in Adolescents. *Thorax* 2007; 62:957-963.

[67] Morgenstern V, Zutavern A, Cyrys J, Brockow I, Koletzko S, Krämer U, Behrendt H, Herbarth O, von Berg A, Bauer CP, Wichmann HE, Heinrich J. Atopic Diseases, Allergic Sensitization, and Exposure to Traffic –related Air Pollution in Children. *American Journal of Respiratory and Critical Care Medicine 2008;* 177: 1331-1337.

[68] Wilhelm M, Qian L, Rizt B. 2009. Outdoor Air Pollution, family and neighborhood environmenta, and asthma in LA FANS children. *Health & Place* 2009 15 (1) 25-36.

[69] Clark NA, Demers PA, Karr CJ, Koehoorn M, Lencar C, Tamburic L, Brauer M. Effect of early life exposure to air pollution on development of childhood asthma. *Environmental Health Perspectives* 2010; 118 (2):284-290.

[70] Pénard-Morand C, Rahersion C, Charpin D, Kopferschmitt C, Lavaud F, Caillaud D, Annesi-Maesano I. Long-term exposure to close-proximity ai pollution and asthma and allergies in urban children. *European Respiratory Journal* 2010; 36: 33-40.

[71] Gehring U, Wijga AH, Brauer M, Fischer P, de Jongste JC, Kerkhof M, Oldenwening M, Smit HA, Brunekreef B. Traffic-related Air Pollution and the Development of Asthma and Allergies during the First 8 Years of Life. *American Journal of Respiratory and Critical Care Medicine* 2010; 181: 596-603.

extensive data. This review examined 36 studies from around the world published between 1992 and 2006, including 14 from Europe.[72]

Lung function in children with asthma decreased, even when breathing very low concentrations of $PM_{2.5}$ over the course of a day, according to the findings of a study of urban air pollution in Windsor, Ontario, by Dales et al (2009). Monitoring 182 elementary school children for 28 days, they found that the lung function declined during the day, even though the daily mean was 7.8 $\mu g/m^3$, well below both the current U.S. $PM_{2.5}$ NAAQS, and adjusted for potential confounders. [73]

In a newer follow up to the SOPHIA study, Strickland et al. (2010) found that data on over 10 million Atlanta emergency department visits gave it strong statistical power to identify impacts of $PM_{2.5}$ and its components on pediatric asthma. They found that even at "relatively low levels" a strong association with emergency department visits for asthma with $PM_{2.5}$. $PM_{10}$ and $PM_{10-2.5}$. The mean 24-hour $PM_{2.5}$ was 16.4 $\mu g/m^3$with a 7.4 standard deviation. That low level of $PM_{2.5}$ reinforces "the need for the continued evaluation" of the NAAQS to "ensure that the standards are sufficient to protect susceptible individuals."[74]

## Disparities in the Impact of Air Pollution Calls for More Protection

The burden of air pollution is not evenly shared. Poorer people and some racial and ethnic groups are among those who often face higher exposure to pollutants and who may experience greater responses to such pollution. The nation has long recognized the differences in exposure and impact. As the 1994 Executive Order 12898 required, the EPA must:

> "make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations in the United States."[75]

EPA has pledged to work to reduce these disparities, most recently, in the Plan EJ 2014 issued in September 2011 that set as the Agency's goal to "protect the environment and health in overburdened

[72] Weinmayr G, Romeo E, De Sario M, Weiland SK, Forastiere F. Short-Term Effects of $PM_{10}$ and $NO_2$ on Respiratory Health among Children with Asthma or Asthma-like Symptoms: A Systematic Review and Meta-Analysis. *Environmental Health Perspectives* 2010; 118 (4): 449-457.
[73] Dales R, Chen L, Frescura AM, Liu L, Villeneuve PJ. Acute effects of outdoor air pollution on forced expiratory volume in 1 s: a panel study of schoolchildren with asthma. *European Respiratory Journal* 2009; 34: 316-323.
[74] Strickland MJ, Darrow LA, Klein M, Flanders WD, Sarnat JA, Waller LA, Sarnat SE, Mulholland JA, Tolbert PE. Short-term Associations between ambient Air Pollutants and Pediatric Asthma Emergency Department Visits. *American Journal of Respiratory and Critical Care Medicine* 2010; 182:307-316.
[75] President William J. Clinton. Executive Order 12898, Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations. 59 *Federal Register* 7629, February 16, 1994.

communities."[76] The particulate matter standards are a powerful opportunity to advance those goals. The evidence is strong and growing that particulate matter disproportionately exposes and harms many groups.

In the ISA, EPA concluded appropriately that people with low socioeconomic status should be included in the groups facing higher vulnerability to particulate matter. While the evidence did not clearly indicate higher susceptibility based solely on racial or ethnic groups, some evidence warns that some groups may face higher risks.

Many studies have looked at differences in the impact of air pollution on premature death. Results have varied widely, particularly for effects between racial groups. Some studies have found no differences among races,[77] while others found greater responsiveness for Whites and Hispanics, but not African-Americans,[78] or for African-Americans but not other races or ethnic groups.[79] Other researchers have found greater risk for African-Americans from air toxics, including those pollutants that also come from traffic sources.[80]

In a 2003 review article, O'Neill et al. concluded that there are three broad reasons why disparities may exist.[81] First, groups may face greater exposure to pollution because of factors ranging from racism to class bias to housing market dynamics and land costs. For example, pollution sources may be located near disadvantaged communities, increasing exposure to harmful pollutants. Second, low social position may make some groups more susceptible to health threats because of factors related to their disadvantage. Lack of access to health care, grocery stores and good jobs, poorer job opportunities, dirtier workplaces or higher traffic exposure are among the factors that could handicap groups and increase the risk of harm. Finally, existing health conditions, behaviors, or traits may predispose some groups to greater risk. For example, diabetics were among the groups most at risk from air pollutants,

[76] U.S. Environmental Protection Agency. Plan EJ 2014. U.S. EPA Office of Environmental Justice, Washington, DC, September 2011. Web link: http://www.epa.gov/environmentaljustice/resources/policy/plan-ej-2014/plan-ej-2011-09.pdf. Downloaded on August 15, 2012.

[77] Zeka A, Zanobetti A, Schwartz J. Individual-Level Modifiers of the Effects of Particulate Matter on Daily Mortality. *American Journal of Epidemiology* 2006: 163: 849-859.

[78] Ostro B, Broadwin R, Green S, Feng WY, Lipsett M. Fine particulate air pollution and mortality in nine California counties: results from CALFINE. *Environmental Health Perspectives* 2006: 114: 29-33; Ostro B, Feng WY, Broadwin R, Malig B, Green S, Lipsett M. The Impact of Components of Fine Particulate Matter on Cardiovascular Mortality in Susceptible Subpopulations. *Occup Environ Med* 2008*;* 65(11):750-6.

[79] Bell et al., 2008.

[80] Apelberg BJ, Buckley TJ, White RH. Socioeconomic and Racial Disparities in Cancer Risk from Air Toxics in Maryland. *Environmental Health Perspectives* 2005: 113:693-699.

[81] O'Neill MS, Jerrett M, Kawachi I, Levy JI, Cohen AJ, Gouveia N, Wilkinson P, Fletcher T, Cifuentes L, Schwartz J. Health, Wealth, and Air Pollution: Advancing Theory and Methods. *Environmental Health Perspectives* 2003; 111(16)1861-1870.

and the elderly, African-Americans, Mexican-Americans and people living near a central city had higher incidence of diabetes.[82]

Socioeconomic position has been more consistently associated with greater harm from air pollution. Lower education levels were associated with the increased risk of premature death from PM in evidence from the large American Cancer Society database[83] and the Harvard Six Cities[84] database, as recognized in EPA's *Policy Assessment*.[85]

Recent studies show increased evidence of that link. Low socioeconomic status consistently increased the risk of premature death from fine particle pollution among 13.2 million Medicare recipients Zeger et al. (2008) studied in the largest examination of particle pollution mortality nationwide.[86] In the Bell et al. (2008) study that found greater risk for premature death for African-Americans, researchers also found greater risk for people living in areas with higher unemployment or higher use of public transportation.[87] Babin et al, in their 2008 study of Washington, DC, found that while poor air quality and worsened asthma went hand-in-hand in areas where Medicaid enrollment was high, the areas with the highest Medicaid enrollment did not always have the strongest association of high air pollution and asthma attacks.[88] Looking at Toronto, Canada, Burra et al. (2009) found that higher $PM_{2.5}$ levels were associated with increased physician visits for asthma, especially for those in the low socioeconomic group.[89]

Newer research published since the ISA shows that EPA is correct to include people in low socioeconomic groups in the list of susceptible populations. Exposure to particulate matter appears to be significantly greater for lower income communities. Brochu et al. (2011) looked at census tracts in six states in the Northeast and found that places with lower socioeconomic populations had higher

82 O'Neill et al., 2003.

[83] Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association 2002;* 287: 1132-1141; Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, Turner MC, Pope AC 3rd. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Cambridge, MA: Health Effects Institute 2009.

[84] Krewski et al., 2009.

[85] U.S. Environmental Protection Agency. Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards. U.S. EPA, Washington, DC, April 2011;EPA-452/R-11-003:2-30.

[86] Zeger SL, Dominici F, McDermott A, Samet J. Mortality in the Medicare Population and Chronic Exposure to Fine Particulate Air Pollution in Urban Centers (2000-2005). *Environmental Health Perspectives* 2008: 116:1614-1619.

[87] Bell et al., 2008.

[88] Babin S, Burkom H, Holtry R, Tabernero N, Davies-Cole J, Stokes L, Dehaan K, Lee D. 2008. Medicaid Patient Asthma-Related Acute Care Visits And Their Associations with Ozone and Particulates in Washington, DC, from 1994-2005. *International Journal of Environmental Health Research* 2008; 18(3)209-221.

[89] Burra TA, Moineddin R, Agha MM, Glazier RH. Social disadvantage, air pollution and asthma physician visits in Toronto, Canada. *Environmental Research* 2009; 109 (2009) 567-574.

levels of particulate matterexposure.[90] Examining all counties with $PM_{2.5}$ monitors, Miranda et al. (2011), found that counties with a higher percentage of poverty had a higher likelihood that these counties would have the worst air quality for both annual and daily $PM_{2.5}$, a relationship that did not hold for ozone[91]. Bell et al. (in press 2012) reviewed exposure by census tracts and found, "the highest $PM_{2.5}$ exposures for non-Hispanic blacks, the least educated, the unemployed, and those in poverty," although the differences between those groups and whites were "small in magnitude."[92]

New evidence published since the ISA adds to the need for improved near-road monitoring of PM. Jephcote and Chen (2011) found that inner city children in Leicester, England faced a "double burden" of roadway emissions of $PM_{10}$ and low income, increasing their risk of hospitalization for respiratory conditions.

This echoes the findings of the research by O'Connor et al. (2008) that found that near-roadway concentrations of $PM_{2.5}$ were associated with significantly lower pulmonary function in the low income children with asthma included in the Inner City Asthma Study.[93] Although the discussion in the ISA reported that this study found that "$PM_{2.5}$ concentration was not statistically associated with respiratory symptoms in this study" (p. 6-85), that conclusion is not consistent with the findings actually reported in the study which found "a significant association between decrements in lung function and increments in $PM_{2.5}$," particularly in the 5-day average readings.[94]

To reduce the burden on the people who have long suffered more than others in society from the harms of particulate matter, EPA should set a much more protective standard and expand the network of near-road monitoring above the levels currently proposed.

## EPA must select a more protective Annual $PM_{2.5}$ Standard

Strong, new evidence and more detailed analyses available to EPA in this review present a compelling case for the selection of a $PM_{2.5}$ annual average standard of 11 $\mu g/m^3$. As our review of these studies

90 Brochu PJ, Yanosky JD, Paciorek CJ, Schwartz J, Chen JT, Herrick RF, Suh HH. Particulate Air Pollution and Socioeconomic Position in Rural and Urban Areas of the Northeastern United States. *American Journal of Public Health* 2011; 101 (S1) S224-S230.

[91] Miranda ML, Edwards SE, Keating MH, Paul CJ. Making the Environmental Justice Grade: The Relative Burden of Air Pollution Exposure in the United States. *International Journal of Environmental Health Research* 2011; 8: 1755-1771.

[92] Bell ML and Ebisu K. Environmental Inequality in Exposures to Airborne Particulate Matter Components in the United States. *Environmental Health Perspectives*2012; doi:10.1289/ehp.1205201. Web link: http://ehp03.niehs.nih.gov/article/fetchObjectAttachment.action;jsessionid=013DBBB173FA1FBB7856371FAC4437A5?uri=info%3Adoi%2F10.1289%2Fehp.1205201&representation=PDF [Accessed August 24, 2012].

[93] O'Connor GT, Neas L, Vaughn B, Kattan M, Mitchell H, Crain EF, Evans R, Gruchalla R, Moragn W, Stout J, Adams GK and Lippmann M. Acute respiratory health effects of air pollution on children with asthma in US inner cities. *Journal of Allergy and Clinical Immunology* 2008; 121(5):1133-1139.e.1.

[94] O'Connor et al. 2008.

will show, the evidence clearly demonstrates that neither retaining the existing standard of 15 $\mu g/m^3$ nor selecting an annual standard of either 13 or 12 $\mu g/m^3$ would meet the legal requirement to protect public health.

## Multiple Studies Provide Repeated, Consistent Evidence That the Annual Standard Must be Strengthened

Evidence during the last review showed clearly that the annual average standard needed to be much lower than the standard of 15 $\mu g/m^3$ that was first set in 1997.[95] The evidence has only grown since then. Multiple, multi-city studies over long periods of time have shown clear evidence of premature death, cardiovascular and respiratory harm as well as reproductive and developmental harm at contemporary concentrations far below the level of the current standard.

### *Extended Analyses of the ACS Cohort*

Stronger evidence of the association between mean long-term ambient $PM_{2.5}$ concentrations and mortality at levels well below the proposed standards came in a major extension of the prospective study of the large cohort of 360,000 people.[96]

Using data drawn from the nearly 1.2 million Cancer Prevention Study–II participants enrolled by the American Cancer Society in 1982 and followed prospectively through 2000, Krewski et al. (2009) found even stronger risk of premature death for each increase of 10 $\mu g/m^3$ of $PM_{2.5}$ than had been reported in earlier studies of this cohort. His team found that the risk increased to 15 percent , up 3 percent over previous estimates. Taking a closer look at Los Angeles and New York City data, the study found evidence that local exposures may have a stronger impact than had previously be identified.[97]

In this study, the mean annual $PM_{2.5}$ associated with premature deaths from all causes was 14.0 $\mu g/m^3$, well below the current standard. The strongest associations were with cardiopulmonary disease, ischemic heart disease and lung cancer.

Further evidence that $PM_{2.5}$ was likely to cause premature death from lung cancer was found in a 2011 follow-up to the Extended ACS study in 26-year time period (1982–2008) that looked at lung cancer mortality in lifelong never-smokers. Turner et al. (2011) found that the risk of lung cancer identified in the Krewski et al. study held up at this mean long-term ambient fine particulate matter air pollution

[95] Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee letter to Stephen L. Johnson, Administrator, U.S. Environmental Protection Agency, March 21, 2006, Subject: Clean Air Scientific Advisory Committee Recommendations Concerning the Proposed National Ambient Air Quality Standards for Particulate Matter, EPA-CASAC-LTR-06-002.

[96] Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, Turner MC, Pope AC III, Thurston G, Calle EE, Thun MJ (2009). Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Health Effects Institute. Cambridge, MA. Report Nr. 140.

[97] Krewski et al., 2009.

concentration (14.0 µg/$m^3$) well below the current standard. Each 10 µg/$m^3$ increase in $PM_{2.5}$ concentrations was associated with a 15–27 percent increase in the relative risk of lung cancer death after detailed adjustment for a number of potential confounders including passive smoking, occupational exposures, and radon. [98]

### *Medicare Cohort*

Evidence showing that levels of $PM_{2.5}$ well below the current standard is associated with premature morality resulted from a study looking at Americans aged 65 or older. Eftim et al. (2008) used Medicare data for the period 2000-2002 for the same geographic locations included in two landmark cohort studies and recent extensions of them: the Harvard Six Cities Study (SCS) (Dockery, et al 1993; Laden et al, 2006) and the American Cancer Society Study (ACS) (Pope et al. 1995, 2002). The analysis considered individual data on only age and sex, and was not adjusted for other individual risk factors. As shown in the table below, risk estimates were comparable to the original studies, but the association was with a lower mean $PM_{2.5}$ of 13.6 and 14.1 µg/$m^3$, both well below the current standard.[99]

**TABLE 2.** Study Characteristics: Med-ACS, ACS,[3] Med-SCS, and SCS[5]

| Characteristics | Med-ACS | ACS | Med-SCS | SCS |
|---|---|---|---|---|
| No. counties | 110* | 50† | 6‡ | 6 |
| No. subjects§ | 7,333,040 | 295,223 | 341,099 | 8096 |
| No. deaths¶ | 1,122,311 | 62,000 | 54,160 | 2732 |
| $PM_{2.5}$ ($\mu g/m^3$); mean (SD) | 13.6 (2.8) | 17.7 (3.7) | 14.1 (3.1) | 16.4‖ (5.6)‖ |
| Range | 6.0–25.1 | 9–33.5 | 9.6–19.1 | 10.2–29.0‖ |
| Study period | 2000–2002 | 1982–1998 | 2000–2002 | 1974–1998 |
| Period of measured exposure | 2000–2002 | 1979–1983, 1999–2000 | 2000–2002 | 1979–1988, 1990–1998 |

*Counties identified by the Reanalysis team[16] as being within the 50 metropolitan statistical areas included in the ACS.[2]
†These are metropolitan statistical areas.
‡The 6 counties that include the 6 cities in the SCS.
§The number of subjects for the Med-ACS and Med-SCS datasets is the number of persons at risk in year 2000. For ACS and SCS, it is the number of persons enrolled at the beginning of the study period.
¶Total deaths occurred during the entire study period. For ACS,[3] the number of deaths is approximately triple the number of deaths in the original ACS.[2]
‖Calculated based on Table 1 and Figure 1 from Laden et al.[5]
SD indicates standard deviation.

### *Women's Health Initiative Cohort Study*

One of the first major studies to rely on direct measurements of fine particle concentrations found

[98] Turner MC, Krewski D, Pope CA, Chen Y, Gapstur SM, Thun MJ. Long-term ambient fine particulate matter air pollution and lung cancer in a large cohort of never-smokers. *Am J Respir Crit Care Med* 2011; 184: 1374-81.
[99] Eftim SE, Samet JM, Janes H, McDermott A, Dominici F. Fine Particulate Matter and Mortality: A Comparison of the Six Cities and American Cancer Society Cohorts with a Medicare Cohort. *Epidemiology* 2008; 19:209-216.

convincing evidence of harm well below the current standard and below EPA's proposed standard. The Women's Health Initiative (WHI) found that women who live incities and neighborhoods with higher levels of fine particulate matter experience higher rates of death and infirmity from heart disease and strokes than women who live in cleaner cities.[100] Increased exposure to $PM_{2.5}$ was associated with increased risk of stroke, heart problems, and death from heart disease, even when adjusted for other pollutants. In this study, Miller et al. found a 10-µg/m3 increase in $PM_{2.5}$ associated with a 76 percent increase in CVD death, as well as a 24 percent increase in a cardiovascular disease event, such as coronary revascularization, heart attack, and stroke, as well as death.[101]

This observational study of cardiovascular disease tracked 66,000 women in 36 U.S. cities. Miller et al. initially reported that these cities had a mean $PM_{2.5}$ concentration of 13.5 µg/$m^3$, well below the current annual standard. However, following reexamination of the air quality data, the authors and EPA scientists found that the corrected actual annual average mean concentration was 12.9 µg/$m^3$, lower than what was reported in the original paper – and below the 13 µg/$m^3$ standard proposed by EPA.[102]. Annual average $PM_{2.5}$ concentrations varied from 3.4 to 28.3 µg/$m^3$.

The figure below illustrates how the risk of death rose as the concentrations of the pollutant increased, relative to a reference value of 11 µg/$m^3$.

[100] Miller, K.A., Siscovick, D.S., Sheppard, L., Shepherd, K., Sullivan, J.H., Anderson, G.L., Kaufman, J.D. (2007). Long-term exposure to air pollution and incidence of cardiovascular events in women. *New Engl. J. Med*. 356(5):447-458.
[101] Miller et al. 2007.
[102] 77 FR 38918

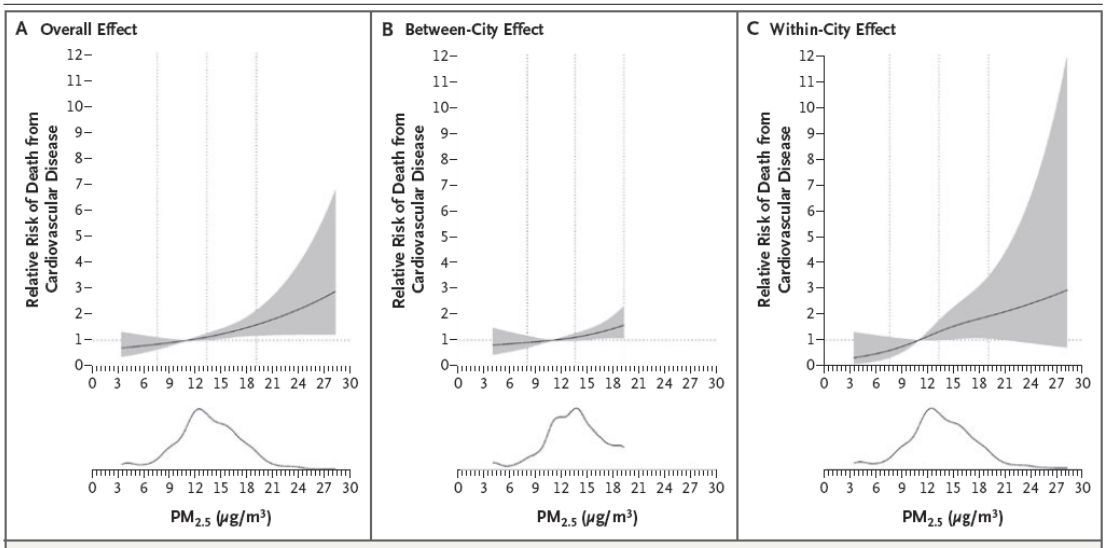



**Figure 1. Level of Exposure to Fine Particulate Matter and the Risk of Death from Cardiovascular Causes in Women.**

The graphs demonstrate the observed relationship between the risk of death from cardiovascular disease and the level of particulate matter of less than 2.5 μm in aerodynamic diameter ($PM_{2.5}$), including both definite and possible deaths from coronary heart disease or cerebrovascular disease. Panel A shows the overall relationship between the $PM_{2.5}$ level and death, Panel B the effects between metropolitan areas, and Panel C the effects within metropolitan areas, with an indicator variable used to adjust for each city. These results suggest a generally linear relationship between exposure and risk, though the 95% confidence intervals (shaded areas) are wide at the extremes of exposure. Risk is depicted in comparison with a reference value of 11 μg per cubic meter. The histogram in each panel illustrates the density of exposure distribution for air pollution. All estimates are adjusted for age, race or ethnic group, educational level, household income, smoking status, systolic blood pressure, body-mass index, and presence or absence of a history of diabetes, hypertension, or hypercholesterolemia.

Researchers concluded that:

> "Our study provides evidence of the association between long-term exposure to air pollution and the incidence of cardiovascular disease. Our study confirms previous reports and indicates that the magnitude of health effects may be larger than previously recognized. These results suggest that efforts to limit long-term exposure to fine particulate pollution are warranted."[103]

Writing in an accompanying editorial, Dr. Douglas W. Dockery of the Harvard School of Public Health and Dr. Peter H. Stone of the Harvard Medical School note that this study established a stronger statistical association between fine particulate air pollution and death from coronary heart disease than found in earlier studies. The WHI study reported a 76 percent increased risk of death from cardiovascular disease for every increase of 10 μg/$m^3$ in the mean $PM_{2.5}$ concentration, as compared to a 12 percent increase reported in the American Cancer Society cohort study. Referring to EPA's last review of the NAAQS for particulate matter, they note:

[103] Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, Kaufman JD. Long-term exposure to air pollution and incidence of cardiovascular events in women. *N Engl J Med* 2007; 356:447-458.

"Unfortunately for public health, the EPA failed to follow the recommendation of its science advisors and reduce the long-term standard for fine particles. The findings of the WHI study strongly support the recommendation for tighter standards for long-term fine particulate air pollution."[104]

### *Canadian Cohort Study*

Confirming evidence available in the current review is a new large study from Canada where $PM_{2.5}$ concentrations were substantially lower (mean, 8.7 μg/$m^3$) than in earlier U.S. studies. Crouse et al. (2012) found that mortality was positively and statistically significantly associated with long-term exposure to $PM_{2.5}$ in nonimmigrant adults aged 25 and older. [105] This cohort study supplies further evidence that not only the current standard, but EPA's proposed standards do not protect public health.

Study subjects consisted of 2.1 million adults who were included in the 1991–2001 Canadian census mortality follow-up study. The cohort included subjects from each province and from every major city, as well as those living in rural locations. For residents of cities with ground-based air quality monitors, the investigators calculated average mean annual concentrations of $PM_{2.5}$ for the period 1987–2001 and assigned exposure levels to individuals based on their residence during that time. They also calculated exposure estimates for the whole cohort for the period 2001–2006 based on satellite remote sensing observations.

The study found positive and statistically significant associations between nonaccidental mortality and estimates of $PM_{2.5}$ generated from both satellite-derived and ground-based observations in a large cohort of nonimmigrant Canadians.

In addition to adjusting for multiple individual, contextual, and spatial effects, a key strength of this study was the large sample size and large number of deaths needed to detect mortality associations at relatively low concentrations of $PM_{2.5}$.

The estimated mean concentration of $PM_{2.5}$ for subjects across Canada was 8.7 μg/$m^3$, which is substantially lower than the corresponding estimate for subjects across the United States as reported

[104] Dockery DW and Stone PH. Cardiovascular Risks from Fine Particulate Air Pollution. *N Engl J Med* 2007; 356:511-513.
[105] Crouse DL, Peters PA, van Donkelaar A, Goldberg MS, Villeneuve PJ, Brion O, Khan S, Atari DO, Jerrett M, Pope CA, Brauer M, Brook JR, Martin RV, Stieb D, Burnett RT. Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study. Environ Health Perspect 2012; 120 (5): 708-14.

in the ACS study (Krewski et al. 2009) and well below the current U.S. standard.

As reflected in the concentration–response curves presented in Figures 2A and 2B below, the study found near linear associations between $PM_{2.5}$ and mortality from nonaccidental and cardiovascular disease. Generally, these plots suggest that associations with mortality were present at concentrations of $PM_{2.5}$ of only a few micrograms per cubic meter.

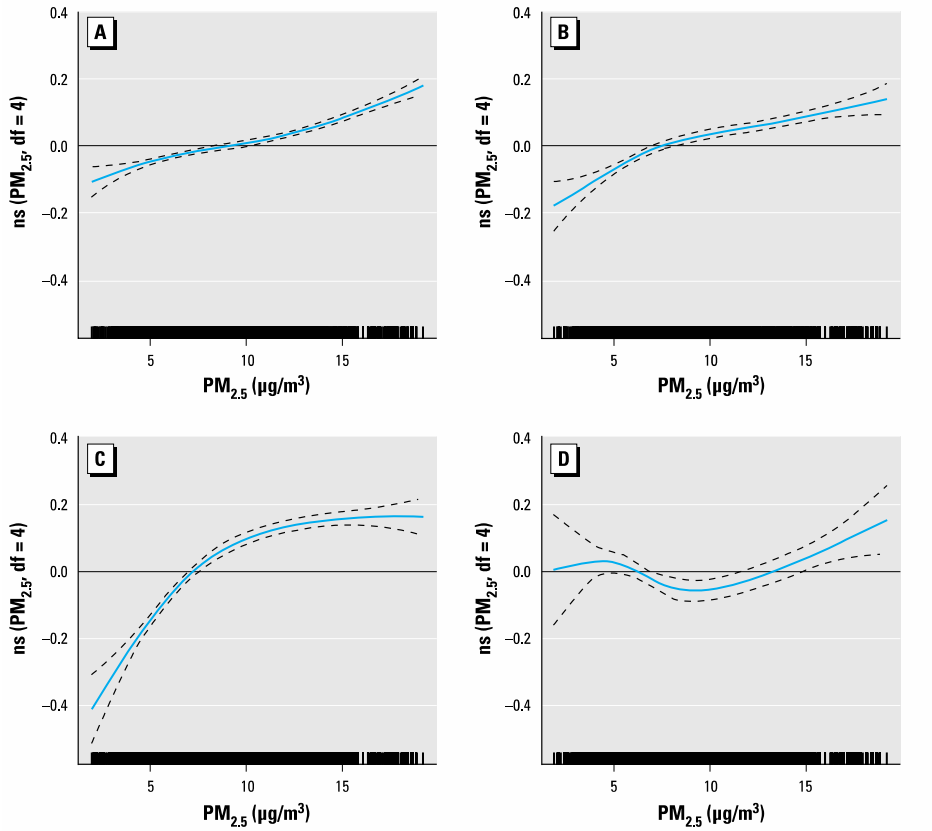



**Figure 2.** Concentration–response curves (solid lines) and 95% CIs (dashed lines) based on natural spline (ns) models with 4 df, standard Cox models stratified by age and sex, adjusted for all individual-level covariates, urban/rural indicator, and ecological covariates. (*A*) Nonaccidental causes. (*B*) Cardiovascular disease. (*C*) Ischemic heart disease. (*D*) Cerebrovascular disease. The tick marks on the *x*-axis identify the location of the $PM_{2.5}$ concentrations.

## Annual Average Concentrations in Short-term studies

In addition to the long-term studies of the effects of chronic exposures to $PM_{2.5}$ on mortality, evidence from the literature of acute effects of fine particulates is relevant to standard-setting and must be taken in account. Many of these studies analyzed data collected over a long period, and several provide strong evidence that annual standards need to be much stronger. The annual average $PM_{2.5}$ standard must be set below the annual average levels shown to be harmful in studies of acute effects.

**Harvard Six City Studies**

In further analysis of the Six City data set to investigate the relationship between $PM_{2.5}$ and daily deaths, Schwartz et al. (2002) used a variety of curve smoothing techniques which demonstrated concentration response relationships well below annual mean concentrations of 15 µg/$m^3$.[106]

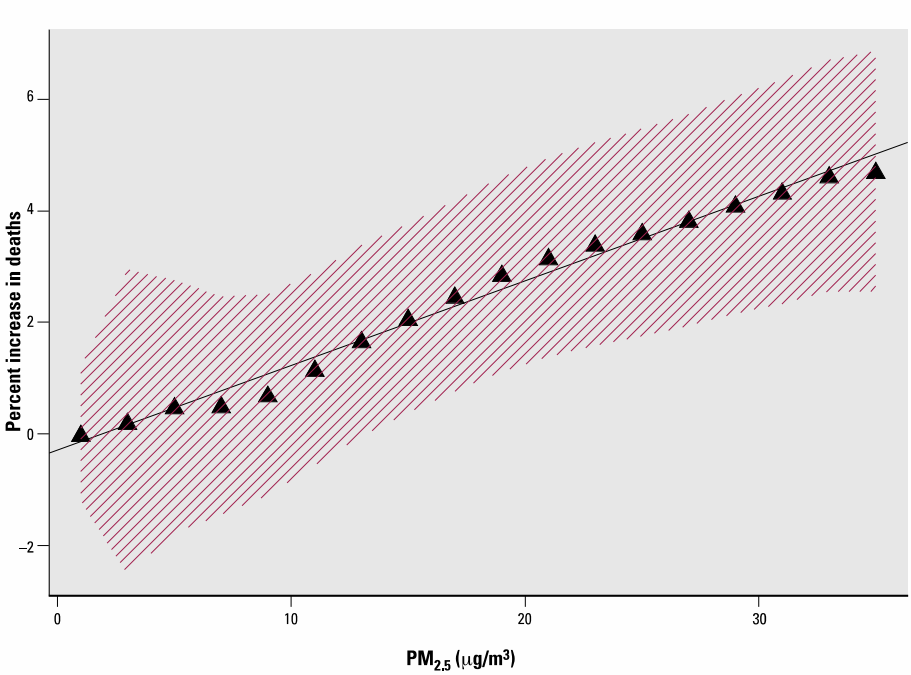



**Figure 1.** Overall estimated dose–response relation between total $PM_{2.5}$ and daily deaths in six U.S. cities. The estimate is obtained by combining the estimated smoothed curves in each of the cities, after controlling for weather, season, and day of the week. The shaded area indicates the pointwise 95% confidence intervals at each point. The line shown is a least-squares regression line through the estimated points.

[106] Schwartz J, Laden F, Zanobetti A. The Concentration-Response Relation Between $PM_{2.5}$ and Daily Deaths. *Environ Health Perspect.* 2002; 110:1025-1029.

### *112 City Study*

Zanobetti and Schwartz (2009) found evidence in a time-series study in 112 U.S. cities of the strong association between PM2.5 and PM10-2.5 and mortality at levels below 11 µg/$m^3$ in 30 cities.[107] The study found a strong association of both fine and coarse particles with daily deaths in cities. These associations are biologically plausible and, at the mean concentrations in the United States, suggest tens of thousands of early deaths per year, which could be avoided by reducing particle concentrations.

As the figure below indicates, the association between short-term exposure to $PM_{2.5}$ and total mortality and cardiovascular- and respiratory- related mortality is consistently positive for an overwhelming majority (99 percent) of the 112 cities across a wide range of air quality concentrations (long-term mean concentrations ranging from 6.6 mg/$m^3$ to 24.7 mg/$m^3$; ISA Figure 6–24, p. 6–178 to 179). Long-term mean concentrations were below 11 µg/$m^3$ for approximately 30 percent of the cities, lending support to the conclusion that an annual average $PM_{2.5}$ standard of 12 or 13 µg/$m^3$ will be inadequate to protect public health.

[107] Zanobetti A and Schwartz J. The Effect of Fine and Coarse Particulate Air Pollution on Mortality: A National Analysis. *Environ Health Perspec* 2009; 117:898-903.

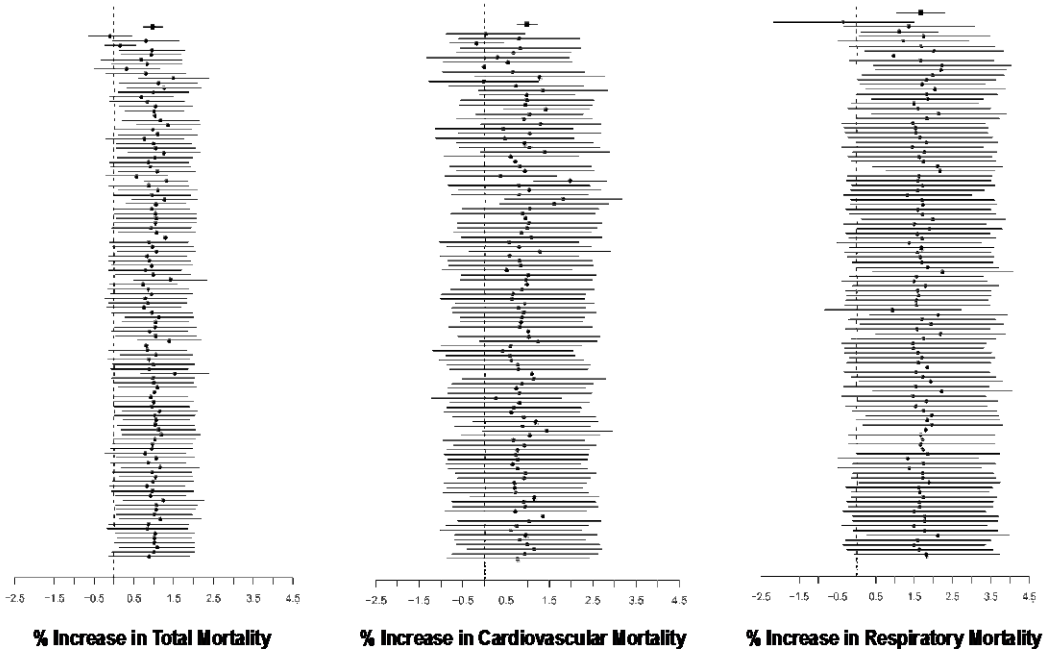



**Figure 6-24. Empirical Bayes-adjusted city-specific percent increase in total (nonaccidental), cardiovascular, and respiratory mortality per 10 μg/m$^3$ increase in the average of 0- and 1-day lagged $PM_{2.5}$ by decreasing mean 24-h avg $PM_{2.5}$ concentrations. Based on estimates calculated from Zanobetti and Schwartz (2009, 188462) using the approach specified in Le Tertre et al. (2005, 087560).**

**Key to Figure 6-24**

| City | Mean | 98th | City | Mean | 98th | City | Mean | 98th | City | Mean | 98th |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Rubidoux, CA | 24.7 | 68.0 | Taylors, SC | 15.0 | 32.2 | Waukesha, WI | 13.4 | 35.3 | Phoenix, AZ | 11.4 | 30.7 |
| Bakersfield, CA | 21.7 | 80.3 | Toledo, OH | 14.9 | 36.6 | Baton Rouge, LA | 13.4 | 30.1 | Tacoma, WA | 11.4 | 38.1 |
| Los Angeles, CA | 19.7 | 51.1 | Anaheim, CA | 14.9 | 44.1 | Memphis, TN | 13.3 | 32.4 | Port Arthur, TX | 11.1 | 25.7 |
| Fresno, CA | 18.7 | 64.9 | New York, NY | 14.7 | 38.1 | Erie, PA | 12.9 | 36.1 | Cedar Rapids, IA | 11.0 | 31.0 |
| Atlanta, GA | 17.6 | 38.2 | Washington, PA | 14.7 | 37.0 | Dallas, TX | 12.8 | 28.7 | Dodge, WI | 10.9 | 32.9 |
| Steubenville, OH | 17.1 | 41.4 | Winston, NC | 14.7 | 34.1 | Houston, TX | 12.8 | 27.5 | Oklahoma, OK | 10.8 | 26.1 |
| Cincinnati, OH | 17.1 | 39.9 | Elizabeth, NJ | 14.6 | 38.2 | Chesapeake, VA | 12.8 | 29.8 | Des Moines, IA | 10.5 | 27.9 |
| Birmingham, AL | 16.5 | 38.8 | Philadelphia, PA | 14.6 | 36.6 | Wilkes-Barre, PA | 12.8 | 32.5 | Jacksonville, FL | 10.5 | 25.3 |
| Middletown, OH | 16.5 | 38.4 | St. Louis, MO | 14.5 | 33.7 | Norfolk, VA | 12.7 | 29.6 | Omaha, NE | 10.5 | 28.0 |
| Indianapolis, IN | 16.4 | 38.2 | Allentown, PA | 14.4 | 38.9 | Sacramento, CA | 12.6 | 45.0 | Denver, CO | 10.5 | 26.4 |
| Cleveland, OH | 16.3 | 40.5 | Richmond, VA | 14.3 | 33.0 | Springfield, MA | 12.5 | 35.1 | Pinellas, FL | 10.4 | 23.1 |
| Dayton, OH | 16.3 | 38.3 | Spartanburg, SC | 14.2 | 31.4 | New Orleans, LA | 12.5 | 29.0 | Austin, TX | 10.4 | 24.5 |
| Columbus, OH | 16.2 | 38.3 | Durham, NC | 14.2 | 32.9 | Ft. Worth, TX | 12.4 | 27.7 | Orlando, FL | 10.3 | 24.3 |
| Detroit, MI | 16.2 | 41.0 | Little Rock, AR | 14.2 | 31.8 | Pensacola, FL | 12.3 | 31.2 | Klamath, OR | 10.2 | 40.7 |
| Akron, OH | 16.0 | 39.0 | Easton, PA | 14.2 | 39.7 | Davenport, IA | 12.3 | 32.1 | Seattle, WA | 10.1 | 27.9 |
| Louisville, KY | 15.9 | 38.0 | Raleigh, NC | 14.1 | 31.8 | Avondale, LA | 12.3 | 28.6 | Medford, OR | 10.0 | 37.3 |
| Chicago, IL | 15.8 | 39.1 | Greensboro, NC | 14.1 | 31.0 | Boston, MA | 12.3 | 30.2 | Bath, NY | 9.6 | 29.3 |
| Pittsburgh, PA | 15.7 | 43.1 | Mercer, PA | 14.1 | 36.4 | Holland, MI | 12.1 | 35.0 | Provo, UT | 9.5 | 38.5 |
| Harrisburg, PA | 15.6 | 40.2 | Annandale, VA | 14.0 | 34.6 | Charleston, SC | 12.1 | 27.9 | Miami, FL | 9.4 | 20.5 |
| Baltimore, MD | 15.6 | 38.8 | Nashville, TN | 13.9 | 31.0 | Tampa, FL | 12.1 | 25.8 | El Paso, TX | 9.0 | 24.4 |
| Youngstown, OH | 15.6 | 38.1 | Dumbarton, VA | 13.8 | 31.9 | Tulsa, OK | 12.1 | 32.3 | Spokane, WA | 8.9 | 30.6 |
| Knoxville, TN | 15.5 | 32.9 | Columbia, SC | 13.7 | 30.7 | Kansas, MO | 12.0 | 28.6 | San Antonio, TX | 8.9 | 21.9 |
| Gary, IN | 15.5 | 37.5 | Milwaukee, WI | 13.7 | 36.3 | Scranton, PA | 11.9 | 33.0 | Portland, OR | 8.9 | 25.4 |
| Charlotte, NC | 15.3 | 32.7 | New Haven, CT | 13.6 | 36.8 | Hartford, CT | 11.8 | 33.5 | Davie, FL | 8.4 | 19.1 |
| Warren, OH | 15.2 | 37.4 | Grand Rapids, MI | 13.6 | 36.4 | Minneapolis, MN | 11.6 | 31.6 | Eugene, OR | 8.1 | 29.9 |
| Washington, DC | 15.2 | 37.2 | El Cajon, CA | 13.5 | 34.9 | Worcester, MA | 11.5 | 30.2 | Palm Beach, FL | 7.8 | 18.4 |
| Wilmington, DE | 15.1 | 37.6 | Gettysburg, PA | 13.4 | 36.5 | Salt Lake, UT | 11.5 | 52.4 | Bend, OR | 7.7 | 23.5 |
| Carlisle, PA | 15.1 | 40.0 | State College, PA | 13.4 | 38.5 | Providence, RI | 11.5 | 30.5 | Albuquerque, NM | 6.6 | 17.9 |

Note: The top effect estimate in the figures represents the overall effect estimate for that mortality outcome across all cities. The remaining effect estimates are ordered by the highest (i.e., Rubidoux, CA) to lowest (i.e., Albuquerque, NM) mean 24-h $PM_{2.5}$ concentrations across the cities examined. In the key the cities are reported in this order, which represents the policy relevant concentrations for the annual standard, but the policy relevant $PM_{2.5}$ concentrations for the daily standard (i.e., 98th percentile of the 24-h average) are also listed for each city (from Zanobetti and Schwartz (2009, 188462))

### *Medicare Air Pollution Study (MCAPS)*

Another key study of older Americans' short-term exposures to PM provides added and substantial evidence that the 1997 annual standard fails to protect public health. Bell et al, 2008 [108] is discussed in more detail in the section focused on the 24-hour standard, but the EPA also appropriately included discussion of its finding s in the analysis of long-term exposures as well. Bell et al. examined data for 202 counties for Americans aged 65 and older who were in the Medicare database for 1999-2005. The researchers examined hospitalizations for cardiovascular and respiratory causes and found both associated with higher $PM_{2.5}$. As discussed below, EPA worked with the authors to calculate the mean $PM_{2.5}$ concentration at 12.9 $\mu g/m^3$.[109]

[108] Bell ML, Ebisu K, Peng RD, Walker J, Samet JM, Zeger SL, Dominici F. Seasonal and regional short-term effects of fine particles on hospital admissions in 202 US counties, 1999-2005. *Am J Epidemiol* 2008; 168: 1301-10.
[109] EPA, 2011.

## EPA's own analysis shows stronger standards are needed.

EPA's approach to interpreting epidemiological studies for standard setting has evolved in the years since the first fine particle standards were established in 1997. Initially, the focus was on consideration of the mean concentrations reported in key studies. In the 2006 review, EPA also looked at concentrations 1 standard deviation (SD) below the mean, in an effort to focus on where the preponderance of evidence lay. EPA also examined four studies in depth to see the concentrations down to the 25$^{th}$ and the 10$^{th}$ percentile to evaluate levels of the strongest certainty. By either analytic measure, the annual standard must be significantly lower than EPA has proposed.

### Harm shown one standard deviation below the mean

The air quality data reported in the epidemiological literature represents a statistical distribution of air quality concentrations. Typically, studies will report the mean annual average concentration, plus-or-minus one SD. This range of values encompasses approximately 68 percent of the data. It is relevant to standard setting because the reported effects do not just occur at the mean, but also above and below the mean. By using 1 SD below the mean and a benchmark for standard setting, EPA can assume it is protecting against the bulk of the health effects in the concentration range where those effects are concentrated. One advantage of this approach is that information about the standard deviation of interquartile range is readily available in published studies.

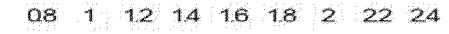
The FR Notice includes several figures, reprinted below, that provide information on the mean PM concentration level and 1 SD below the mean for studies judged to be most relevant to standard setting. The following figure, labeled Figure 1, provides summaries of effects estimates and air quality distributions for multi-city, long-term $PM_{2.5}$ exposure studies of the general population and older adults. The mean minus 1 SD in these studies ranges 9.8 to 11.3 μg/m$^3$, indicating that a standard of 12 μg/m$^3$ would be insufficient to protect against the majority of health effects reported in the long-term multi-city studies of the general population and older adults, and points to the need for a standard of in the range of 9-11 μg/m$^3$.

EPA identified a number of multi-city short-term studies of particular relevance to the review of the annual average $PM_{2.5}$ standard. Annual mean $PM_{2.5}$ concentrations ranged from 15.6 down to 12.6 μg/m$^3$. The studies in the Figure labeled 3 below , also excerpted from the FR notice, shows that the means minus 1 SD ranged from 10.5 to 3.9 μg/m$^3$, though this data point was not available for all the short-term studies of the general population and older adults in the table. This analysis of the annual

concentration data in the short-term studies supports the conclusion that the annual average standard must be set at 11 μg/$m^3$, the lowest level under consideration, or below.

**Figure 1. Summary of Effect Estimates (per 10 µg/m$^3$) and Air Quality Distributions for Multi-City, Long-term $PM_{2.5}$ Exposure Studies of the General Population and Older Adults**

| Study | Cite | Geographic Area | Years of Air Quality Data | Endpoint | Air Quality Data (µg/m³) Author Reported Data | | | Effect Estimate (95% CI) |
|---|---|---|---|---|---|---|---|---|
| | | | | | Mean | Mean - 1SD | Range | |
| *General Population* | | | | | | | | |
| WHI | Miller et al. (2007) | 36 US cities | 2000 | Mortality-CV | 12.9[a] | 10.2 | 3.4 – 28.3 | |
| | | | | All CVD | | | | |
| | | | | Incident MI | | | | |
| | | | | Revascularization | | | | |
| | | | | Stroke | | | | |
| | | | | CBVD | | | | |
| Cystic Fibrosis | Goss et al. (2004)[b] | 6 US regions (NE, SE, NC, SC, NW, SW) | 2000 | Mortality-All-cause | 13.7 | 9.5 | 11.8-15.9 (IQR) | |
| | | | | Pulmonary exacerbation | | | | |
| ACS-Reanalysis II | Krewski et al. (2009) | 116 US MSAs | 1999-2000 | Mortality-all cause | 14.0 | 11.0 | 5.8 - 22 | |
| | | | | Mortality-IHD | | | | |
| | | | | Mortality-CPD | | | | |
| | | | | Mortality-Lung cancer | | | | |
| VA | Lipfert et al. (2006) | | 1999-2001 | Mortality-all cause | 14.3 | 11.3 | 5.0 - ? | |
| Harvard Six Cities (SCS)-Extended | Laden et al. (2006) | 6 US cities (Northeast/ Midwest) | 1979-1998 | Mortality-all cause | 16.4[c] | 10.8 | 10-22 | |
| | | | | Mortality-CV | | | | |
| | | | | Mortality-Respiratory | | | | |
| | | | | Mortality-Lung cancer | | | | |
| *Older Adults* | | | | | | | | |
| MCAPS-Western US | Zeger et al. (2008) | 62 US counties | 2000-2005 | Mortality-all cause | 13.1[d] | - | 10.4-18.5 (IQR) | |
| Medicare-ACS | Eftim et al. (2008) | 51 US MSAs | 2000-2002 | Mortality-all cause | 13.6 | 10.8 | 6.0-25.1 | |
| MCAPS-Eastern US | Zeger et al. (2008) | 421 US counties | 2000-2005 | Mortality-all cause | 14.0[c] | - | 12.3-15.3 (IQR) | |
| Medicare -SCS | Eftim et al. (2008) | 6 US cities | 2000-2002 | Mortality-all cause | 14.1 | 11.0 | 9.6-19. 1 | |




[a]Update of Miller et al. (2007) $PM_{2.5}$ data included in Curl, 2009
[b]Cohort included persons with cystic fibrosis age 6 and older, mean age: 18.4 yrs
[c]Estimated from data provided by study author (Laden, 2009)
[d]Median (IQR: Interquartile range); overall US reported median (IQR) of 13.2 µg/m$^3$ (11.1-14.9)

Source: US EPA, 2011a, Figure 2-4

**Figure 3. Summary of Effect Estimates (per 10 μg/m$^3$) and Air Quality Distributions for Multi-City, Short-term $PM_{2.5}$ Exposure Studies of the General Population and Older Adults**

| Study/Cite | Geographic Area | Years of Air Quality Data | Endpoint | Air Quality Data (μg/m$^3$) | | | | Effect Estimate (95% CI) |
|---|---|---|---|---|---|---|---|---|
| | | | | Author Reported Data | | | | |
| | | | | Mean | Mean - 1SD | Range | 98th percentile | |
| *General Population* | | | | | | | | |
| Burnett et al. (2004) | 12 Canadian Cities | 1981-1999 | Nonaccidental mortality | 12.8 | - | - | 38.0 | |
| Zanobetti & Schwartz (2009) | 112 US counties | 1999-2005 | Nonaccidental mortality | 13.2[a] | 10.3[a] | 6.6-24.7 | 34.3 | |
| Burnett & Goldberg (2003) | 8 Canadian Cities | 1986-1996 | Nonaccidental mortality | 13.3 | 3.9[b] | - | 38.9[a] | |
| Harvard Six Cities/ Klemm and Mason (2003) | 6 US cities (Northeast/ Midwest) | 1979-1988 | Nonaccidental mortality | 14.7[c] | - | 9-23 (IQR) | - | |
| Franklin et al. (2008) | 25 US communities | 2000-2005 | Nonaccidental mortality | 14.8[a] | - | 9.9-27.4[a] | 43.0 | |
| Franklin et al. (2007) | 27 US communities | 1997-2002 | Nonaccidental mortality | 15.6[a] | - | 8.8-23.9 | 45.8 | |
| *Older Adults/Children*[d] | | | | | | | | |
| MCAPS/Bell et al. (2008) | 202 US counties | 1999-2005 | CVD HA | 12.9[a] | 10.2[a] | 4-20 | 34.2 | |
| | | | Resp HA | | | | | |
| MCAPS/Dominici et al. 2006 | 204 US counties | 1999-2002 | IHD HA | 13.4[a] | 10.5[b] | 4-23 | 34.8 | |
| | | | CHF HA | | | | | |
| | | | Dysrhythmia HA | | | | | |
| | | | CBVD HA | | | | | |
| | | | PVD HA | | | | | |
| | | | COPD HA | | | | | |
| | | | RTI HA | | | | | |
| O'Connor (2008) | 7 US Cities | 1998-2001 | Wheeze/Cough | 14.0 | - | - | 39.0[a] | |




[a]Estimated from data provided by study author or published study
[b]Estimated from coefficient of variation reported in original study by Burnett et al. (2000)
[c]Mean value not reported in study, median presented from original study by Schwartz et al. (1996)
[d]MCAPS cohort included adults ≥ 65 yrs; O'Connor (2008) cohort included children, mean age: 7.7 yrs
IQR: interquartile range

Source: US EPA, 2011a, Figure 2-6

A second summary table, labeled Figure 2-5 and developed by EPA (PA at p. 247), looks at air quality

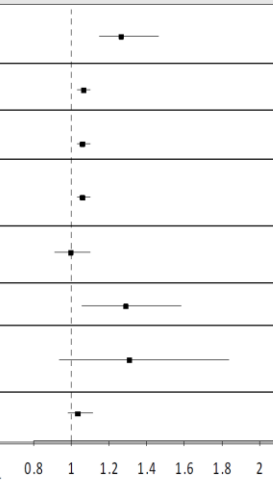
distributions for the multi-city, long-term $PM_{2.5}$ studies of children. The annual average means in these studies range from 11.9 to 14.9 µg/$m^3$, indicating the need for a standard of 11 µg/$m^3$ based on mean concentrations alone. However, when considering concentrations 1 SD below the mean, the studies for which this data is reported range from 6.1 to 10.3 µg/$m^3$, against suggesting that a standard of 10 µg/$m^3$ or below may be necessary to provide protection against effects such as low birth weight and bronchitis in children.

**Figure 2-5. Summary of Effect Estimates (per 10 µg/$m^3$) and Air Quality Distributions for Multi-City, Long-term $PM_{2.5}$ Exposure Studies of Children**

| Children | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Study | Cite | Geographic Area | Years of Air Quality Data | Endpoint | Air Quality Data (µg/m³) Mean | Mean – 1SD | Range | Effect Estimate (95% CI) |
| | Bell et al. (2007) | CT,MA | 1998-2002 | Low Birth Weight | 11.9[a] | 10.3 | | |
| | Liu et al. (2007) | 3 Canadian cities | 1985-1999 | IUGR -1st trimester | 12.2 | - | 6.3-15 | |
| | | | | IUGR -2nd trimester | | | | |
| | | | | IUGR – 3rd trimester | | | | |
| | Parker and Woodruff (2008) | Continental US | 2000-2003 | Low Birth Weight | 13.5[a] | - | 10.9-16.1 (IQR) | |
| S CA CHS | McConnell et al. (2003) | 12 communities – S CA | 1996-1999 | Bronchitic Symptoms | 13.8 | 6.1 | 6-29 | |
| 24-Cities | Dockery et al. (1996) | 24 communities – US, Canada | 1988-1991 | Bronchitis | 14.5 | 10.3 | 5.8-20.7 | |
| | Woodruff et al. (2008) | 96 US counties | 1999-2002 | Infant mortality | 14.9[b] | -- | 12.0-18.6 (IQR) | |

[a]Gestational mean

[b]Median for all cause mortality; median (IQR: interquartile range) for survivors = 14.8 (11.7-18.7) µg/m³. Exposure period was first 2 months of life.




## Harm shown at EPA-accepted confidence levels

In the current review, the CASAC panel urged EPA to go beyond a simple examination of the mean minus 1 SD approach to the evaluation of the epidemiological studies. CASAC recommended that EPA focus on information related to the concentrations that were most influential in generating the health effect estimates in individual studies to inform alternative annual standard levels (Samet, 2010a, p. 2).

> "In commenting on the *Second External Review Draft* of the *Quantitative Health Risk Assessment,* CASAC recommended that EPA develop criteria for setting the lower bound for the scenarios considered in the risk analysis. That same issue emerges with the *Policy Assessment* as lower bounds are considered for the range for the annual and 24-hr $PM_{2.5}$ standards. The approach could be statistically based, using, for example, interquartile range, standard deviation, 10$^{th}$ percentile level, or lower-bound confidence interval, but also based on broader

consideration of uncertainty and of the level of health protection to be achieved. In fact, selection of the lower bound of the inter-quartile range may not be sufficiently health protective. (emphasis added) Specifying the criterion by which a lower bound concentration is chosen becomes central in decision-making under a no-threshold model, since any suite of NAAQS above policy-relevant background leaves residual morbidity and premature mortality."[110]

In other words, CASAC indicated that the approach conveyed in the tables described above may not be sufficiently health protective.

CASAC went on to make a critically important recommendation regarding the interpretation of the epidemiological studies for standard setting, stating:

> "Further consideration should be given to using the 10th percentile as a level for assessing various scenarios of levels for the PM NAAQS."(emphasis added)[111]

To address the concerns raised by CASAC, EPA undertook a more detailed analysis of the distributions in the key epidemiological studies for both population data such as health endpoints to better identify the concentration ranges in which the majority of the adverse health effects occurred.

> "Consistent with the Panel's comments to consider more information from epidemiological studies related to the concentrations that were most influential in generating the health effect estimates in individual studies to inform staff conclusions on alternative annual standard levels that would provide appropriate protection for both long- and short-term exposures, we contacted several study investigators to obtain additional information on population-level data (i.e., health events, number of study participants). In new analyses using distributional statistics, we considered these data in conjunction with air quality data to identify the broader range of $PM_{2.5}$ concentrations that were most influential in generating health effect estimates in epidemiological studies, and, specifically, the range of $PM_{2.5}$ concentrations below the long-term means over which we continue to have confidence in the associations observed in these epidemiological studies (Figures 2-7 and 2-8 and associated text)."[112]

[110] CASAC Letter May 17, 2010
[111] CASAC letter, May 17, 2010.
[112] Memorandum from Lydia N. Wegman, Director, Health and Environmental Impacts Division, Office of Air Quality Planning and Standards to Holly Stallworth, Designated Federal Officer, Clean Air Scientific Advisory Committee, EPA Science Advisory Board Staff Office. Subject: Transmittal of *Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards* – Final Document, April 20, 2011. Attachment -- CASAC Comments on Second Draft PM Policy Assessment and Responses to those Comments.

The results of this distributional analysis are described in a memo to the docket[113] and in the Policy Analysis.[114] EPA's analysis tracks lower bounds of the confidence levels that were in the four studies where EPA secured population-level data from the ACS Reanalysis II and the Women's Health Initiative long-term $PM_{2.5}$ exposure studies and from the Medicare Air Pollution Study (MCAPS) and the 112-Cities Mortality short-term $PM_{2.5}$ exposure studies.

Figure "2" below shows the distribution of population-level (health event, or study population as a surrogate for health event) data relative to annual mean $PM_{2.5}$ concentrations for key multi-city studies of short-term effects. The graph indicates that a range of the $10^{th}$ to $25^{th}$ percentile of the population data -- corresponds to annual average $PM_{2.5}$ concentrations in the range of approximately 9.5 to 12.5 $\mu g/m^3$.

[113] Memorandum to PM NAAQS Review Docket EPA-HQ-OAR-2007-0492. From: Pradeep Rajan, OAR/OAQPS/HEID, Mark Schmidt, OAR/OAQPS/AQAD, Beth Hassett-Sipple, OAR/OAQPS/HEID. Subject: $PM_{2.5}$ Distributional Statistical Analyses. April 7, 2011..

[114] EPA Policy Analysis, 2011.

**Figure 2. Distribution of Population-Level Data and Corresponding $PM_{2.5}$ Concentrations for Selected Short-term $PM_{2.5}$ Epidemiological Studies**

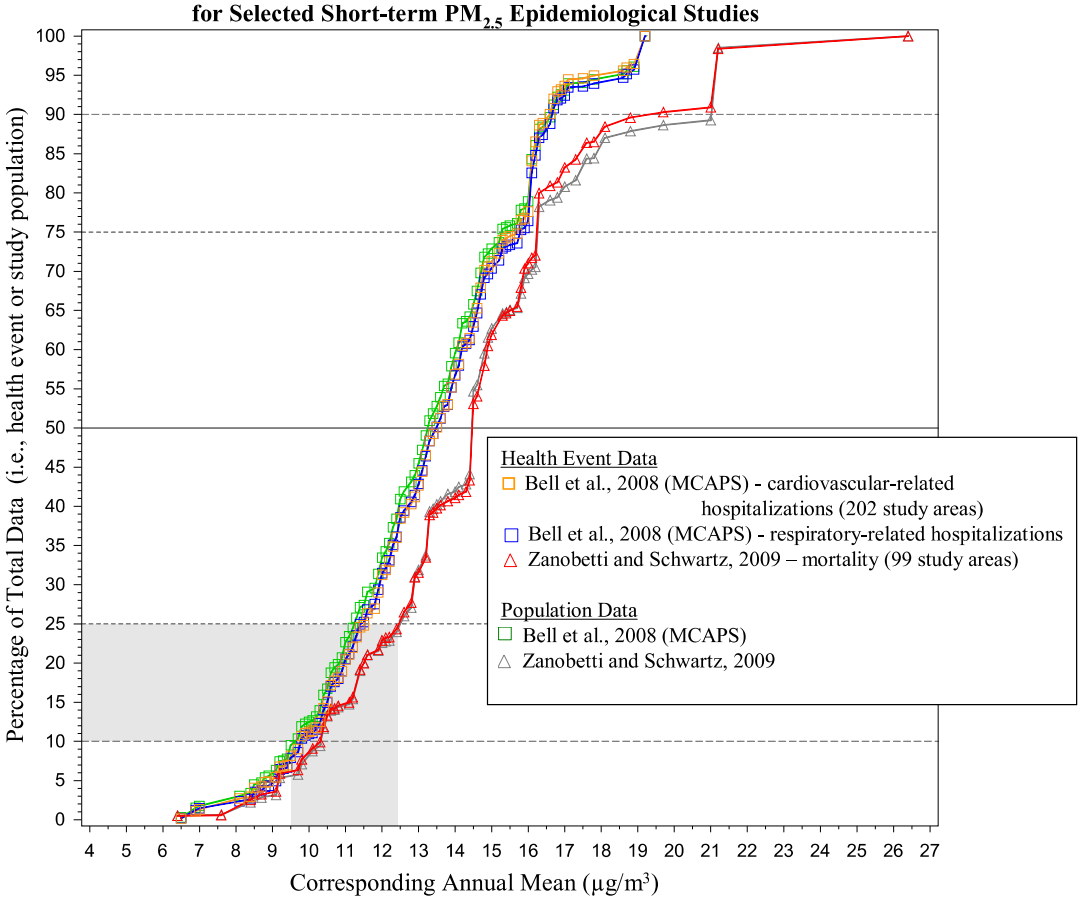
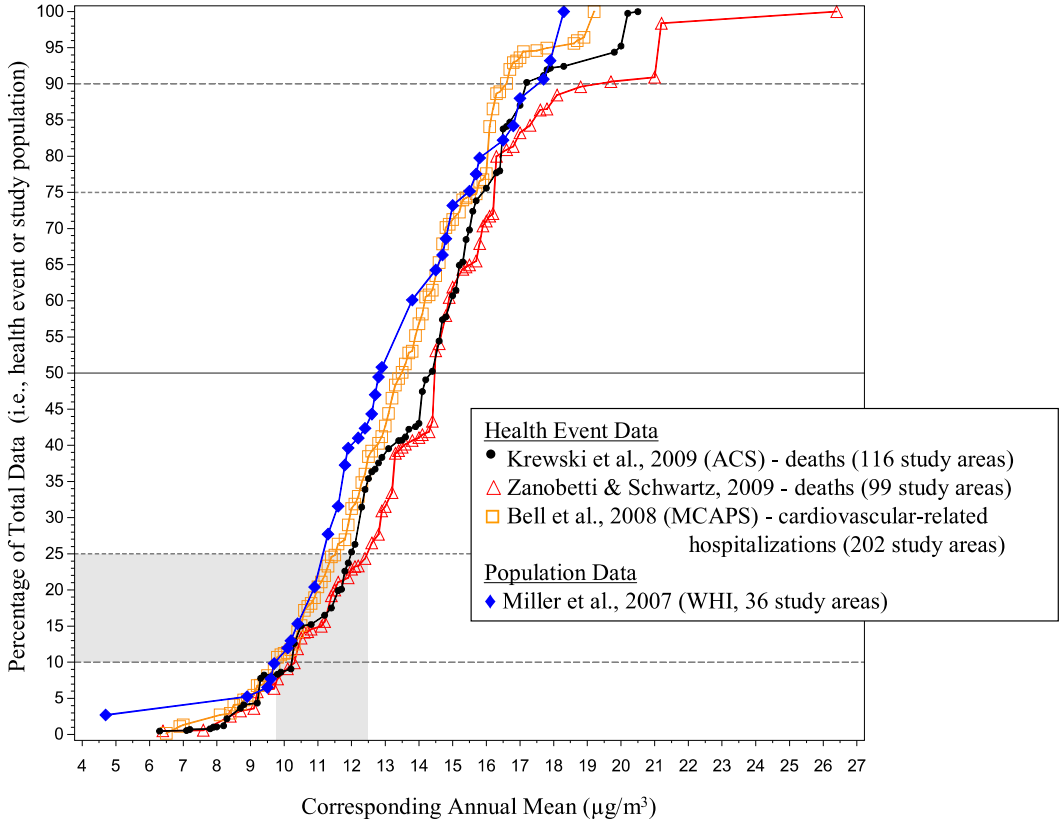


**Figure 3. Distribution of Population-Level Data and Corresponding $PM_{2.5}$ Concentrations for Selected Multi-City Epidemiological Studies**




A comparable graph (labeled Figure 3 above), demonstrates the relationship between health outcomes in the key, multi-city, long-term studies relative to annual mean $PM_{2.5}$ concentrations. The graph indicates that a range of the 10th to 25th percentiles of the population data -- corresponds to annual average $PM_{2.5}$ concentrations in the range of approximately 9.5 to 12.5 µg/$m^3$.

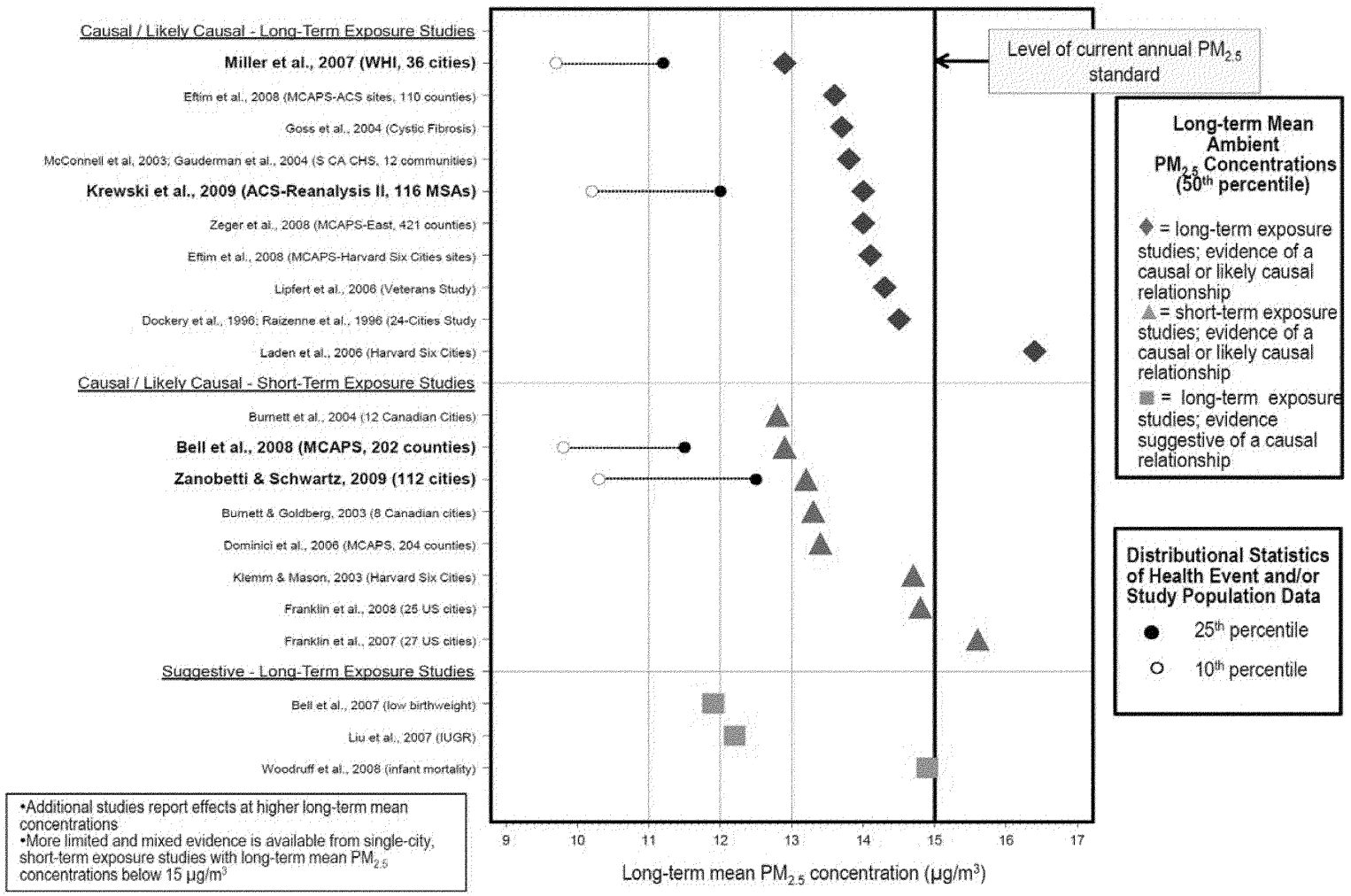
Finally, the summary figure 4 below places all of these studies and the confidence intervals into one chart, which shows clearly the translation of the epidemiological evidence from these four studies as well as other major long-term and short-term exposure studies into graphic form.

Figure 4 below, from 77 FR 38929 and also from the Policy Analysis (p. x), shows that if the strongest confidence intervals as EPA has determined lay above the 25 percentile, the large Women's Health Initiative and the Medicare Study both had data showing harm with strong certainty well below 12. Even the study that EPA's scientists found the most robust—the extended Cancer Society cohort—had its 25th percentile at 12 µg/$m^3$and had 11 µg/$m^3$ as one standard deviation below the mean values. If

EPA seeks to follow CASAC's comments and consider that they have confidence down to the 10th percentile of data, then even a standard of 11 μg/m3would fail to provide adequate protection. Given the evidence that no threshold of harm exists, we believe this calls for an annual standard of no higher than 11 μg/m3.

In each of these studies in Figure 4 the long-term mean $PM_{2.5}$ concentrations in the multi-city U.S. studies selected by EPA (including both short-and long-term exposure studies) range from below 12 ug/m3 to above 15 μg/m$^3$. Looking just at mean concentrations, the figure clearly demonstrates the inadequacy of the current annual average NAAQS of 15 μg/m$^3$. In addition, the figure demonstrates that an annual average standard of 13 μg/m$^3$, at the upper end of the range proposed by EPA, is also inadequate. The mean concentrations in a number of key studies including Miller et al 2007, Burnett et al 2004, and Bell et al 2008, and Liu et al 2007 have mean $PM_{2.5}$ concentrations below 13 μg/m$^3$, and Bell et al 2007 has mean concentrations below 12 μg/m$^3$, suggesting that based on mean concentrations alone a standard of 11 is justified.

**Figure 4. Translating Epidemiological Evidence from Multi-City Exposure Studies into an Annual $PM_{2.5}$ Standard**




Source: US EPA, 2011a, Figure 2-8

Thus, examination of both the long and short-term studies clearly indicates that an annual average standard of 13 µg/$m^3$ would fall above the 25$^{th}$ percentile of the health event data and would not be protective of public health.

The analysis indicates that even a standard of 11 µg/$m^3$, the lowest concentration for which EPA is seeking public comment, is above the 10$^{th}$ percentile benchmark recommended by CASAC.

In conclusion, distributional data from key multi-city long- and short-term studies of children and adults, including mean annual average concentrations, concentrations one standard deviation below the mean, and EPA's 10$^{th}$ and 25$^{th}$ percentile distributional analysis of concentrations where health impacts are concentrated all show the need for a standard of 11 µg/$m^3$ or below.

## Retaining the 24-hour $PM_{2.5}$ Standard at 35 µg/$m^3$ Will Not Protect Public Health

Hundreds of studies from around the world have demonstrated that short-term exposure to fine particle pollution causes mortality from cardiopulmonary diseases, hospitalization and emergency room visits for cardiopulmonary diseases, increased respiratory symptoms, decreased lung function, and cardiac effects. That is, as air pollution rises, it is followed by an increase in adverse effects within a few hours, the next day, or over several days. Multi-city studies from Europe and the U.S. have documented increased morbidity and mortality from daily exposures at levels below the current standards. An annual standard alone is not sufficient to protect against the effects of short-term exposures, nor from the effects of more acute, sub-daily exposures. This is particularly true in areas that experience high daily concentrations relative to the annual average due to seasonal sources or atmospheric conditions. According to the CASAC panel:

> A substantial body of empirical evidence is summarized indicating that significant health effects are reported at or just below the current 24-hour and annual standards, justifying the conclusion in the *Policy Assessment* that the observed effects are important from a public health perspective.[115]

EPA proposes to retain the current 24-hour $PM_{2.5}$ standard of 35 µg/$m^3$. This proposal disregards important scientific findings and will not make a substantial dent in the epidemic of air quality-related illness and death associated with high short-term exposures.

[115] CASAC letter, May 17, 2010.

The Policy Assessment recommended consideration of a standard of 30 μg/$m^3$ in conjunction with an annual average standard of 13μg/$m^3$. CASAC concurred that a reduction in the 24-hour standard was needed, but cautioned that an option of an 11 μg/$m^3$standard coupled with a 24-hour standard 30 μg/$m^3$ would not offer optimal protection to sensitive populations:

> Reducing the annual standard from 15 to 13 μg/$m^3$ is predicted to provide a significant public health benefit. Reducing the 24-hour standard from 35 to 30 μg/$m^3$ is also predicted to provide significant public health benefit. The 30/11 option would provide the greatest protection to the largest number of people in the U.S., yet **even this option will probably not offer optimal protection the most at risk populations** (emphasis added), e.g. those with greater susceptibility to the effects of PM.[116]

When OMB asked EPA to revise its proposal for the annual $PM_{2.5}$ standard from 12 to a range from 12 to 13, no change was made to the proposal to adjust the daily standard to 30 μg/$m^3$, as recommended by EPA staff scientists in the Policy Assessment, with the concurrence of CASAC, to complement an annual standard of 13 μg/$m^3$.

EPA's proposal for the 24-hour standard, therefore, is not based on what the scientific record demonstrates is needed.

As EPA pointed out in a briefing for OMB: "An annual standard of 13 μg/$m^3$ (absent a very low 24-hour standard level) could raise issues similar to those which resulted in the remand of the 2006 $PM_{2.5}$ annual standard."[117]

Commenters assert that the record clearly supports a more stringent 24-hour standard of 25 μg/$m^3$to provide uniform protection in all regions of the country, particularly from short-term spikes in pollution and from the sub-daily exposures that trigger heart attacks and strokes. Such a standard would be consistent with the recommendations of the World Health Organization working group which favored a 24-hour $PM_{2.5}$ standard of 25 μg/$m^3$.[118] [119]

[116] CASAC letter, September 10, 2010.
[117] U.S. EPA Briefing for Interagency Review, Draft Proposal for the Particulate Matter (PM) National Ambient Air Quality Standards (NAAQS), June 4, 2012. Docket: EPA-HQ-OAR-0492-0451.
[118] World Health Organization, WHO Air Quality Guidelines Global Update 2005, Report on a working group meeting, Bonn, Germany, 18-20 October 2005. WHOLIS number E87950.
[119] A tightened 24-hour standard would also be consistent with the standards adopted by Canada for $PM_{2.5}$ in 2000, and reaffirmed these standards in a 2005 review. The 24-hour $PM_{2.5}$ Canadawide standard of 30 μg/$m^3$ is based on a 98$^{th}$ percentile concentration, averaged over 3 years,[119] a stricter standard than that proposed by U.S. EPA.

## Multi-city Studies Have Demonstrated Associations Between Short-term $PM_{2.5}$ Concentrations Below 35 $\mu g/m^3$ and Significant Mortality and Morbidity

As the Policy Assessment points out (p. 106), multi-city epidemiological studies consider $PM_{2.5}$ concentrations and potential health impacts across a range of diverse locations providing spatial coverage for different regions across the country, reflecting differences in $PM_{2.5}$ sources, composition, and potentially other exposure-related factors which might impact $PM_{2.5}$ -related risks. These studies encompass larger study populations that afford the possibility of generalizing to the broader national population and provide higher statistical power than single-city studies to detect potentially statistically significant associations with relatively more precise effect estimates.

A very large study using hospital admission rates from the Medicare database in 204 urban counties in the U.S., found significant morbidity at daily levels below 35 $\mu g/m^3$.[120] The annual average $PM_{2.5}$ levels within these counties were generally at or below the current standard -- the average of the county means was 13.4 $\mu g/m^3$, and the interquartile range was 11.3-15.2 $\mu g/m^3$. The researchers found significant increases in hospital admissions for cerebrovascular disease, peripheral vascular disease, ischemic heart disease, cardiac arrhythmias, heart failure, chronic obstructive pulmonary disease (COPD), and respiratory tract infection with each 10 $\mu g/m^3$ daily increase in $PM_{2.5}$. The principal investigator conducted additional analysis to restrict the data set to days with daily concentrations below 35 $\mu g/m^3$:

> "To provide more targeted evidence toward the adequacy of the proposed 24-hr $PM_{2.5}$ NAAQS standards as to whether they protect public health with an adequate margin of safety, we have conducted an additional analysis which was not included in the Journal of American Medical Association report. Specifically, we have re-estimated national average relative rates of hospitalization with the exclusion from the data set of days with 24-hour average levels of $PM_{2.5}$ exceeding 35$\mu g/m^3$ (subset analysis). Table 1 below shows the results using the entire data set (same as Table 1 of Dominici et al. 2006) and the results from the subset analysis. In spite of the diminished statistical power due to the restriction of the analysis to a smaller number of days, we still find statistically significant associations between short-term exposure to $PM_{2.5}$ and hospital admissions for cerebrovascular disease, heart rhythm, heart failure, and respiratory infections."[121]

[120] Dominici F, Peng RD, et al. Fine particulate matter air pollution and hospital admission for cardiovascular and respiratory diseases. J*AMA* 2006; 295: 1127 -1134.
[121] Letter from Francesca Dominici to U.S. EPA, March 23, 2006. Docket ID No. EPA-HQ-OAR-2001-0017-0988.

**Table 1.** Percent change in hospitalization rate per $10\mu g/m^3$ increase in $PM_{2.5}$ on average across 204 counties*

| Cause-specific admission | Lag (days) | National Average Relative rate | National Average Relative rate (Subset analysis) |
|---|---|---|---|
| Injury | 0 | -0.41 (-1.00,0.18) | -0.52 (-1.27,0.24) |
| Cerebro-vascular disease | 0 | 0.81 (0.30,1.32) | 0.84 ( 0.12,1.56) |
| Peripheral vascular disease | 0 | 0.86 (-0.06,1.79) | 0.39 (-0.81,1.59) |
| Ischemic heart disease | 2 | 0.44 (0.02,0.86) | 0.30 (-0.19,0.80) |
| Heart rhythm | 0 | 0.57 (-0.01,1.15) | 0.84 (0.10,1.57) |
| Heart failure | 0 | 1.28 (0.78,1.78) | 1.12 ( 0.51,1.74) |
| COPD | 0 | 0.91 (0.18,1.64) | 0.58 (-0.30,1.47) |
| Respiratory infection | 2 | 0.92 (0.41,1.43) | 0.68 (0.03,1.32) |

This analysis shows increased hospital admissions in the elderly at daily concentrations below 35 µg/$m^3$ and is clearly indicative of the failure of EPA's proposed 24-hour standard to protect public health.

The Policy Assessment points to a multi-city time-series analysis of 112 U.S. cities, as further evidence that short-term concentrations of PM2.5 persist even in areas that would meet the current air quality standards. According to the Policy Assessment:

> Zanobetti and Schwartz (2009) reported a positive and statistically significant association with all-cause, cardiovascular-related (e.g., MI, stroke), and respiratory-related mortality and short-term $PM_{2.5}$ exposure, in which the aggregate long-term mean $PM_{2.5}$ concentration was 13.2 µg/$m^3$ (US EPA, 2009a, Figure 6-24). Furthermore, city-specific effect estimates in Zanobetti and Schwartz (2009) indicate the association between short-term exposure to $PM_{2.5}$ and total mortality and cardiovascular- and respiratory-related mortality is consistently positive for an overwhelming majority (99%) of the 112 cities across a wide range of air quality concentrations (ranging from 6.6 µg/$m^3$24.7 µg/$m^3$; US EPA, 2009a, Figure 6-24, p. 6-178 to 179). We note that for all-cause mortality, city-specific effect estimates were statistically significant for 55% of the 112 cities, with long-term city-mean $PM_{2.5}$ concentrations ranging from 7.8 µg/$m^3$ to 18.7 µg/$m^3$and 24-hour $PM_{2.5}$ city-mean 98$^{th}$ percentile concentrations ranging from 18.4 to 64.9 µg/$m^3$ (personal communication with Dr. Antonella Zanobetti, 2009).[26]

Bell, et al 2008 investigated whether short-term effects of $PM_{2.5}$ on risk of cardiovascular and respiratory hospitalizations among the elderly varied by region and season in 202 US counties for 1999-

2005.[122] The authors found that respiratory disease effect estimates were highest in winter, with a 1.05 percent (95% posterior interval: 0.29, 1.82) increase in hospitalizations per 10 μg/$m^3$increase in same-day $PM_{2.5}$. Cardiovascular diseases estimates were also highest in winter, with a 1.49 percent (95% confidence interval: 1.09, 1.89) increase in hospitalizations per 10 μg/$m^3$increase in same-day $PM_{2.5}$ with associations also observed in other seasons. The strongest evidence of a relation between $PM_{2.5}$ and hospitalizations was in the Northeast for both respiratory and cardiovascular diseases. This study reports positive and statistically significant effects with an overall 98$^{th}$ percentile value below the level of the current 24-hour standard, in conjunction with an overall long term mean concentration slightly less than 13 μg/$m^3$.

Taken together, evidence from the multi-city epidemiological studies demonstrate the need to strengthen the 24-hour standard in conjunction with a lower annual average standard.

## Single City Studies Show Excess Mortality and Morbidity at Levels Below 35 μg/$m^3$

Single-city studies are more limited in terms of statistical power and geographic coverage than multi-city studies, but the relationship between $PM_{2.5}$ concentrations and health effects can be more straightforward to establish. Single city studies also provide valuable information regarding impacts on susceptible populations and on health risks in areas with high peak to mean concentration ratios.

A large case-crossover study in the greater Boston area reported a significant increase in myocardial infarction associated with short-term exposures to $PM_{2.5}$.[123] The researchers divided the study group into quintiles of exposure, and found statistically significant increased odds of myocardial infarction in the fourth quintile OR=1.31 (CI = 1.01, 1.69). The 24-hour $PM_{2.5}$ concentrations in the fourth quintile ranged from 11.6 - 16.2 μg/$m^3$, well below the proposed 24-hour standard. In fact, the 95th percentile in this study was only 24.3 μg/$m^3$, reinforcing the need to seriously lower the 24-hour standard in order to protect against heart attacks.

A Vancouver study focusing on hospitalization for COPD also found effects at 24-hour concentrations below 35 μg/$m^3$.[124] In this study, the 100th percentile 24-hour $PM_{2.5}$ concentration was 32 μg/$m^3$. There was a statistically significant increase in hospitalizations for COPD within the pollution range of

[122] Bell ML, Ebisu K, Peng RD, Walker J, Samet JM, Zeger SL, Dominici F. Seasonal and regional short-term effects of fine particles on hospital admissions in 202 US counties, 1999-2005. *Am J Epidemiol* 2008; 168: 1301-10.
[123] Peters A, Dockery DW, et al. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation* 2001; 103: 2810-2815.
[124] Chen Y, Yang Q, et al. Influence of relatively low level of particulate air pollution on hospitalization for COPD in elderly people. *Inhal Toxicol* 2004; 16: 21-25.

this study. Although the effect was not completely independent of $NO_2$ concentrations, the results should not be dismissed, because they occurred at conditions that are prevalent in many U.S. cities.

A study of emergency room visits in Atlanta also reported significant associations between short-term $PM_{2.5}$ concentrations and visits for pneumonia among adults of all ages.[125] In this study, the 90th percentile 24-hour concentration of $PM_{2.5}$ was 32.3 $\mu g/m^3$.

In a newer follow up to the SOPHIA study, Strickland et al. (2010) found that data on over 10 million Atlanta emergency department visits gave it strong statistical power to identify impacts of $PM_{2.5}$ and its components on pediatric asthma. They found that even at "relatively low levels" a strong association with emergency department visits for asthma with $PM_{2.5}$. $PM_{10}$ and $PM_{10-2.5}$. The mean 24-hour $PM_{2.5}$ was 16.4 $\mu g/m^3$with a 7.4 standard deviation. That low level of $PM_{2.5}$ reinforces "the need for the continued evaluation" of the NAAQS to "ensure that the standards are sufficient to protect susceptible individuals."[126]

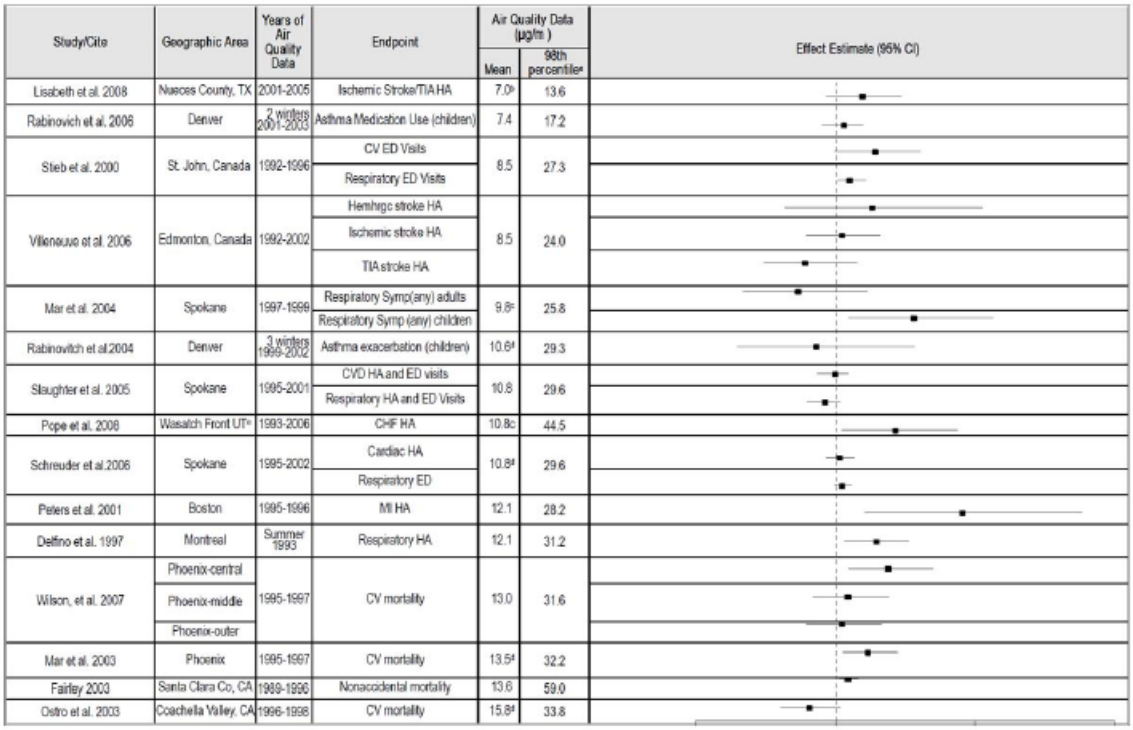
Statistically significant associations with mortality in areas that met the current annual and 24- hour $PM_{2.5}$ standards have long been reported.[127,128,129] In particular, the 98th percentile values for the 24-hour concentrations in these studies range down to 32 $\mu g/m^{3,}$ meaning that a standard set above this level is clearly within a range that is demonstrated to be associated with excess mortality. Therefore, the standard must be set well below 32 $\mu g/m^3$, 98th percentile, in order to provide a margin of safety to protect against excess mortality.

The figure below, from EPA's Policy Assessment (p. 2-87) indicates that studies have reported positive effect estimates for illness and death even in cities that meet an annual average $PM_{2.5}$ standard of 13 $\mu g/m^3$, and a daily standard of 35 or 30 $\mu g/m^3$.

[125] Peel JL, Tolbert PE, et al. Ambient air pollution and respiratory emergency department visits. *Epidemiology* 2005; 16: 164-174.
[126] Strickland MJ, Darrow LA, Klein M, Flanders WD, Sarnat JA, Waller LA, Sarnat SE, Mulholland JA, Tolbert PE. Short-term associations between ambient air pollutants and pediatric asthma emergency department visits. *Am J Respir Crit Care Med* 2010; 182 (3): 307-16.
[127] Burnett RT, Brook J, et al. Association between particulate- and gas-phase components of urban air pollution and daily mortality in eight Canadian cities. *Inhal Toxicol* 2000; 12 Suppl 4: 15-39.
[128] Mar TF, Norris GA, et al. Associations between air pollution and mortality in Phoenix, 1995-1997. *Environ Health Perspect* 2000; 108: 347-353.
[129] Fairley D. Daily mortality and air pollution in Santa Clara County, California 1989-1996. *Environ Health Perspect* 1999; 107: 637-641.

**Figure 2-9. Summary of Effect Estimates (per 10 µg/m$^3$) and Air Quality Distributions for Single-City, Short-term $PM_{2.5}$ Exposure Studies**

| Study/Cite | Geographic Area | Years of Air Quality Data | Endpoint | Air Quality Data (µg/m$^3$) Mean | Air Quality Data (µg/m$^3$) 98th percentile[a] | Effect Estimate (95% CI) |
|---|---|---|---|---|---|---|
| Lisabeth et al. 2008 | Nueces County, TX | 2001-2005 | Ischemic Stroke/TIA HA | 7.0[b] | 13.6 | |
| Rabinovich et al. 2006 | Denver | 2 winters 2001-2003 | Asthma Medication Use (children) | 7.4 | 17.2 | |
| Stieb et al. 2000 | St. John, Canada | 1992-1996 | CV ED Visits | 8.5 | 27.3 | |
| | | | Respiratory ED Visits | | | |
| Villeneuve et al. 2006 | Edmonton, Canada | 1992-2002 | Hemhrgc stroke HA | 8.5 | 24.0 | |
| | | | Ischemic stroke HA | | | |
| | | | TIA stroke HA | | | |
| Mar et al. 2004 | Spokane | 1997-1999 | Respiratory Symp(any) adults | 9.8[c] | 25.8 | |
| | | | Respiratory Symp (any) children | | | |
| Rabinowitch et al.2004 | Denver | 3 winters 1999-2002 | Asthma exacerbation (children) | 10.6[d] | 29.3 | |
| Slaughter et al. 2005 | Spokane | 1995-2001 | CVD HA and ED visits | 10.8 | 29.6 | |
| | | | Respiratory HA and ED Visits | | | |
| Pope et al. 2008 | Wasatch Front UT[e] | 1993-2006 | CHF HA | 10.8c | 44.5 | |
| Schreuder et al.2006 | Spokane | 1995-2002 | Cardiac HA | 10.8[d] | 29.6 | |
| | | | Respiratory ED | | | |
| Peters et al. 2001 | Boston | 1995-1996 | MI HA | 12.1 | 28.2 | |
| Delfino et al. 1997 | Montreal | Summer 1993 | Respiratory HA | 12.1 | 31.2 | |
| Wilson, et al. 2007 | Phoenix-central | 1995-1997 | CV mortality | 13.0 | 31.6 | |
| | Phoenix-middle | | | | | |
| | Phoenix-outer | | | | | |
| Mar et al. 2003 | Phoenix | 1995-1997 | CV mortality | 13.5[d] | 32.2 | |
| Fairley 2003 | Santa Clara Co, CA | 1989-1996 | Nonaccidental mortality | 13.6 | 59.0 | |
| Ostro et al. 2003 | Coachella Valley, CA | 1996-1998 | CV mortality | 15.8[d] | 33.8 | |




[a]98th percentile of $PM_{2.5}$ distribution was either provided by study authors or calculated from data provided by authors. [b]Mean not reported in study; median presented. [c]Mean estimated from data in study. [d]Mean value slightly different from value reported in published study; mean was either provided by study authors or calculated from data provided by study authors. [e]3 nearly contiguous metro areas: Ogden, Salt Lake City, Provo/Orem

These studies provide strong support for a daily $PM_{2.5}$ standard of 25 µg/m$^3$or below. For example, even with an annual average concentration of 10.8, the Pope et al 2008 study of the Wasatch Front shows that 98$^{th}$ percentile $PM_{2.5}$ concentrations of 44.5 µg/m$^3$were increasing the risk of congestive heart failure. The Wilson, et al 2007 study in central Phoenix reported positive statistically significant associations with daily $PM_{2.5}$ 98$^{th}$ percentile concentrations of 31.2 µg/m$^3$and deaths from heart disease, in an area with annual average concentrations of 13 µg/m$^3$. This study demonstrates that the proposed 24-hour standard of 35 µg/m$^3$ will not be protective in conjunction with an annual average standard of 13 µg/m$^3$. The Delfino, et al 1997 study of respiratory hospital admissions in Montreal indicates that the proposed standard of 35 will not be sufficient, even in conjunction with an annual average standard of 12 µg/m$^3$. Stieb et al, 2000 reported a positive statistically significant association with emergency department visits for cardiovascular causes in St. John, Canada, with annual average $PM_{2.5}$ concentrations of 8.5 µg/m$^3$, and 98$^{th}$ percentile concentrations of 27.3 µg/m$^3$. This study demonstrates that even with an annual standard of 11 µg/m$^3$or below, a daily $PM_{2.5}$ standard of 30 will not be protective of public health. The Mar, et al 2004 study in Spokane makes the same case, though 24-hour 98$^{th}$ percentile concentrations in this study are even lower, at 25.8 µg/m$^3$.

Beyond the studies discussed here, there are additional single-city studies discussed in the ISA that were conducted in areas that would likely not have met both the current annual and 24-hour

standards (e.g., Ito et al., 2007; Sheppard, 2003; Burnett, 1997). These studies provide further evidence that even in areas where the mean daily exposures are kept below 12, peak 24-hour concentration days below 35 µg/$m^3$ 98$^{th}$ percentile, are still causing serious adverse health effects.

Taken together, the single-city studies provide strong support for lowering the 24-hour average standard for $PM_{2.5}$ to 25 µg/$m^3$or below, for all standard combinations under consideration.

## Subdaily $PM_{2.5}$ Exposures Impact Heart Health

Short-term exposures to $PM_{2.5}$ for as little as 30 minutes have been shown to cause adverse cardiovascular effects. 106 nonsmokers were equipped with personal $PM_{2.5}$ monitors and were given electrocardiograms to investigate the acute effects and the time course of fine particulate pollution $PM_{2.5}$ on predictors of atrial fibrillation, or flutter. A personal $PM_{2.5}$ monitor was used to measure individual-level, real-time $PM_{2.5}$ exposures during a 24-hour period, and corresponding 30-min average $PM_{2.5}$ concentration were calculated. Higher $PM_{2.5}$ was found to be associated with increases in measures of atrial fibrillation. Maximal effects were observed within 2 hours. These findings suggest that $PM_{2.5}$ adversely affects atrial fibrillation predictors; thus, $PM_{2.5}$ may be indicative of greater susceptibility to atrial fibrillation.[130]

A related study examined the effects and time course of exposure to $PM_{2.5}$ on cardiac arrhythmia in 105 middle-age community-dwelling healthy nonsmokers in central Pennsylvania. The 30-min mean ± SD for $PM_{2.5}$ exposure was 13 ± 22 µg/$m^3$. $PM_{2.5}$ exposure within approximately 60 min was associated with increased premature ventricular contraction counts in healthy individuals.[131]

An important 2012 study demonstrates that $PM_{2.5}$ levels below the current NAAQS increases the risk of ischemic stroke. Stroke is the third largest cause of death and a major cause of disability in the United States.

The study examined the medical records for 1705 Boston area patients hospitalized with confirmed ischemic stroke, and extracted information on the time of symptom onset and clinical characteristics. $PM_{2.5}$ concentrations were measured at a central monitoring station. A time-stratified case-crossover design assessed the association between risk of ischemic stroke onset and $PM_{2.5}$ levels in the days and hours preceding each event. Risk of stroke was 34 percent higher on days with moderate $PM_{2.5}$ levels compared with days with good levels, as characterized by the Air Quality Index. Researchers found

[130] Liao D, Shaffer ML, He F, Rodriguez-Colon S, Wu R, Whitsel EA, Bixler EO, Cascio WE. Fine particulate air pollution is associated with higher vulnerability to atrial fibrillation--the APACR study. *J Toxicol Environ Health A* 2011; 74: 693-705.
[131] He F, Shaffer ML, Rodriguez-Colon S, Yanosky JD, Bixler E, Cascio WE, Liao D. Acute effects of fine particulate air pollution on cardiac arrhythmia: the APACR study. *Environ Health Perspect* 2011; 119: 927-32.

that the risk of stroke onset was most strongly associated with $PM_{2.5}$ exposure, but also significantly associated with exposure to black carbon and $NO_2$, markers of traffic pollution. In conclusion, these results suggest that $PM_{2.5}$ exposure increases the risk of ischemic stroke at levels below those currently considered safe under US regulations. These associations can be observed within hours of exposure and are most strongly associated with pollution from local or transported traffic emissions.[132]

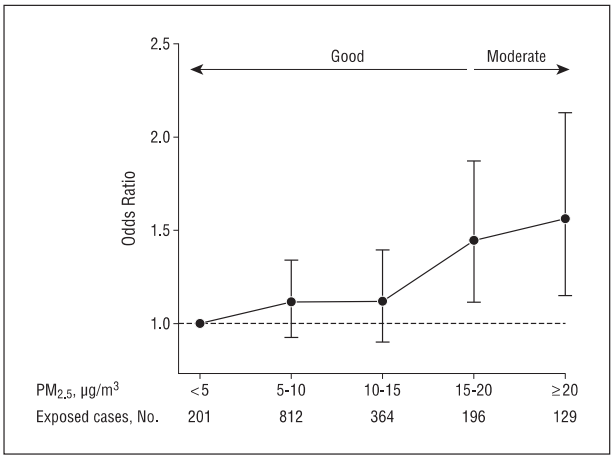



**Figure 1.** Odds ratio of ischemic stroke onset for US Environmental Protection Agency categories (*good* and *moderate*) of mean ambient fine particulate matter air pollution ($PM_{2.5}$) levels in the 24 hours preceding stroke onset. Error bars indicate 95% CIs.

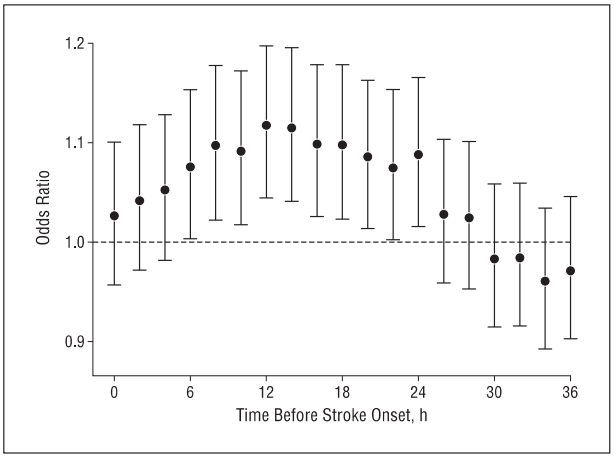



**Figure 2.** Odds ratio of ischemic stroke onset per interquartile range increase in concentration of ambient fine particulate matter air pollution (6.4 µg/m$^3$) in the hours preceding stroke onset. Error bars indicate 95% CIs.

[132] Wellenius GA, Burger MR, Coull BA, Schwartz J, Suh HH, Koutrakis P, Schlaug G, Gold DR, Mittleman MA. Ambient Air Pollution and the Risk of Acute Ischemic Stroke. *Arch Intern Med* 2012; 172: 229-234.

This study is significant because positive, statistically significant associations were observed in an area that is in attainment of the PM NAAQS. Investigators estimate that a 2 $\mu g/m^3$ reduction in daily $PM_{2.5}$ levels might have averted more than 6,000 stroke hospitalizations in the Northeastern U.S. in 2007 alone.

Studies of subdaily exposures inform the need to strengthen the short-term $PM_{2.5}$ standard, because the 24-hour standard must protect against high subdaily exposures in the absence of a short-term hourly standard.

## EPA Must Strengthen the 24-hour $PM_{2.5}$ Standards to Protect the Public from Health Effects of Short-Term Exposures

There is strong evidence of the adverse effects of short-term exposure to $PM_{2.5}$. The scientific evidence supporting a lower 24-hour $PM_{2.5}$ standard includes studies examining the temporal lag patterns of the health effects of $PM_{2.5}$ as well as mechanistic evidence suggesting immediate triggers of PM-induced effects. The Integrated Science Assessment (ISA) concluded that there was a causal relationship between short-term $PM_{2.5}$ exposures and cardiovascular effects and mortality, and a likely causal relationship with respiratory effects.[133] These are serious health outcomes that demand stronger 24-hour air quality standards for $PM_{2.5}$.

Kim et al. investigated temporal patterns in a recent study examining $PM_{2.5}$ concentrations by disease category on hospital admissions in Denver. They found that $PM_{2.5}$ exposure was associated with cardiovascular hospital admissions on the same day with the pattern most dominant for elemental carbon and organic carbon, and for ischemic heart disease, while respiratory admissions had the strongest associations with elemental carbon and organic carbon and asthma after a delay of 2-5 days.

In the ISA, EPA identifies a plethora of new inhalation studies that examine modes of action specific to acute exposure of PM. As reported in the ISA, the following results of acute PM exposure have been reported in the literature:

> "Altered lung function including changes in respiratory frequency and AHR following short-term exposures to CAPs and combustion-derived PM (Section 6.3.2.3)
>
> Mild pulmonary inflammation in response to short-term exposures to CAPs, urban air, combustion-derived PM and carbon black (Section 6.3.3.3)

[133] EPA, ISA, 2009.

Mild pulmonary injury in response to short-term exposure to CAPs and combustion derived PM (Section 6.3.5.3)

Inhibition of cell proliferation in the proximal alveolar region of neonatal animals following short-term exposure to iron-soot (Section 6.3.5.3)

Pulmonary oxidative stress in response to short-term exposure to CAPs, urban air, combustion-derived PM, carbon black and iron-soot; pulmonary nitrosative stress in response to titanium dioxide (TiO2) (Section 6.3.4.2)

Antioxidant intervention which ameliorates PM effects on oxidative stress, allergic responses, and AHR (Sections 6.3.4.2 )

Allergic sensitization and exacerbation of allergic responses in response to CAPs and combustion-derived PM (Section 6.3.6.3)

Altered methylation of promoter regions of IFN-γ and IL-4 genes suggestive of proallergic Th2 gene activation following short-term exposure to combustion-derived PM in an allergy model (Section 6.3.6.3)

Increased susceptibility to respiratory infection following exposure to combustion derived PM (Section 6.3.7.2)

Effects on nasal epithelial mucosubstances, airway morphology and airway mucosubstances following chronic exposure to urban air-derived PM and woodsmoke (Section 7.3.5.1)

Worsening of papain-induced emphysema following chronic exposure to urban air derived PM (Section 7.3.5.1)

Effects on lung development following chronic exposure to urban air-derived PM (Sections 7.3.2.2 and 7.3.5.1)

Prolonged exposure to CAPs and combustion-derived PM leading sometimes to mild pulmonary inflammation, oxidative stress and injury and sometimes to loss of inflammatory, oxidative stress and AHR responses which were observed after short-term exposures (Sections 7.3.2.2, 7.3.3.2, 7.3.4.1, 7.3.5.1 and 7.3.6.2)

Hypermethylation of lung DNA following chronic exposure to combustion-derived PM (Section 7.3.5.1)

A role for TRPV1 irritant receptors in activating local axon and CNS reflexes following short-term exposure to CAPs and combustion-derived PM (Section 6.2.9.3)

A role for TRPV1 irritant receptors in mediating lung and heart oxidative stress through increased parasympathetic and sympathetic activity in response to CAPs (Sections 6.2.9.3 and 6.3.4.2)

Altered heart rate variability in response to CAPs, combustion-derived PM and carbon black (Section 6.2.1.3)

Arrhymthmic events in response to CAPs and combustion-derived PM (Section 6.2.2.2)

Altered cardiac contractility following short-term exposure to CAPs and carbon black (Section 6.2.6.1)

Enhanced myocardial ischemia following short-term exposure to CAPs (Section 6.2.3.3)

Endothelial dysfunction and altered vascular reactivity following short-term exposure to CAPs, combustion-derived PM and TiO2 (Section 6.2.4.3)

Increases in blood pressure following short-term exposure to CAPs and carbon black (Section 6.2.5.3)

Changes in blood leukocyte counts following short-term exposure to CAPs and carbon black (Section 6.2.7.3)

Increased levels of blood coagulation factors following short-term exposure to CAPs and on-road highway aerosols (Section 6.2.8.3)

Systemic and cardiovascular oxidative stress in response to short-term exposure to CAPs, road dust and combustion-derived PM (Section 6.2.9.3)"[134]

[134] EPA, ISA 2009.

This data in part provides evidence that led EPA to conclude that a causal relationship exists between short-term exposures to $PM_{2.5}$ and cardiovascular effects (ISA 2-10).[135]

## Risk Assessment Limitations and Findings

The recent risk analysis completed by EPA in estimating $PM_{2.5}$ exposures using alternative and daily standards was limited. EPA focused on only 15 urban areas to represent the continental U.S. and only examined a fraction of the available combinations of annual and daily standards.[136] EPA indicates that the Risk Assessment likely underestimates the risks PM poses to public health:

> we believe it unlikely that the [Risk Assessment] as implemented has over-stated risk, particularly for long-term $PM_{2.5}$ exposure-related mortality. In fact, the core risk estimates for this category of health effect endpoint may well be biased low based on consideration of alternative model specifications evaluated in the sensitivity analysis.[137]

In light of the likelihood that the Risk Assessment underestimates risk, and the mandate that a NAAQS protect human health "allowing an adequate margin of safety," the measurements of risk should be treated conservatively.

The Risk Assessment examines 15 urban areas "representative of urban areas in the U.S. experiencing elevated levels of risk related to ambient $PM_{2.5}$ exposure" in order to estimate the reduction in risk that would accrue from bringing those areas into compliance with various combinations of annual and 24-hour standards.[138] The standards considered are the current standards, as well as different combinations of annual and 24-hour standards.

In the Risk Assessment the alternative suites of standards were chosen in order to result in "a mixture of behavior in terms of which standard would control across the various urban study areas."[139] In addition to the current standard, the alternative suites of standards considered were 14 $\mu g/m^3$ annual and 35 $\mu g/m^3$ daily, 13 $\mu g/m^3$ annual and 35 $\mu g/m^3$ daily, 12 $\mu g/m^3$ annual and 35 $\mu g/m^3$ daily, 13 $\mu g/m^3$ annual and 30 $\mu g/m^3$ daily, and 12 $\mu g/m^3$ annual and 25 $\mu g/m^3$ daily.[140]

---

[135] EPA ISA, 2009.
[136] American Lung Association, Clean Air Task Force, and Earthjustice, 2011. Sick of Soot: How the EPA Can Save Lives by Cleaning Up Particle Pollution. Available at: http://earthjustice.org/sites/default/files/SickOfSoot_2011.pdf
[137] Risk Assessment at 5-16.
[138] Risk Assessment at 2-7, 5-6.
[139] Risk Assessment at 2-13.
[140] Risk Assessment at 2-13.

The standard proposed by the Administrator is both narrower and less protective of human health than the suites of alternative standards considered in the Risk Assessment: the Administrator has proposed retaining the 35 μg/$m^3$ 24-hour standard in conjunction with an annual standard of between 12 and 13 μg/$m^3$.[141]

Given the higher levels of $PM_{2.5}$-related risk faced by residents of these urban areas, it is telling that the least ambitious combination of standards proposed by EPA (13 μg/$m^3$ annual and 35 μg/$m^3$ daily) is not anticipated to reduce risk in 6 of the 15 cities.[142] Indeed, as the Risk Assessment is careful to note, even the most aggressive standard in the proposed rulemaking (12 μg/$m^3$ annually and 35 μg/$m^3$ daily) is not anticipated to have any measurable effect on risk in three of the 15 cities.[143] Only "when alternative 24-hour standards were considered," i.e., standards more stringent than the 35 μg/$m^3$ standard in the proposed rule, did the Risk Assessment anticipate risk reductions for these three cities.[144] Two of these three cities, Tacoma and Salt Lake City, are in Northwest region, which is the region most likely to be affected by peaks from seasonal burning, which strongly undermines the claim that 35 μg/$m^3$ provides "supplemental" protection.

Dr. McCubbin's analysis expanded on EPA's analysis to include alternative standards that are national in scope. As demonstrated in the Table below, the largest health benefits are achieved with an annual standard of 11 μg/$m^3$ and daily standard of 25 μg/$m^3$. The analysis also makes clear that there are enormous incremental benefits to lowering 24-hour standard to 30 μg/$m^3$or 25 μg/$m^3$at every annual average standard under consideration

[141] 77 Fed. Reg. at 38890.
[142] Risk Assessment at 4-24. The six cities were: Dallas, TX; Fresno, CA; Los Angeles, CA; Phoenix, AZ; Salt Lake City, UT; and Tacoma, WA.
[143] Risk Assessment at 4-24. The three cities were: Fresno, CA; Salt Lake City, UT; and Tacoma, WA
[144] *Id.*

**Table 1. Updated Air Quality Standards for Fine Particles Will Save Lives: Estimated Premature Deaths Avoided in U.S. by Alternative PM2.5 Air Quality Standards and Epidemiological Study (relative to current air quality), Mean and 90% Confidence Interval**

| Scenario | Krewski *et al.* (2009) | Laden *et al.* (2006) |
|---|---|---|
| A15D35 | 2,540 | 5,240 |
| | (1,850 – 3,220) | (2,850 – 7,570) |
| **A13D35** | **3,700** | **8,190** |
| | **(2,700 – 4,700)** | **(4,450 – 11,900)** |
| **A13D30** | **6,410** | **13,500** |
| | **(4,680 – 8,130)** | **(7,330 – 19,600)** |
| A13D25 | 16,700 | 32,700 |
| | (12,200 – 21,200) | (17,700 – 47,300) |
| **A12D35** | **6,380** | **15,000** |
| | **(4,650 – 8,100)** | **(8,140 – 21,800)** |
| A12D30 | 7,980 | 17,500 |
| | (5820 – 10,100) | (9,490 – 25,400) |
| A12D25 | 16,800 | 33,000 |
| | (12,300 – 21,300) | (17,900 – 47,800) |
| **A11D35** | **11,200** | **27,300** |
| | **(8,200 – 14,300)** | **(14,800 – 39,600)** |
| A11D30 | 12,100 | 27,900 |
| | (8,830 – 15,400) | (15,100 – 40,400) |
| A11D25 | 17,900 | 35,700 |
| | (13,100 – 22,700) | (19,400 – 51,800) |

Bold type highlights options put forth by EPA in its Policy Assessment

EPA's proposed standards are not adequate to protect public health, and will result in thousands of additional deaths and illnesses every year, particularly when compared to levels of 25 µg/m$^3$ or below.

## Health Benefits of Alternative PM2.5 Standards

In November 2011, the American Lung Association (ALA), Clean Air Task Force (CATF) and Earthjustice released a pair of reports to build upon EPA's June 2010 Quantitative Health Risk Assessment for Particulate Matter (EPA-452/R-10-005) and Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards (EPA 452/R-11-003). The primary report, *Sick of Soot*, recommended that EPA tighten the current standard to an annual level of 11 µg/m$^3$ and a daily level of

25 µg/m$^3$.[145] The findings presented in *Sick of Soot* were drawn directly from a technical report prepared by Dr. Donald McCubbin, (Health Benefits of Alternative $PM_{2.5}$ Standards, July 2011).[146]

The McCubbin report expands upon EPA's efforts to characterize the potential health benefits from revised PM standards by conducting a nationwide analysis of potential $PM_{2.5}$ standards. EPA's risk assessment focused on 15 major urban areas while looking at alternative annual standard levels that spanned 12 to 15 µg/m$^3$ annual combined with a daily standard covering a range of 25 to 35 µg/m$^3$. EPA's staff concluded "...that the evidence most strongly supports consideration of an alternative annual standard level in the range of 12 to 11 µg/m$^3$." As a result, the *Sick of Soot* work covered the full range of possible $PM_{2.5}$ standards under consideration by EPA.

The goal of the report was to conduct a national analysis of the mortality and morbidity benefits of a range of annual and daily standards relying on the same types of tools that EPA uses for its own work. The report employed Benefits Mapping and Analysis Program (BenMAP), the model which EPA regularly uses in regulatory benefit analyses to estimate $PM_{2.5}$-related human health impacts and their associated economic valuation.

This effort differed in some key aspects from EPA's assessments, and adds substantial new information for consideration of the impacts and benefits of setting new air quality standards for $PM_{2.5}$. First, while EPA's risk assessment focused on 15 major urban areas in the country using air quality data from 2005-2007, the *Sick of Soot* work covered the entire U.S. using more recent PM data from 2007-2009. Since our air quality continues to improve, relying on more recent data better reflects the potential benefits to meeting new air quality standards. The Sick of Soot study followed a novel approach in its assessment by using three-year average air quality values to determine whether or not a region would need to improve its air quality to meet the standards. The rationale is based on the requirement that three years of data be used to determine compliance with the air quality standard. This is in contrast to EPA's risk assessment, which considers each year individually.

To help avoid overestimating the likely impacts of alternative standards, conservative assumptions were used throughout the analysis. When calculating mortality, no effects were quantified below the lowest measured level found in the study. This was particularly important for estimates based on Laden et al. (2006), with a lowest measured level (LML) of 10 µg/m$^3$; assuming no effect below the LML reduced the estimated mortality by almost 30 percent for some standard combinations. In

[145] American Lung Association, Clean Air Task Force, and Earthjustice, 2011. Sick of Soot: How the EPA Can Save Lives by Cleaning Up Particle Pollution. Available at: http://earthjustice.org/sites/default/files/SickOfSoot_2011.pdf

[146] McCubbin, Donald. Health Benefits of Alternative $PM_{2.5}$ Standards. A technical report prepared for the American Lung Association, Clean Air Task Force, and Earthjustice, July 2011. Available at: http://earthjustice.org/sites/default/files/Health-Benefits-Alternative-$PM_{2.5}$-Standards.pdf

addition, the rollback approach used in the analysis tends to result in relatively small changes in $PM_{2.5}$ levels, in comparison to the rollback approaches used by EPA in their risk assessment. $PM_{2.5}$ reductions were constrained to areas within 60 km of a monitor exceeding a standard.

Importantly, health studies continue to observe adverse health effects with no apparent threshold. Of particular note is the 2012 Canadian National Cohort study by Crouse et al. published in *Environmental Health Perspectives*, which observed similar hazard ratios as previous studies at substantially lower $PM_{2.5}$ exposures (mean of 8.7 $\mu g/m^3$). Even the lowest levels of annual $PM_{2.5}$ assumed in our analysis far exceeds the level from the Canadian study. The Crouse study further supports the position that the $PM_{2.5}$ NAAQS be revised to the lowest level to best protect public health with an adequate margin of safety.

The figures and table that follow highlight some of the key results of the *Sick of Soot* study. All benefits are relative to air quality from 2007-2009, results that are more current than EPA's risk assessment. Considering that these results are based on actual air quality data, they may be more informative than those presented in EPA's June 2012 Regulatory Impact Analysis (RIA) (EPA-452/R-12-003). Key uncertainties of that review include the use of a 2005 air quality baseline and reliance on air quality modeling projections to 2020. Future modeled air quality depends strongly on assumptions about future emissions, whose true location and magnitude cannot easily be determined, especially for rules that have yet to be fully implemented. Case in point, on August 21 the courts struck down the Cross State Air Pollution Rule (CSAPR), one of the key rules shown to reduce future $PM_{2.5}$ concentrations in the RIA.

The results help to illustrate the increasing benefits of more stringent standards. Given our current best understanding of the health impacts of $PM_{2.5}$, we concluded the most protective choice of the options considered was an annual standard of 11 $\mu g/m^3$ paired with a daily standard of 25 $\mu g/m^3$. Our results, (based on Laden et al (2006), suggest that up to 35,000 premature deaths could be avoided, in addition to substantial reductions in other associated adverse health consequences. The benefits are valued at nearly $300 billion. This benefit far exceeds what the public might enjoy under the two options EPA has highlighted in its proposal 12/35 and 13/35, with nearly 21,000 to 28,000 lives saved each year.

Figure 1 displays key avoided premature mortality results from the *Sick of Soot* study. Clearly a revised standard of 13/35 would have only marginal health benefits. The benefits nearly double under the 12/35 combination and quintuple for our preferred option of 11/25. Reductions in either or both the annual or daily standards will provide public health benefits, which increase markedly under the more protective options.

Although the direct health impacts from the study are important considerations, related results are given in Table 1. Data for the current and five potential revised standard combination are tabulated. The first row shows the percent of counties that would experience reductions in $PM_{2.5}$ under the different standards, ranging from 14 to 75 percent. The associated population covers a range of 36 to 86 percent. From this we can conclude that counties that currently exceed the 15/35 standard have higher populations on average than counties with lower $PM_{2.5}$ concentrations. As the level of the standard gets reduced, the general trend will be for more counties and people to benefit from reduced exposure to $PM_{2.5}$. The final row provides the average $PM_{2.5}$ reduction per person that would be required to meet the various standard levels. The average per person benefit grows as the standards are tightened. Considering that benefits of reduced exposure are in many cases linear, this finding suggests an overall health benefit that is more substantial than the simple increase in populations. In other words, as the standards are lowered, benefits accrue across a wider spatial domain, for increasing populations, with an increased average benefit per person. Thus, each unit reduction in the standard will likely have increasing public health benefits.

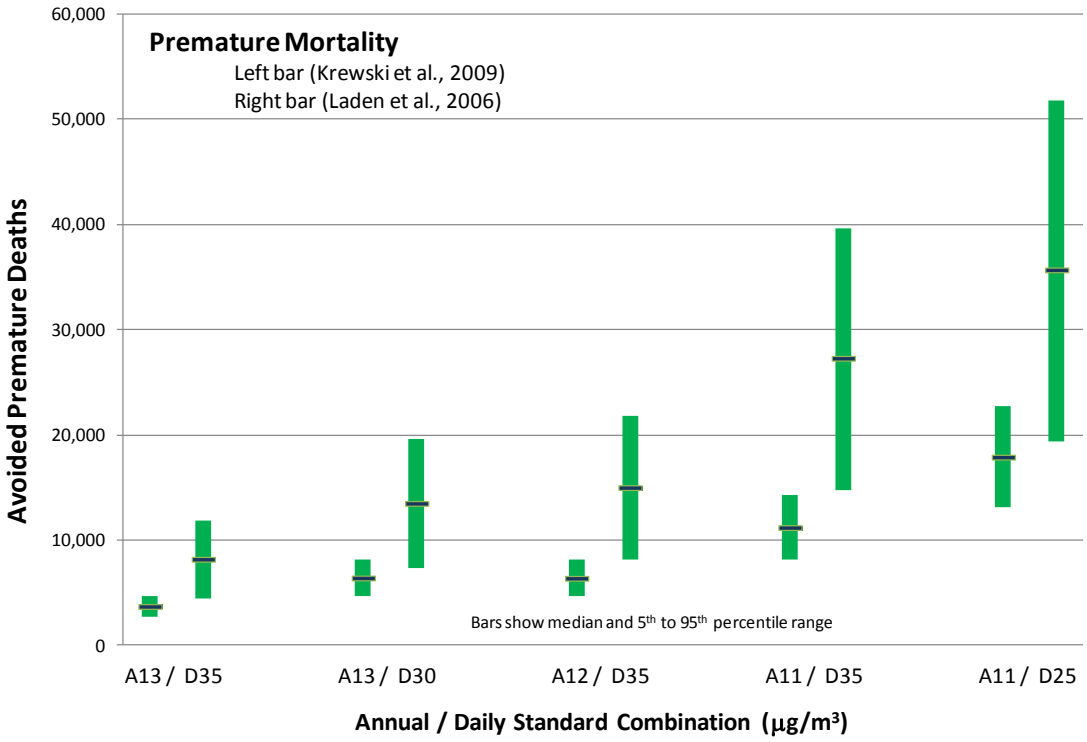



**Figure 1 BenMAP results for premature mortality for various standard combinations considered in *Sick of Soot* study**

| Standard Pair | 15 / 35 | 13 / 35 | 13 / 30 | 12 / 35 | 12/25 | 11 / 25 |
|---|---|---|---|---|---|---|

| (annual / daily) | | | | | | |
|---|---|---|---|---|---|---|
| **% of counties** | 14 | 33 | 41 | 45 | 72 | 75 |
| **% of population** | 36 | 56 | 64 | 63 | 85 | 86 |
| **Average PM benefit** | 0.24 | 0.33 | 0.58 | 0.55 | 1.46 | 1.53 |

**Table 1 Results from *Sick of Soot* analysis for various standard combinations. Note 91% of adults in the U.S. were included in the total population, derived from 2,523 counties that were with 60 km of an existing air quality monitor.**

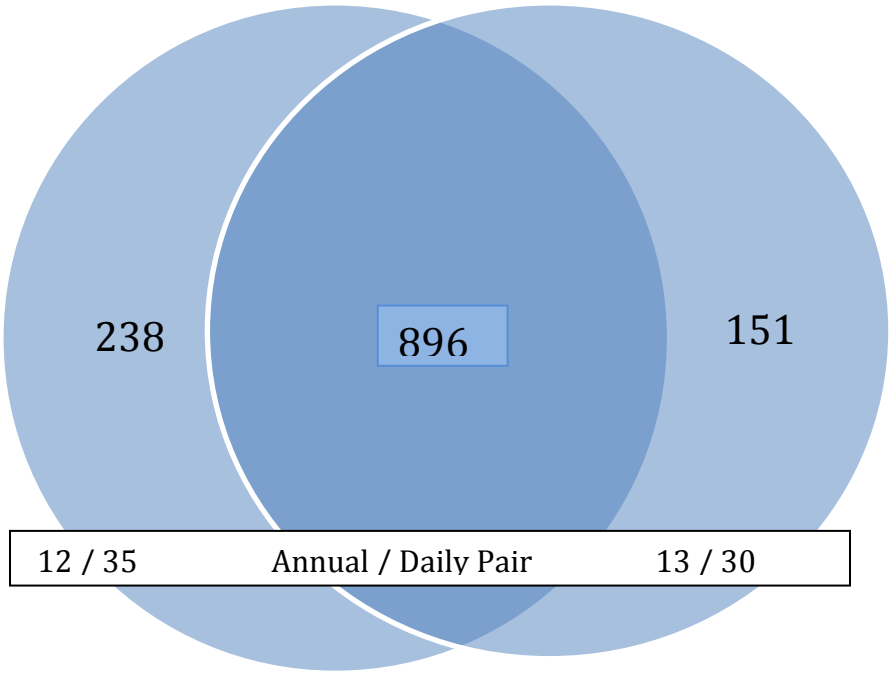



**Figure 2 Comparison of counties that would experience reduced $PM_{2.5}$ to meet a standard pairing of 12/35 or 13/30**

Results from this work revealed very similar health benefits, as shown in Figure 1, for the two standard pairings 13/30 and 12/35. These avoided premature mortalities are consistent with the results shown in Table 1. Under the 13/30 combination, the average $PM_{2.5}$ benefit is slightly larger, but covers a somewhat smaller number of counties and overall population. The Venn diagram reveals that 896 counties with roughly 93 million people (about 90% of the covered population under either standard combination) would enjoy air quality benefits under either standard combination. The population weighted $PM_{2.5}$ benefit would be 0.97 and 0.95 $\mu g/m^3$ for the 12/35 and 13/30 standards, respectively.

The average $PM_{2.5}$ reduction in the counties not jointly covered would be 0.16 and 0.24 $\mu g/m^3$ for the 12/35 and 13/30 standards.

The key observations from the *Sick of Soot* study are: a standard combination of 11 $\mu g/m^3$ annual and 25 $\mu g/m^3$ daily provides by far the most health protection of any of the options considered. As standard levels move toward lower concentrations, one expects increases in three parameters: spatial coverage, populations protected and average $PM_{2.5}$ reduction. Additionally, different standard combinations may yield similar net health benefits, but would likely cover somewhat different populations.

## EPA Cannot Assume that an Annual Average $PM_{2.5}$ Standard of 13 $\mu g/m^3$ or Below Will Reduce 24-hour Peak Concentrations Below Harmful Levels

Despite the considerable evidence demonstrating health impacts from short-term exposures at levels far below the current 24-hour standard, EPA proposes not to strengthen the 24-hour $PM_{2.5}$ standard based on the claim that changes in air quality designed to meet a lower annual standard would likely result not only in lower annual average $PM_{2.5}$ concentrations but also in fewer and lower peak 24-hour $PM_{2.5}$ concentrations. EPA, however, fails to provide the analysis necessary to support the conclusion that the resulting short-term concentrations will protect public health with an adequate margin of safety. Instead, EPA's argument appears to focus on generalized claims regarding the belief that the annual standard should be controlling. The justifications offered by the preamble simply do not hold up.

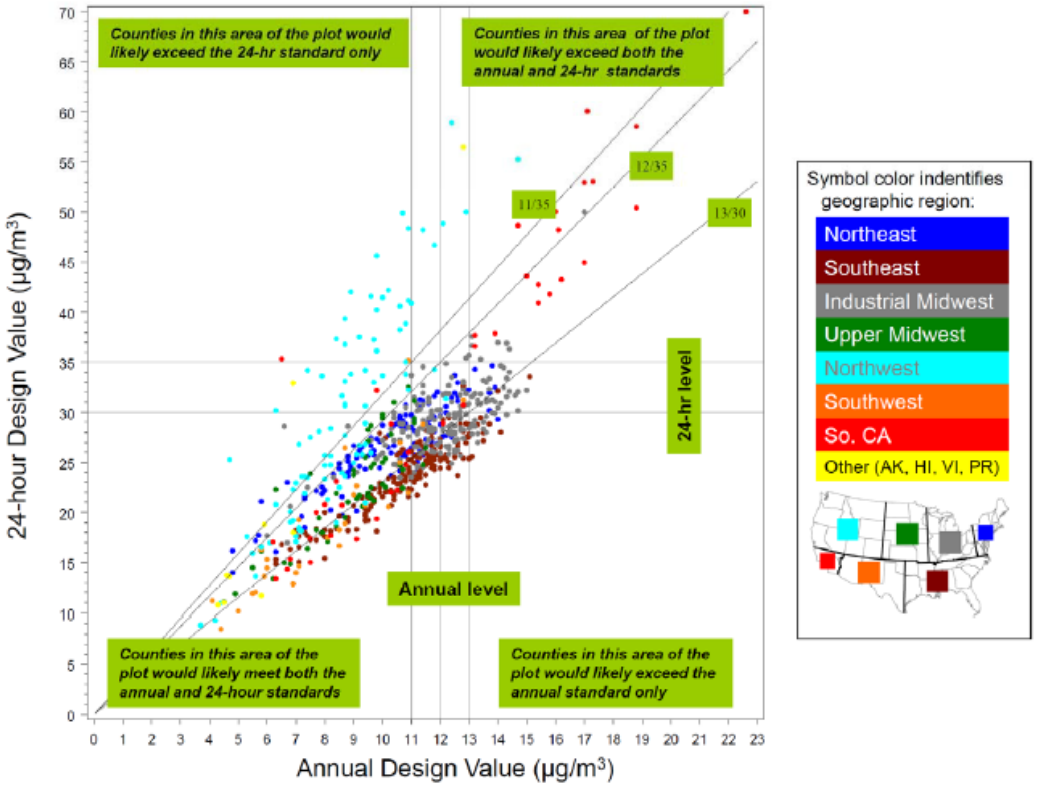
First, the preamble states that the annual standard should be controlling -- but the National Ambient Air Quality Standards must protect public health -- even if the 24-hour standard is controlling in some areas. This whole rationale of the "controlling" standard is not germane to selecting standards, and has no statutory basis in the Clean Air Act.

Second, the Policy Assessment purports to set a 24-hour standard based on the average peak to mean ratio of 2.5 in most, but not all, regions of the country. (In the Northwest, the ratio is approximately 3.5). The Policy Assessment states that it is reasonable to focus on 24-hour standards that are at least 2.5 times the annual standard. This is used to argue that the current level of the standard of 35 $\mu g/m^3$ meets this criterion. But again, the goal is to identify a level that will protect health, not to maintain the annual standard as controlling.

Different areas have different distributions of 24-hour exposures, with some areas having fairly steady levels year-round, and other areas having large variation between peak and average concentrations. The health effects associated with acute and chronic $PM_{2.5}$ exposures are distinct. EPA, therefore,

must adopt standards that individually or collectively address both sets of exposures. As reflected in the scatterplot of the county level 24-hour design values versus annual design values in the Figure below, the Northwest has significantly higher ratios of 24-hour to annual design values for particulate matter as compared to other parts of the country.

**Figure 2-10. County-level 24-hour DVs versus Annual DVs, 2007-2009**




Source: Schmidt, 2011a, Analysis B

CASAC, in commenting on the second draft Policy Assessment, noted that the approach which focused on peak-to-mean ratios was not relevant to informing the actual level (Samet 2010a, p. 4).

The assertion that the annual average should be used a controlling standard is not scientifically valid. As pointed out by CASAC member Dr. Christopher Frey,

> First, it is not at all clear as to why the annual standard should be "generally controlling." The NAAQS should provide health protection for both long-term and short-term health effects. It is not clear, for example, as to why the 24-hour level should be at least 2.5 times higher than the annual standard. Such a statement seems to be independent of consideration of health effects. A statement is made on page 2-73, lines 26-27 that "based on this consideration" consideration should be given to retaining the 35 µg/m$^3$24-hr level in conjunction with annual standards of 13

to 11µg/$m^3$. Setting aside the math problem here (e.g., 11*2.5 = 27.5, not 35), the rationale here does not appear to be based on health effects, and thus appears not to be valid. While it is useful to have insight as to what combinations of annual and 24 hour levels would lead to the annual standard being controlling in a given area, it is not clear why the policy objective should be set both levels such that the annual standard is generally controlling. [147]

CASAC member Mr. Rich Poirot also highlights the illogical assessment of the annual standard as controlling:

> "I also wanted to confess ignorance and request clarification on the rationale for the proposed need to "pair" the annual and 24-hour $PM_{2.5}$ standards such that the annual standard would remain the "controlling" standard. I don't understand why this is logical or desirable, as it would seem inconsistent with the observations of separate kinds of effects resulting from acute and chronic exposures to $PM_{2.5}$ pollution. It also seems like this has become, is becoming or soon will become a less desirable air quality management approach as progress is made (and continues with CAIR) on reducing the large regional source influences most important for high annual concentrations over large areas. The scatter plot Figure 2-10 (page 2-75) and the Figure 2-9 box/whiskers on the preceding page do seem to indicate that a majority of US sites have 98th percentile 24-hour concentrations which are about 2.5 times their annual means, but that there are a number of sites particularly in the Northwest that have ratios of 3.5 to 1 or higher. Taking a closer look at data from that region, I think many of these sites are in relatively deep mountain valley locations, with strong winter seasonal early morning peaks under stagnation/ inversion conditions. Much of the "peakiness" here is due to wood-smoke, other heating fuel burning and gasoline motor vehicle and diesel exhaust, which not only reach much higher than average concentrations on bad days but see even more extreme short-term hourly morning peaks during rush hour. Are these sources, their associated carbonaceous aerosols, and extreme temporal exposure regimes so benign that control efforts should focus instead on the summer ammonium sulfate that tends to dominate chronic exposures in areas which exceed annual standards but not the current 24-hour standard?
> … the short term standard should reflect short-term concentrations at which effects may be expected for sensitive groups – regardless of whether a controlling annual standard is useful for other purposes."[148]

Support for a more protective 24-hour standard is consistent with a study from Northeast States for Coordinated Air Use Management (NESCAUM), demonstrating that peak concentrations of 24-hour PM

[147] CASAC Letter, September 10, 2010.
[148] CASAC Letter, September 10, 2010.

persist even with lower annual standards. NESCAUM's 2005 report indicates that a more protective 24-hour standard would bring additional health benefits to an overwhelming percentage of the population in the Northeast (see Figure below).

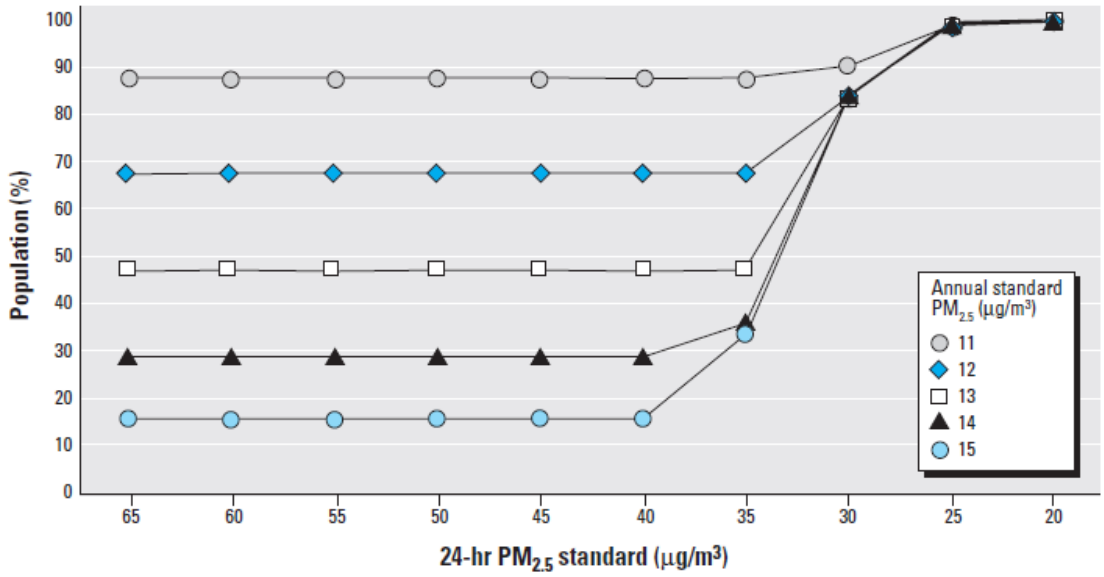



**Figure 1.** Percentage of the northeastern population that would benefit from compliance with alternative annual/24-hr $PM_{2.5}$ (98th percentile) standards.

This research provides additional support for the idea that annual standards alone would not adequately protect all Americans, especially those living in areas with higher daily PM values, such as those in the Northeast and Northwest.

Recent evidence suggests that seasonal sources such as wood burning and agricultural burning can cause high daily PM concentrations as well as weather events known as inversions. In a recent article by the European Journal of Epidemiology, Madsen et al. note an association between elevated short-term exposure to common urban traffic pollutants together with occasional wood burning during the wintertime and non-accidental mortality among individuals above 50 years of age. The daily average indicated a higher excess in risk from $PM_{2.5}$ exposure that was possible due to sources other than traffic-related PM (spring dust and wood burning, for instance).[149]

Because reductions in the distribution of 24-hour peaks will depend on the types of emission sources controlled in order to comply with the annual average standard, and because different regions have different mixes of sources, it is not reasonable to believe that merely controlling the mean daily

[149] Madsen et al. (2012). The short-term effect of 24-h average and peak air pollution on mortality in Oslo, Norway. Eur. Journal of Epidemiology. DOI 10.1007/s10654-012-9719-1

concentration will be sufficient to address the peak bad days in these areas. Furthermore, EPA cannot presume that peak to mean ratios present today will continue into the future.

For all of these reasons, EPA's "short-cut" analysis for justifying the 24-hour standard based on ratios to a "controlling" annual standard must fail. EPA must demonstrate that the 24-hour standard will protect public health in all areas of the country with an adequate margin of safety. The current 24-hour standard of 35 $\mu g/m^3$ simply does not provide such protection, even with a lowered annual standard.

Conclusion

In conclusion, evidence from long- and short-term community health studies, risk assessments, and air quality analyses compel EPA to lower the 24-hour $PM_{2.5}$ standard, at level of 25 $\mu g/m^3$. Reliance on the annual standard to address the distinct short-term health impacts has no rational basis.

## Coarse Particle Standards

In light of the strengthened evidence that has become available since the last review, the proposal to merely retain the coarse particle standard -- set in 1987 -- is unacceptable. Commenters call on EPA to follow the law and science, and to set more protective coarse standards, as other standard-setting bodies have done.

EPA has proposed no changes to the current air quality standards for $PM_{10}$ – a 24-hour average standard of 150 $\mu g/m^3$, not to be exceeded more than once per year on average over 3 years. This standard was set in 1987, some 25 years ago, and before the completion of thousands of epidemiological studies linking $PM_{10}$ pollution to an array of adverse health effects ranging from increased incidence of respiratory symptoms to emergency department visits and early deaths.

This proposal stands in contrast to the action taken by state of California in 2002, when after a thorough review vetted by the Air Quality Advisory Committee, the California EPA adopted a 24-hour average $PM_{10}$ standard of 50 $\mu g/m^3$, and an annual average $PM_{10}$ standard of 20 $\mu g/m^3$. Both standards are based on "not to be exceeded" forms.[150]

[150] California Air Resources Board. Ambient Air Quality Standards for Suspended Particulate Matter (PM) and Sulfates. Effective July 5, 2003. Accessed at http://www.arb.ca.gov/regact/aaqspm/aaqspm.htm on August 29, 2012.

In contrast to the EPA action in the last review to rescind the annual average $PM_{10}$ standard, the World Health Organization announced in 2006 that it was strengthening its recommended annual average standard for $PM_{10}$ from 70 to 20 $\mu g/m^3$, and its daily $PM_{10}$ guideline from 150 to 50 $\mu g/m^3$ based on the 99th percentile.[151]

A 2011 review of global 24-hour $PM_{10}$ standards found that 43 nations in Asia, Africa, Latin America, Oceana, Europe, and North America had stricter daily $PM_{10}$ standards than the U.S.[152] The United States, once a leader in environmental protection, is now lagging behind many developed and developing nations with respect to air quality standards for $PM_{10}$.

CASAC, in their May 17, 2010 letter to EPA on the first draft PA, found the evidence of coarse particle health effects was much stronger than in the last review.

> Since the last NAAQS review, findings from additional epidemiological studies support the association between $PM_{10-2.5}$ and cardiovascular and respiratory morbidity and mortality. While the certainty of the associations between health effects for the association with $PM_{10-2.5}$ exposure does not match that for $PM_{2.5}$, CASAC also agrees with EPA's conclusion that there is sufficient evidence to consider an adjustment of the $PM_{10}$ standard.

The letter goes on to say:

> although the limited evidence is only suggestive, there are independent differential potential health effects of the $PM_{10-2.5}$ portion of $PM_{10}$ in both urban and rural areas that can be separated from the health impact of $PM_{2.5}$...

The CASAC review letter points to "evidence from the PM ISA showing that coarse thoracic PM exposures are associated with acute respiratory system responses that are not accounted for by fine PM."

Coarse particles are emitted from a variety of sources including traffic, poorly controlled combustion, industrial sources, agricultural burning, construction and demolition. In rural areas, coarse particles may be contaminated by toxicants such as pesticides, polycyclic organic matter, metals, and endotoxin.

---

[151] World Health Organization, 2006. WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global update 2005. Summary of risk assessment. Available at http://whqlibdoc.who.int/hq/2006/WHO_SDE_PHE_OEH_06.02_eng.pdf. Accessed August 29, 2012.

[152] Vahlsing C & Smith KR. Global review of national ambient air quality standards for $PM_{10}$ and $SO_2$ (24 h). *Air Qual Atmos Health*. 2011. DOI 10.1007/s11869-010-0131-2.

Coarse particles are referred to as "thoracic" coarse particles because they can evade the body's defense mechanisms and be breathed deep into the lungs. "Coarse" particles is a misnomer; these are very tiny particles less than 10 microns in diameter.

Coarse particles are associated with serious adverse health effects. As Huang and Brook summarized in a 2011 editorial, most of those documented health effects were based on studies published by 2009 and include decreased lung function, increased respiratory symptoms in children, increased hospital admissions for heart and lung disease, increased doctors' visits for respiratory ailments, and premature death in people with heart or lung disease.[153]

In this review, it appears that for all of the studies using $PM_{10}$ as an indicator, the Agency is assuming that 100 percent of the increased health risk is due to the $PM_{2.5}$ component of $PM_{10}$. Actually, the evidence that this is not the case has grown over the years, so if this assumption ever existed, it should have been stripped away long before now.

Clear evidence that the coarse fraction has independent impacts on health has been available for some time. In their review of coarse particle research in 2005, Brunekreef and Forsberg found "some evidence for effects of coarse particles on mortality" independent of fine. The strongest evidence, they concluded, was found in studies of short-term effects of coarse PM on hospital admissions for COPD, asthma and other respiratory conditions, where "coarse PM has a stronger or as strong short-term effect as fine PM." In addition, they concluded that the toxicological evidence as of 2005 suggested that these particles could "cause respiratory and cardiovascular morbidity that leads to hospital admissions." [154]

EPA's comprehensive review of the overall health evidence for particulate matter in the ISA discusses studies that used $PM_{2.5}$, $PM_{10}$, and $PM_{10-2.5}$ as the indicator pollutant. However, in making causal determinations for "coarse particles," the ISA discusses only the $PM_{10-2.5}$ studies as providing "suggestive" evidence of causality for short-term impacts on cardiovascular effects, respiratory effects and mortality.

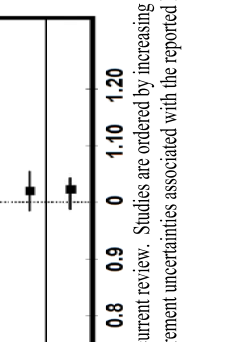
The figure below, taken from the Policy Assessment PA (p. 3-38) is a table summarizing $PM_{10}$ concentrations and effect estimates in eight U.S. single city morbidity studies of $PM_{10-2.5}$. The 98$^{th}$ percentile $PM_{10}$ concentrations range from 49 $\mu g/m^3$ in the Bronx to 105 $\mu g/m^3$ in Seattle, with intermediate concentrations in Manhattan, Boston, Atlanta, Spokane, and Detroit. The studies

[153] Huang Y-C T and Brook RD. The Clean Air Act: Science, Policy, and Politics. *Chest* 2011; 140; 1-2.
[154] Brunekreef B and Forsberg B. Epidemiological evidence of effects of coarse airborne particles on health. *Eu Respir J* 2005; 26; 309-318.

presented in this figure, while not all positive or statistically significant, point to the inadequacy of the current 24-hour $PM_{10}$ standard of 150 µg/$m^3$.

**Figure 3-6. 98th Percentile $PM_{10}$ Concentrations in Locations of U.S. Single-City $PM_{10-2.5}$ Morbidity Studies***

| Study | Location | Morbidity Endpoint | Time Period | Age | 98th $PM_{10}$ (µg/$m^3$) |
|---|---|---|---|---|---|
| NYS DOH, 2006 | Bronx | Asthma HA | 1999-2000 | All | 49 |
| | Manhattan | | | | 52 |
| Peters, 2001 | Boston | MI HA | 1995-1996 | Mean 62 | 59 |
| Peel, 2005 | Atlanta | URI ED visits | 1993-2000 | All | 67 |
| | | Pneumonia ED visits | | All | |
| | | COPD ED visits | | 65+ | |
| | | Asthma ED visits | | All | |
| | | RD ED visits | | All | |
| Metzger, 2004 | Atlanta | IHD ED visits | 1993-2000 | All | 67 |
| | | CHF ED visits | | | |
| Tolbert, 2007 | Atlanta | RD ED visits | 1993-2004 | All | 71 |
| | | CVD ED visits | | | |
| Slaughter, 2005 | Spokane | COPD | 1995-2001 | 65+ | 81 |
| | | Asthma | | All | |
| | | RD | | All | |
| Ito, 2003 | Detroit | RI HA | 1992-1994 | 65+ | 102 |
| | | COPD HA | | | |
| | | IHD HA | | | |
| | | CHF HA | | | |
| Sheppard, 2003 | Seattle | Asthma HA | 1987-1994 | All | 105 |




*These studies are a combination of those assessed in the last review and those assessed in the ISA in the current review. Studies are ordered by increasing 98th percentile $PM_{10}$ concentrations. See 2005 Staff Paper (US EPA, 2005, pp. 5-63 to 5-66) describing measurement uncertainties associated with the reported $PM_{10}$ levels in Ito (2003).

3-38

In a key multi-city study of mortality, (Zanobetti and Schwartz, 2009) 98th percentile $PM_{10}$ concentrations were 77 µg/$m^3$, according to an EPA analysis cited in the PA.[155] Furthermore, in six

[155] Policy Assessment, p. 2-48.

cities in this study where positive and statistically significant associations with mortality were reported, 98$^{th}$ percentile $PM_{10}$ concentrations ranged from 91 to 138 µg/m$^3$, all well below the current standard.[156]

In this scientific review, the Agency sought to distinguish between fine and coarse particles. There is a growing literature on the health effects of coarse particles (defined as $PM_{10-2.5}$) though far less information is available than for fine particles ($PM_{2.5}$) or for $PM_{10}$. In fact, the vast majority of community health studies have focused on $PM_{10}$ as the indicator pollutant, because the $PM_{2.5}$ monitoring network was not fully deployed until several years after the adoption of the 1997 NAAQS for $PM_{2.5}$.

Unfortunately, in this review, EPA did not evaluate the record on the impact of $PM_{10}$ on human health with respect to the $PM_{10}$ standard. Instead, EPA narrowed its analysis of revisions to the coarse particle standards to focus only on studies of $PM_{10-2.5}$. Yet in the Policy Assessment and in the proposal, EPA proposes to retain $PM_{10}$ as the indicator pollutant for coarse particles. This leads to a contradictory situation where EPA disregarded the results of the vast number of studies indicating associations with $PM_{10}$, and then concluded that there was not sufficient information to consider strengthening changes to the $PM_{10}$ standard.

There is a disconnect here. The most relevant studies to the setting of a $PM_{10}$ standard are the thousands of studies that have reported adverse effects associated with $PM_{10}$ pollution. Furthermore, the bulk of the coarse particle studies reviewed in the ISA reported positive associations. There is no basis in the health literature for relaxing the level or form of the $PM_{10}$ standard, and certainly no basis for rescinding the standard as advocated by some industry groups.

What is clear, is that the current $PM_{10}$ 24-hour average standard of 150 µg/m$^3$, set in 1987, is grossly outdated, totally inadequate to protect public health, and completely out of step with international standards and those established by California, a leader in air quality protection. At a minimum, our organizations support a strengthened standard of 50 µg/m$^3$, as recommended by the World Health Organization and as adopted by the State of California.

## Long-Term $PM_{10}$ Effects on Cardiovascular and Respiratory Morbidity

[156] U.S. EPA, Memorandum to PM NAAQS Review Docket from Mark Schmidt, OAR/OAQPS/AQAD and Scott Jenkins, OAR/OAQPS/HEID, "$PM_{10}$ and $PM_{10-2.5}$ Air Quality Analyses," July 22, 2010

As noted, the ISA fails to conclude an evaluation of the causal determination for $PM_{10}$, but instead focuses only on $PM_{10-25}$. The ISA concludes the effects of long-term exposure to $PM_{10-2.5}$ on mortality are merely suggestive.

EPA should re-evaluate this conclusion and consider separately a classification of $PM_{10}$ effects on mortality as likely causal, in light of evidence. A number of new studies are available since the last review that report persuasive evidence of adverse effects of long-term exposures to $PM_{10}$.

Consideration of the full range of evidence reviewed in the ISA, including a number of studies based on $PM_{10}$ measurements, suggests that $PM_{10}$ cardiovascular effects are "likely causal." However, we emphasize that EPA must set standards that protect against effects that are deemed suggestive by the evidence.

The ISA, by focusing on studies of $PM_{10-2.5}$, ignores evidence from studies of $PM_{10}$ that indicate that long-term exposures to $PM_{10}$ are "likely to be causal" for respiratory morbidity. In its 2006 revisions to the National Ambient Air Quality Standards, EPA revoked the annual average $PM_{10}$ standard of 50 $\mu g/m^3$. Several new studies support a conclusion that long-term $PM_{10}$ exposure and respiratory morbidity is likely to cause effects at levels below the prior standard of 50 $\mu g/m^3$, and call into question EPA's 2006 action.

Many of the studies discussed in the prior section on long-term fine particle health effects also report positive associations with $PM_{10}$. These include: the California Teachers Study, Nurses Health Study, the Medicare Cohort Study, and the Cystic Fibrosis study. Additionally, studies of cardiovascular effects including increased risk of hospital admission, myocardial infarction, and congestive heart failure, provide further evidence of the harms of long-term exposure to $PM_{10}$.

## Monitoring Requirements for Coarse Particles

We strongly oppose EPA's proposal to rescind the speciation requirements for $PM_{10}$ monitoring. We find no legal, practical, or scientific justification for eliminating requirements for better characterization of coarse particle concentrations. Current scientific research efforts on particle pollution health effects are exploring whether particular portions of the particulate matter stream could be responsible for the health impacts observed. The Health Effects Institute, with the support of EPA and a broad cross-section of industry groups, has been exploring this question with respect to fine particles in its NPACT study. Other studies have looked at source attribution to fine and coarse particle

fractions in relation to health effects, including several studies in the California Regional $PM_{10}/PM_{2.5}$ Air Quality Study[157] and studies in the Phoenix area.[158]

Many questions have been raised about the coarse particle fraction, and industry groups representing agricultural and mining interests have argued for exemption from $PM_{10}$standards[159] or for the elimination of the $PM_{10}$ standard in its entirety. Speciation data will be important in the future as scientists examine the sources and composition of $PM_{10}$ particles most responsible for health effects.

It is reasonable to conclude that future research and regulatory efforts may depend upon access to speciated data for coarse particles.

### *Retention of Fine Particle Monitoring Filters*

EPA is proposing to require retention of $PM_{2.5}$ filters for a period of five years, but to require cold storage for only one year. It is vitally important to collect and retain PM filters and to analyze their content. We support the archiving of particle filters in cold storage, to preserve the constituents collected on the filters.

Indeed, one of the groundbreaking studies discussed [Ghio et al] to provide toxicological evidence in support of epidemiologic findings in the Utah Valley examined the toxicity of particles extracted from archived filters.[160] The federal government and the states have made a large investment in collecting $PM_{2.5}$ and $PM_{10}$ monitoring data which is not only necessary for compliance purposes but also critical for source attribution and health effects research.

## PM NAAQS Implementation Issues

[157] Kim BM, Teffera S, Zeldin MD. Characterization of $PM_{2.5}$ and $PM_{10}$ in the South Coast Air Basin of southern California: Part 1—Spatial variations. *J Air Waste Manag Assoc*. 2000; 50(12): 2034-2044.

[158] Upadhyay N, Clements A, Fraser M, Kerckes P. Chemical speciation of $PM_{2.5}$ and$PM_{10}$ in south Phoenix, AZ, USA. *J Air Waste Manag Assoc*. 2011; 61(3)302-310.

[159] Whitfield, Rep. Ed. Opening Statement of the Honorable Ed Whitfield Subcommittee on Energy and Power Markup of H.R. 1633, The Farm Dust Regulation Prevention Act. As prepared for delivery November 3, 2011. Accessed at http://republicans.energycommerce.house.gov/Media/file/Markups/Energy/110311/Whitfield.pdf on August 29, 2012; Pompeo, Rep. Mike. Press Release: EPA must stop playing in the dust. December 8, 2011. Accessed at http://republicans.energycommerce.house.gov/News/PRArticle.aspx?NewsID=9139 on August 29, 2012.

[160] Ghio AJ, Devlin RB. Inflammatory lung injury after bronchial instillation of air pollution particles. *Am J Respir Crit Care Med* 2001; 164: 704-708.

## Designations

Commenters support EPA's proposed designation process. Commenters agree that the air quality control region boundaries should be the same for both the primary and secondary standards. *See* 77 Fed. Reg. at 39017. This reflects the fact that the geography and sources that affect compliance with these standards are likely to be the same. Commenters also agree that the timing of such designations should be the same and be completed no later than two years from the date of promulgation of these final standards. *See* 42 U.S.C. § 7407(d)(1)(B).

In preparing new designations, EPA must consider needed redesignations under the 24-hour standard, even if EPA decides to retain the current standard unchanged. To ignore evidence of violations of the 24-hour standard would be arbitrary and capricious and contrary to the health protection goals of the statute.

Designations must be accurate to avoid irrational outcomes, for example, around the proper permitting program for new and modified sources. EPA reasonably proposes to apply the nonattainment permitting programs in areas violating either the 24-hour or annual primary standards, but this approach, if the 24-hour designations are not revisited, would not guard against the situation where an area is designated attainment for both the annual and 24-hour standard even though it is violating the 24-hour standard. This would create the untenable situation wherein new sources in the area, governed by the PSD program, would be required to show that they will not cause or contribute to a violation of a NAAQS that the area is in fact already violating.

New 2009-2011 data that identify areas that don't attain current standard yet are officially in attainment.[161] For example, EPA data have these areas in violation of a 24-hour standard of 35 µg/$m^3$, even though they are 'attainment' areas:

| | |
|---|---|
| California | Inyo County |
| Idaho | Lemhi County |
| Idaho | Shoshone County |
| Montana | Lewis and Clark County |
| Montana | Silver Bow County |
| New Mexico | Doña Ana County |
| Oregon | Lake County |

[161] http://www.epa.gov/airtrends/values.html.

Those counties above currently violate the daily standard but are not designated as nonattainment.

Finally, commenters agree that designations cannot and should not be delayed pending the placement of new near-highway or visibility monitors. See 77 Fed. Reg. at 39018. As discussed further in these comments, near-highway impacts should nonetheless be determined through modeling pending such placement. EPA should also make clear that monitors used to designate an area as nonattainment may not be relocated to address the new monitoring requirements. Moreover, EPA should also clarify that any equivalent speciated monitor must be considered in designating areas under the visibility standard – not just IMPROVE or CSN monitors.

## Infrastructure SIPs

Commenters support EPA's proposal to require both the primary and secondary standard "infrastructure SIPs" at the same time within three years of the promulgation of these standards. *See* 77 Fed. Reg. at 39019. As EPA notes, there is no justification for delaying submission of the secondary standard SIP, which will largely overlap with the infrastructure SIP for the primary standards.

Commenters caution that the streamlined "certification" option may not be available to States that last updated their infrastructure SIPs before the applicability of requirements governing condensable particulate emissions and PSD permitting elements specific to $PM_{2.5}$. Thus, infrastructure SIPs adopted to address the 1997 $PM_{2.5}$ standards may no longer be sufficient to satisfy currently applicable requirements. In any future guidance on these infrastructure SIPs, EPA should identify the changes in applicable requirements that have become effective since the last round of required infrastructure SIPs.

## Implementation Scheme

### *Implementation Framework*

EPA states that it intends to develop and propose a revised implementation rule that will address any new implementation requirements as a result of the proposed standards and monitoring requirements. 77 Fed. Reg. at 39020. EPA adds that for many issues, regulatory text similar to that of existing implementation rule for the 1997 $PM_{2.5}$ standards can be included in the future implementation rule. *Id.* EPA nonetheless requests comments on implementation issues that EPA "should consider updating." EPA needs to be clear what implementation issues EPA is deciding in this

rulemaking and what issues will be decided in a future rulemaking. EPA cannot use these open ended requests for comments regarding the content of a future rulemaking to support arguments at a later date that this rulemaking represented EPA's final decision-making on a particular topic.

For example, the preamble states that: "EPA believes that the overall framework and policy approach of the implementation rule for the 1997 NAAQS provides effective and appropriate guidance on the general approach for states to follow in planning for attainment of the revised primary annual standard." 77 Fed. Reg. at 39020. Presumably the framework and policy approach for implementing these new standards will be finally determined in the future implementation rulemaking, and this statement is not prejudging that determination. To the extent EPA intends this statement to represent a conclusion on the appropriate framework, however, commenters disagree because the framework of the 1997 implementation rule does not comply with the applicable requirements of Clean Air Act Title I, Part D, Subpart 4. The framework for the 1997 $PM_{2.5}$ standards undermines Congress's clear intentions and runs counter to years of case law recognizing Congress's efforts to limit the Agency discretion that had resulted in decades of delay in cleaning up the air.

Subpart 4 governs state planning requirements to meet national ambient air quality standards for $PM_{10}$. Clean Air Act section 302(t) defines "$PM_{10}$" as "particulate matter with an aerodynamic diameter less than or equal to a nominal ten micrometers . . . ." 42 U.S.C. § 7602(t). All $PM_{2.5}$ particles fit within this definition. In other words, $PM_{2.5}$ *is* $PM_{10}$.

Congress adopted Subpart 4 to address the problem of $PM_{10}$ because that was the national ambient air quality standard in place at the time of the 1990 Amendments. *See* H.R. Rep. No. 101-490, at 207 (1990), *reprinted in* Comm. on Env't and Pub. Works, 103d Cong., *A Legislative History of the Clean Air Act Amendments of 1990* ("*Legislative History*"), at 3231 (1993) (describing history of national particulate standards); *see also Legislative History*, at 2996 (statement of Rep. Murtha recognizing that "[t]he Title I $PM_{10}$ provisions of H.R. 3030 somewhat reschedule the attainment dates that would otherwise apply under the $PM_{10}$ standards as promulgated by EPA"). In adopting these statutory requirements, however, Congress was not focused only on the coarse particles that EPA now equates with the $PM_{10}$ standards, but was explicitly seeking to address the sources and health effects associated with $PM_{2.5}$ pollution.

For example, the list of pollutants Congress intended to address through the Subpart 4 requirements for $PM_{10}$ includes both the fine and coarse particle components identified by EPA:

> Many different substances can be components of $PM_{10}$ including dust, dirt, smoke, and 'secondary particulates' . . . formed by the transformation of pollutant gases such as sulfur dioxide, nitrogen oxides, or volatile organic compounds into airborne particulates.

*Legislative History*, at 2501; *see also* H.R. Rep. No. 101-490, at 207 (1990), *reprinted in Legislative History*, at 3231 (same). It was well understood that addressing $PM_{10}$ under Subpart 4 meant addressing a wide variety of sources, many of which EPA has now identified strictly as sources of fine particulate matter. *See Legislative History* at 2501 (appended fact sheet explaining "within the broad category of man-made sources [of $PM_{10}$] there are three major subsets of sources: fugitive emissions (e.g., dust and dirt), direct emissions (e.g., diesel particulates and wood smoke), and secondary particulates (e.g., sulfates and nitrates)"); *see also id.* at 2502 (providing more detail on source types); H.R. Rep. No. 101-490, at 212 (1990), *reprinted in Legislative History*, at 3231 (same). Congress assumed all of these sources, not just the sources of coarse particulate matter, would be addressed with the Subpart 4 requirements for $PM_{10}$. Legislators understood, for example, that $PM_{10}$ sources "include[d] major industrial polluters such as steel plants and oil refineries, small area sources such as woodburning stoves, as well as fugitive dust from unpaved roads, heavy construction equipment and agricultural dust." *Legislative History*, at 1244. Indeed, the express language of Subpart 4 includes control measure requirements for coarse particulate matter sources (*e.g.*, fugitive dust) and fine particulate matter (*e.g.*, wood combustion). *See* 42 U.S.C. § 7513(b) (requiring control measure guidance for both). Finally, when discussing the health concerns associated with $PM_{10}$, the legislative history again identifies impacts associated not just with coarse particulate matter, but impacts such as premature mortality and lung cancer that EPA has isolated as being caused by $PM_{2.5}$. *See, e.g.*, *Legislative History*, at 2501 (appended fact sheet explaining "$PM_{10}$ can produce an array of adverse health effects, ranging from temporary reductions in lung capacity, to aggravation of pre-existing respiratory diseases, to cancer and premature death"); *see also* H.R. Rep. No. 101-490, at 210 (1990), *reprinted in Legislative History*, at 3231 (same).

EPA's desire to avoid the detailed requirements of Subpart 4, and revive the more "flexible" pre-1990 implementation scheme under Subpart 1, ignores the history and intent behind the 1990 Amendments. EPA's interpretation that Congress would give EPA more flexibility to address a pollution problem that is even graver than Congress assumed has repeatedly been rejected by the courts as irrational. *See, e.g., Whitman v. American Trucking Ass'ns.*, 531 U.S. 457, 481-86 (2001). The Court in *Whitman* rejected EPA's similar attempt to backtrack from the more prescriptive ozone implementation requirements of Subpart 2 holding:

> To use a few apparent gaps in Subpart 2 to render its textually explicit applicability to nonattainment areas under the new standard utterly inoperative is to go over the edge of reasonable interpretation. The EPA may not construe the statute in a way that completely nullifies textually applicable provisions meant to limit its discretion. *Id.* at 485.

For $PM_{2.5}$, there is no similar statutory "gap" in Subpart 4. Nothing in Subpart 4 fails to "fit" with the implementation of revised particulate matter standards. The provisions in Subpart 4 clearly

contemplate $PM_{10}$ nonattainment areas that come into existence in the future, and are thus given dates for various requirements that key off of some future nonattainment designation. Every provision dictating the requirements that address $PM_{10}$ pollution is just as relevant and applicable to $PM_{2.5}$ pollution because, as noted above, Congress expressly defined $PM_{10}$ to include $PM_{2.5}$ and adopted Subpart 4 to address the problems, sources, and emissions associated with $PM_{2.5}$ pollution. As with Subpart 2, EPA cannot simply split $PM_{10}$ into component standards and render obsolete the Subpart 4 requirements that Congress intended to "limit [EPA's] discretion" and govern "far into the future." *Whitman*, 531 U.S. at 485.

For all these reasons, EPA must require $PM_{2.5}$ nonattainment areas to adhere to the schedules and control requirements in Subpart 4. Those requirements include a tiered scheme of deadlines and controls that require the most polluted areas to do more in exchange for additional time to attain. Subpart 4 mandates attainment of the NAAQS as expeditiously as practicable, but no later than 6 years from designation for moderate areas, and 10 years for serious areas. 42 U.S.C. § 7513(c). It also requires implementation of reasonably available control measures within four years, and implementation of best available control measures within four years of reclassification to serious. *Id.* § 7513a. In addition, Subpart 4 mandates control of precursor emissions, establishes rate-of-progress milestones, and imposes more stringent definitions of major sources in serious nonattainment areas. *Id.* §§ 7513a(c), (b)(3) and (e). Application of these and all of the other Subpart 4 provisions will help to ensure timely $PM_{2.5}$ and precursor emission reductions within nonattainment areas, and timely attainment of the standards.

### *Other "Updates" to the Current Implementation Rules*

EPA should also acknowledge that several provisions in the current implementation rules for the 1997 standards are currently under reconsideration. These issues include the use of emission reductions from outside a nonattainment area for purposes of demonstrating reasonable further progress, the presumption that compliance with the Clean Air Interstate Rule would satisfy the requirement to install reasonably available control technology, and the criteria for analyzing economic feasibility of controls. *See* Letter from Lisa P. Jackson, Administrator, EPA, to Paul Cort, Earthjustice (April 25, 2011). These issues must be resolved in the upcoming implementation rulemaking and cannot merely be extended from the implementation rule for the 1997 standards.

Several other transition issues included in the previous implementation rule, *e.g.*, the treatment of condensable emissions and delays in state deadlines for permitting programs, are no longer justified with these new standards and should not, therefore, be copied.

Finally, several technical conclusions, *e.g.*, the presumptions regarding significant precursors, were specific to the facts and data available at the time of the previous implementation rule and cannot be carried forward for these standards without a new analysis. To extend these presumptions without a new analysis now that current $PM_{2.5}$ plans have provided better information and changed the mix of pollutants in the atmosphere would be irrational and arbitrary.

All of these "updates" must be made through notice and comment rulemaking. EPA has illegally attempted to provide similar updates for the 2006 $PM_{2.5}$ standard through guidance. Commenters support EPA's new promise that updates to references, deadlines and other requirements will be included "in a proposed implementation rule for any revised $PM_{2.5}$ NAAQS . . . ." 77 Fed. Reg. at 39020.

### *Implications of New Monitors and Transition Periods*

EPA is not free to ignore valid air quality data. EPA states that it "would not expect that data from any new near-road PM2.5 monitors would be available in time to consider during the initial area designations process, and therefore such monitoring data would not be the basis for designating a new nonattainment area at the time of initial designations." 77 Fed. Reg. at 39020. As discussed elesewhere in these comments, even in the absence of near-highway monitoring data, these near-highway levels must still be considered in designations. Moreover, EPA should clarify that if for some reason such new near-road monitoring data *is* available, it will be used in such designations. EPA offers no reason to ignore any valid data that is available at the time.

Similarly, EPA cannot reasonably justify ignoring valid data if it calls into question the assumptions or conclusions of plans for the 1997 or 2006 $PM_{2.5}$ standards. To the extent EPA is proposing a "transition period" to allow planning decisions to ignore relevant data, s*ee* 77 Fed. Reg. at 39021, there is no such authority in the Clean Air Act. To the contrary, 42 U.S.C. § 7410(l) places upon EPA an affirmative duty to ensure that SIP revisions will not interfere with any applicable requirement concerning attainment. A blanket exemption allowing agencies to ignore data no matter how relevant or significant to the plan decision is the definition of arbitrary. *See Ass'n of Irritated Residents v. EPA*, 632 F.3d 584, 593 (9th Cir. 2011) (rejecting "unlimited discretion" to ignore evidence indicating SIP may be substantially inadequate). EPA should be clear that agencies must consider available data and determine, on a case-by-case basis, whether it is relevant and significant to the planning decision being made.

## PSD

### *EPA Has No Authority to "Grandfather" Sources Out of PSD Requirements*

EPA proposes to add a grandfathering provision to the federal PSD program that would exempt certain pending PSD permit applications from having to be revised to address any new $PM_{2.5}$ NAAQS. 77 Fed. Reg. at 39023. The plain and unambiguous language of Clean Air Act section 165 does not confer EPA with any such authority. As section 165(a) provides:

> No major emitting facility on which construction is commenced after August 7, 1977, may be constructed in any area to which this part applies unless—
> …
> (3) the owner or operator of such facility demonstrates, as required pursuant to section 7410(j) of this title, that emissions from construction or operation of such facility will not cause, or contribute to, air pollution in excess of any
>
> > (A) maximum allowable increase or maximum allowable concentration for any pollutant in any area to which this part applies more than one time per year,
> > (B) national ambient air quality standard in any air quality control region, or
> > (C) any other applicable emission standard or standard of performance under this chapter

42 U.S.C. § 7475(a). The plain language of section 165 defines the applicability of these provisions based on when construction commences, not on any stage of the permit application process. *Id.* (imposing requirements on facilities "on which construction is commenced after August 7, 1977"). The only major emitting facilities that are exempted by this plain language are those for which construction commenced by August 7, 1977. *Id.* § 7475(a); *see also id.* § 7478(b). The statutory language is clear and provides no exception for the grandfathering exemptions being proposed here.

The express purposes of the PSD program include:

> (1) to protect health and welfare from any actual or potential adverse effect which may be reasonably anticipate[d] to occur from air pollution…notwithstanding attainment and maintenance of all national ambient air quality standards;
> (2) to preserve, protect, and enhance air quality in national parks…and other areas of special…value;
> (3) to insure that economic growth will occur in a manner consistent with the preservation of existing clean air resources; [and]
> (5) to assure that any decision to permit increased air pollution…is made only after careful evaluation of all the consequences of such a decision and after adequate procedural opportunities for informed public participation in the decision-making process.

42 U.S.C. § 7470. EPA's proposal – which would allow projects to be built without a demonstration that they will not cause or contribute to a violation of the new $PM_{2.5}$ standards, which are being promulgated specifically to protect public health and welfare – cannot be reconciled with any of these stated purposes. "Grandfathering" projects does not protect public health, preserve air quality, or insure economic growth is consistent with the preservation of air resources, and precludes careful decision-making and informed public participation.

When Congress adopted the PSD permitting program, it understood that certain sources might be affected by changing permit requirements. *See* H.R. Rep. No. 95-294, 95th Cong., 1st Sess., at 171 (1977) ("Safeguards against moratorium growth"). Consequently, Congress limited the applicability of these new requirements in several ways, such as exempting existing sources and requiring only "major sources of air pollution" to obtain PSD permits. *Id*.; *see* 42 U.S.C. § 7475(a). Congress also provided specific "grandfathering" relief to sources on which "construction had commenced" before the enactment of the 1977 Clean Air Act Amendments. *See* 42 U.S.C. § 7478(b) ("In the case of a facility on which construction was commenced…after June 1, 1975, and prior to August 7, 1977, the review and permitting of such facility shall be in accordance with the regulations for the prevention of significant deterioration in effect prior to August 7, 1977."). Where, as here, Congress has provided express exemptions and not others, EPA is not free to invent new authority to waive otherwise applicable statutory requirements. *See Andrus v. Glover Constr. Co.*, 446 U.S. 608, 616-17 (1980) ("Where Congress explicitly enumerates certain exceptions to a general prohibition, additional exceptions are not to be implied, in the absence of evidence of a contrary legislative intent.").

In enacting the PSD program, Congress also made the fundamental policy choices that (1) it is preferable to prevent air pollution from becoming a problem in the first place by limiting pollution created by newly constructed sources; and (2) controls should be installed when new sources are being constructed rather than as retrofits on existing sources. *See* S. Rep. No. 95-127, 95th Cong., 1st Sess., at 11 (1977) ("This legislation defines 'significant deterioration' in all clean air areas as a specified amount of additional pollution.... This definition is intended to prevent any major decline in air quality currently existing in clean air areas and will provide a margin of safety for the future."); H.R. Rep. No. 94-1175, 94th Cong., 2d Sess., at 101 (1976) (noting that "'an ounce of prevention is worth a pound of cure'" and explaining that "[p]ermitting unrestricted deterioration of air quality up to ambient standards involves trying to cure a condition after it has developed rather than using practical and currently available means to prevent or minimize the condition in the first place"); *id*. at 108 ("Common sense dictates that it is substantially less expensive to prevent air pollution problems – and health problems – before they develop than it is to abate dangerous pollution levels.... This approach will allow us to avoid future massive air pollution concentrations which endanger public health and restrict further economic growth, require expensive retrofitting of pollution control technology and

produce demands for economically and socially disruptive restrictions on the use of automobiles and on indirect sources."). EPA's proposal would actively defeat both of these policy choices.

EPA's proposal would allow projects to be built without demonstrating that they will not cause or contribute to violations of the $PM_{2.5}$ standards. If these sources are built and it is subsequently determined that violations are occurring as a result of their emissions, the States will be responsible for developing plans to control emissions to meet the standards. 42 U.S.C. §§ 7410, 7502. Such plans would require the adoption of reasonably available control technology requirements for existing major sources. 42 U.S.C. § 7502(c)(1). The result is that these same sources given a pass under the PSD program could be required to address these emissions in a much less cost-effective manner through retrofit controls. Grandfathering sources from section 165's requirements, and ignoring the foreseeable pollution problems that the PSD program is specifically designed to avoid, clearly undermines the "prevention" purpose of the PSD program and the policy choices made by Congress.

The statutory language of Clean Air Act section 165(a) is plain – a new source must demonstrate that it will not cause or contribute to a violation of any national ambient air quality standards and that it will install the best available control technology for all regulated pollutants. Unless the source can meet these criteria, it may not be built. EPA has no authority to waive these fundamental requirements.

### *EPA Cannot Leave In Place the Current de minimis PSD Screening Tools: Significant Impact Levels (SILs), and Significant Monitoring Concentrations (SMCs).*

EPA proposes to leave in place the PSD screening tools adopted with the previous PM NAAQS. *See* 77 Fed. Reg. at 39027. This decision has no rational basis. As previous commenters have pointed out, EPA (1) has no such authority to create these *de minimis* exemptions in the PSD program and (2) even if EPA could adopt such exemptions, to leave in place exemptions based on PM NAAQS promulgated at higher, less protective levels has no technical basis.

EPA has tried to justify these tools by invoking the *de minimis* doctrine. But the de minimis doctrine does not apply where Congress has expressly directed that the letter of the law applies in all circumstances. *Shays v. FEC*, 414 F.3d 76 (D.C. Cir. 2005). With respect to whether an "impact" is significant, Congress has explicitly mandated that no major facility may be constructed in an attainment area unless the facility demonstrates that emissions "will not cause or contribute" to a violation of "any" increment for "any" pollutant in "any area" covered by the PSD program. 42 U.S.C. § 7475(a)(3). It has further expressly mandated in section 166(e) a detailed demonstration of such compliance. There is therefore no room for *de minimis* exceptions in this statute. That Congress left EPA no discretion to invent new *de minimis* exceptions is further shown by the fact that: a) Congress

*did* include an express exemption in section 165(b) for sources emitting less than 50 tons per year; and b) Congress applied the permit program to only *major* sources. Thus, Congress itself decided what types of sources were too insignificant to include in the program.

Likewise, the statute is explicit that monitoring is always required, leaving no room for SMC *de minimis* exemptions. That Congress meant to foreclose EPA from inventing such exemptions is further shown by the fact that Congress *did* specify a narrow test for allowing less than a full years' monitoring, and by the fact that Congress itself defined the universe of sources too insignificant to justify regulation by applying the permit program only to major sources.

Even if use of the *de minimis* doctrine was not precluded by the statute, it would be arbitrary to invoke here. The *de minimis* doctrine applies only where regulation will yield a gain that is demonstrably trivial or zero. EPA has offered no evidence that sources with impacts below the proposed SILs or SMCs will never cause or contribute to violations of increments or NAAQS, or that the gain from regulating such sources will in fact be trivial. The *de minimis* theory is particularly inapplicable to monitoring requirements, which are by their nature meant to provide information to accurately determine the nature and degree of a source's impact on air quality. It is illegal and irrational in the extreme for EPA to assert that monitoring will yield trivial or no benefit due to the supposed insignificance of a source's air quality impact, when Congress required such monitoring precisely "for the purpose of determining whether emissions from such facility will exceed the maximum allowable increases or the maximum allowable concentration permitted" under the Act. 42 U.S.C. § 7475(e)(2).

EPA's proposal to leave the preexisting exemptions in place is particularly unreasonable given that the analysis supporting those exemptions was tied to a specific standards that EPA has now found inadequate to protect public health and welfare. Further, the notion that EPA can develop a single national number that defines "trivial" impact for all attainment areas is fundamentally flawed, given that even a very small impact can be of great significance in an area that is very close to exceeding an increment or a NAAQS.

Likewise, EPA must reassess its significant emission rate (SER) determination. The SER cannot be disconnected from the level of the NAAQS. Should EPA lower the NAAQS for PM, it must provide a rational basis for concluding that the existing SER continues to be appropriate.

## Nonattainment New Source Review

EPA proposes to apply nonattainment new source review requirements in any area that violates either the 24-hour or annual standard, and to require all areas designated nonattainment under the new

standards to submit any necessary SIP revisions to implement permitting under the new standards within 3 years of the date of designation. 77 Fed. Reg. at 39029. In the interim period between the effective date of the NAAQS and approval of the SIP revision, new and modified major sources in these nonattainment areas will be subject to the permitting requirements in 40 C.F.R. part 51, appendix S. *Id.*

Commenters agree with the proposed approach, with the one caveat noted above that EPA must also reassess whether areas are violating the 24-hour standard even if that standard remains unchanged. Otherwise, as noted above, the wrong permitting requirements could be applied leading to absurd results wherein sources under the PSD program would be required to show that they will not cause or contribute to a violation of any NAAQS even if the area is in fact in violation of the 24-hour PM NAAQS.

## Exceptional Events Timetable

EPA solicits comments on the revised schedule for exceptional events flagging and documentation. These proposed extensions seem reasonable, given that they will not delay designations with respect to any new standards promulgated in this rulemaking. There is no reason, however, to provide an extended deadline for flagging exceptional events related to the implementation of the current 1997 or 2006 PM standards. Accordingly, the proposal should only allow the extension of time for events with PM levels between the newly revised NAAQS and the current NAAQS. Under this approach, if the new 24-hour $PM_{2.5}$ NAAQS is set at 30 micrograms per cubic meters, the extended timetable would only apply to regions with violations between 30 micrograms per cubic meter and 35 micrograms per cubic meter. Events that violate the current $PM_{2.5}$ NAAQS should continue on the same schedule. Finally, the August 1, 2014 revision for supporting documentation for exceptional events in 2013 is appropriate to ensure the designation process is not delayed.

## Near-Roadway Monitoring

Commenters welcome and support EPA's proposal to monitor the concentrations of $PM_{2.5}$ that occur in near-road communities where millions of Americans are exposed to emissions from major highways, truck terminals and facilities that attract concentrations of motor vehicles. This NAAQS proposal seeks to begin for the first time the implementation of the $PM_{2.5}$ NAAQS within the zone where emissions of fine particles from existing transportation facilities that attract large concentrations of mobile sources adversely affect concentrations of $PM_{2.5}$.

We especially welcome EPA's proposal to abandon and revoke regulatory language in Part 58 that effectively prevented the implementation of the $PM_{2.5}$ NAAQS to protect populations exposed to incremental concentrations near transportation facilities. These include the current prohibitions against comparison of micro- and middle-scale monitors with the annual NAAQS, and the requirement that only "population-oriented" monitors be compared with the 24-hour NAAQS. For the reasons discussed below, we believe these policies impose limitations on the applicability of the NAAQS that are inconsistent with the text, purpose and intent of the Clean Air Act, and must be revoked.

However, commenters are concerned that EPA's proposed policies for implementation of the revised NAAQS near transportation facilities are limited to exclusive reliance on monitoring $PM_{2.5}$ concentrations near transportation facilities, and that such monitoring stations are not required to commence operations before 2017. The requirement in Appendix N that data from these monitors cannot be used for regulatory purposes until three years of data have been collected, will preclude action to protect the public in near-highway communities from NAAQS violations caused by emissions from transportation facilities until 2020. Commenters do not ask that the requirement for three-years of data to demonstrate attainment be changed or revoked, but we do ask that the statutory schedule for implementation of the NAAQS near transportation facilities not be delayed. To expeditiously attain the NAAQS in these areas, we ask that modeling data be used initially to identify areas where the NAAQS is being violated by emissions from such facilities.

EPA's exclusive reliance on monitoring data from monitors that will not produce useable data before 2020 will unlawfully delay implementation of the revised NAAQS near highways. The resulting delay is not consistent with the statutory scheme. Section 107(d)(1) of the Clean Air Act requires that nonattainment area designations be made within 2 years following the effective date of the 2012 NAAQS revision, i.e., by 2015. The Act requires that "any area that does not meet (or that contributes to ambient air quality in a nearby area that does not meet) the national primary or secondary ambient air quality standard for the pollutant" must be designated "nonattainment." Section 107(d)(1)(A)(i). Therefore EPA must include in its implementation rules and policies one or more procedures suitable for identifying areas near transportation facilities that do not meet the revised NAAQS within the two-year period allowed by the Act for making designations. The failure to develop such procedures will interfere with timely attainment of the NAAQS in these areas, and defeat the Congressional purpose underlying the enactment of a statutory attainment deadline.

Section 172(b) requires that control strategies to attain the 2012 NAAQS be submitted as revisions to each State's Implementation Plan no later than three years after final designation of nonattainment areas (2018). This submission deadline coincides with the first year when near-highway monitors are required to commence operation. Since three years of data from these near-highway monitors will not

be available when SIP revisions must be submitted, other sources of information will be needed to determine the magnitude of emissions reductions that will be necessary to attain the NAAQS near high-traffic transportation facilities. In addition, to meet the Act's requirement that SIPs must demonstrate that the control measures adopted in the plan provide for attainment near highways no later than the statutory deadline, sections 110(a)(2) and 172(c), modeling must be used to predict the ambient impact of control measures on future traffic emissions. Monitoring provides real-time data, and cannot predict future emissions or air quality.

Therefore Commenters request that EPA apply the same modeling tools adopted for the quantitative analysis of emissions from proposed transportation facilities in the Transportation Conformity Hot Spot rule to ensure that the revised NAAQS will be implemented near major transportation facilities in the next round of nonattainment area designations required by § 107(d), and in the State Implementation Plans required by § 110 and Part D of the Clean Air Act. Without the application of these, or similar, modeling tools to identify areas near transportation facilities where $PM_{2.5}$ levels violate the NAAQS, and to determine the magnitude of emissions reductions needed to attain the revised NAAQS, the current proposal to rely exclusively on monitoring that commences in 2017 cannot provide for timely attainment in all areas where emissions from major highways and other transportation facilities cause or contribute to NAAQS violations. [162]

Commenters ask EPA to revise the implementation policies related to emissions from transportation facilities to ensure that NAAQS violations linked to emissions from such facilities are identified before nonattainment designations are made so that SIPs will be required to adopt control strategies adequate to bring such areas in violation of the NAAQS into attainment by the statutory deadline for attaining the 2012 NAAQS. Commenters also ask EPA to revise its Modeling Guidance for States making attainment demonstrations to require, rather than merely recommend, supplemental high-resolution modeling to demonstrate that the control strategy submitted by the State will be adequate to provide for attainment in unmonitored local areas near large sources of direct particles where local concentrations are expected to be higher than at previously monitored locations.

The issues addressed in this transportation facility section of comments include –

- An analysis of the statutory and regulatory provisions governing NAAQS implementation;
- Background from EPA's Hot Spot rule showing that emissions of $PM_{2.5}$ from highways and other transportation facilities cause or contribute to elevated levels of $PM_{2.5}$ above regional background concentrations, and that these elevated concentrations present a significant risk of causing or contributing to NAAQS violations in the communities adjacent to such facilities;

[162] 42 U.S.C. §§ 7407(d); 7410; 7502-7509.

- A review of the health effects literature demonstrating that $PM_{2.5}$ emitted from transportation facilities has a significant impact on human health;
- A review of studies demonstrating the magnitude of the incremental contribution of emissions from highways to nearby ambient concentrations of $PM_{2.5}$;
- Discussion of policy options for obtaining information regarding the contribution of $PM_{2.5}$ emitted from transportation facilities in time to designate areas that are nonattainment because of such emissions;
- Requested changes to EPA's modeling guidance for the development of attainment demonstrations to ensure that the ambient impacts of $PM_{2.5}$ emitted from transportation facilities will be included in the control strategy for each nonattainment area to ensure that communities adjacent to such facilities will be protected from the adverse health effects that the revised NAAQS is intended to prevent.

## EPA's Proposals to Revoke Policies that Selectively Enforce the NAAQS for $PM_{2.5}$ Are Necessary to Comply with CAA Requirements for Implementation of NAAQS.

The problem that this section of the comments asks EPA to resolve is the legacy of EPA's selective enforcement of the NAAQS for $PM_{2.5}$ since 1997. EPA's past implementation procedures failed to ensure that communities adjacent to transportation facilities and other major sources of primary particles would be protected from the adverse health effects observed when ambient concentrations exceed the NAAQS. SIP development to implement the NAAQS was focused on demonstrating attainment at monitors showing NAAQS violations.[163] Guidance for the placement of $PM_{2.5}$ monitors only required regional scale monitors aimed at measuring regional air quality.

> Section 2.8.1.3.7 required core monitoring sites to represent neighborhood or larger spatial scales. States could at their initiative place additional monitors anywhere, but monitors in relatively unique microscale, localized hot spot, or unique middle-scale locations cannot be compared to the annual NAAQS, and any monitoring site must be population-oriented to be compared to either NAAQS. Part 58 App. D section 2.8.1.2.3. [164]

EPA's rationale for only requiring monitors to represent neighborhood or larger spatial scales was "because PM2.5 is a secondary pollutant, large spatial scales are relevant because monitors in such locations will reflect regional emissions trends and transport patterns."[165] The large spatial scales are dominated by secondary particles (e.g., sulfates, nitrates and ammonium) formed in the atmosphere

[163] "The modeled attainment test applies to locations with monitored data." Clean Air Fine Particle Implementation Rule, 72 Fed. Reg. 20585, 20607 (April 25, 2007).
[164] 71 Fed. Reg. 61235, 61264 (October 17, 2006).
[165] *Id.*, 61266.

from primary pollutants emitted as gasses at the source. Because these secondary particles are formed downwind from the source after the primary gasses have mixed in the atmosphere, they contribute to largely uniform concentrations across regions such as metropolitan areas.

But the effect of this large scale monitoring approach was to ignore the incremental concentrations above those associated with regionally distributed secondary particles that occur near major sources of primary ("direct") particles.[166] Monitors sited to represent neighborhood scales, the smallest required scale, were required to be located "within some extended area of the city that has relatively uniform land use with dimensions in the 0.5 to 4.0 kilometers range."[167] This meant citing the monitor in a location that would not be affected by a large local source within 0.5 kilometer (500 meters). This had the effect of excluding from the monitoring data used to determine the design value for an area the incremental impact of emissions from even the most heavily travelled highways which are rarely detected more than 500 meters away. Unlike secondary particles formed miles away from the source, primary particles are most concentrated at their point of emission and are transported relatively short distances from the source. The species of $PM_{2.5}$ associated with traffic emissions (elemental carbon (EC), black carbon (BC) and metals) have steep concentration gradients that are highest at the roadside and drop to regional background levels within 500 meters or less.[168]

To detect these incremental concentrations caused by direct particles from transportation facilities, a monitor would need to be sited within the range where the incremental contribution of emissions from traffic are likely to be greatest, i.e., 50 meters or less. Under the monitoring rules adopted in 2006, EPA recognized that to properly measure the contribution of $PM_{2.5}$ from highways, monitors should be located within 15 meters of the traffic source.

If the area is primarily affected by mobile sources and the maximum concentration area(s) is judged to be a traffic corridor or street canyon location, then the monitors should be located near roadways with the highest traffic volume and at separation distances most likely to produce the highest concentrations. For the microscale traffic corridor site, the location must be between 5 and 15 meters from the major roadway.[169]

---

[166] 40 C.F.R. §51.1000 Definitions. "Direct PM2.5 emissions means solid particles emitted directly from an air emissions source or activity, or gaseous emissions or liquid droplets from an air emissions source or activity which condense to form particulate matter at ambient temperatures. Direct PM2.5 emissions include elemental carbon, directly emitted organic carbon, directly emitted sulfate, directly emitted nitrate, and other inorganic particles (including but not limited to crustal material, metals, and sea salt)."

[167] 40 C.F.R. Part 58, Appdx D § 1.2(b)(3).

[168] Karner AA, Eisinger DS, Niemeier DA. Near Roadway Air Quality: Synthesizing the Findings from Real-World Data. *Environ Sci Technol* 2010; 44: 5334-5344.

[169] 40 C.F.R. Part 58, Appdx E, § 6.3(b).

But monitors operating at the "micro scale,"[170] which is intended to detect emissions within 100 meters of the monitor, had the effect of exempting the monitor from compliance with annual $PM_{2.5}$ NAAQS.

$PM_{2.5}$ data that are representative, not of areawide but rather, of relatively unique population-oriented microscale, or localized hot spot, or unique population-oriented middle-scale impact sites are only eligible for comparison to the 24-hour $PM_{2.5}$ NAAQS.[171]

EPA acknowledged that "population-oriented microscale areas …. may have higher concentrations than neighborhood scale sites on at least some days because they may be close to and downwind of large emission sources,"[172] but EPA justified excluding these areas from compliance with the NAAQS because "the number of people exposed to such concentrations is not large relative to the surrounding communities."[173] Monitors located within the 15 meter distance called for in EPA's siting criteria for measuring the ambient impact of $PM_{2.5}$ emitted from highways were also excluded from compliance with the 24-hour NAAQS if they were not deemed to be "population oriented."

Arguably, EPA's 2006 monitoring guidance, which directed states to design their monitoring network so that "at least one monitoring station is to be sited in a population-oriented area of expected maximum concentration," [174] did require monitoring near large sources of primary ("direct") particles, such as highways, quarries and steel mills, to detect the local ambient peaks caused by such sources. In these areas near major sources of primary particles, concentrations of $PM_{2.5}$ are likely to be the highest in a metropolitan region because the locally emitted primary particles are added to the regional concentrations associated with secondary particles. But this requirement was not implemented in practice by the States, and not enforced by EPA through its approval of State monitoring plans as required by Part 58. By not monitoring where sources of primary particles would have their greatest impact on local air quality, EPA effectively denied the residents of these areas the protections of the CAA where NAAQS violations were likely to be greatest.

The states did not remedy this omission by exercising the discretion that EPA allowed in the design of $PM_{2.5}$ monitoring networks. EPA acknowledged in its 2006 monitoring rulemaking that it was aware of "fewer than ten PM2.5 monitors that are sited in relatively unique population-oriented microscale areas, localized hot spots, or unique population-oriented middle-scale areas."[175] EDF submitted comments to EPA on the transportation conformity Hot Spot rule that included a survey of all $PM_{2.5}$

[170] 40 C.F.R. Part 58, Appdx D § 1.2(b)(1).
[171] 40 C.F.R. Part 58, § 58.30(a).
[172] 71 Fed. Reg. 61235, 61264.
[173] *Id.*
[174] 4.7.1(b)(1).
[175] 71 Fed. Reg. 61235, 61264.

monitors reported on EPA's AirData website as being in operation during 2004.[176] This analysis showed only 11 monitors located within the 15-meter distance required to monitor highway emissions. An analysis of monitors in California submitted by NRDC to EPA as a comment on the adequacy of the monitoring plans submitted by California air districts showed that no monitors in the South Coast Air Basin were located within 300 meters of major highways (100,000 AADT), and only one within 500 meters. Statewide only one monitor was located within the 100-meter microscale distance from a major highway (100,000 AADT). [177]

EPA's failure to require monitoring at these locations to measure the highest exposure to $PM_{2.5}$ in metropolitan areas has allowed the states to ignore the health of populations exposed near highways. The primary lesson learned from this policy of allowing states discretion to decide whether monitors will be located to protect near-highway communities is that a nationally uniform policy is needed to ensure that near-highway communities will be protected. We support EPA's decision to require protection for these communities.

Another lesson learned since EPA's 2006 decision to exclude so-called microscale areas close to highways from compliance with the annual NAAQS is that the communities close to highways are not Hot Spots, but rather are linear Hot Neighborhoods that extend as 300 to 500-meter corridors paralleling each side of the highway network throughout each metropolitan area. These continuous collection of neighboring communities represent large portions of the population of a metropolitan area. For example, a GIS study performed by EDF in 2008 demonstrated that 1.5 million residents of the South Coast Air Basin in California live within 300 meters of major freeways carrying an annual average of 125,000 vehicle trips daily.[178]

In EPA's Integrated Science Assessment for $PM_{2.5}$, the Agency estimates that about 45 million Americans live within the zone adjacent to major highways where $PM_{2.5}$ concentrations are affected by direct particles emitted from highways. In another study relying on census tract data to estimate national near-highway populations, Gould estimates that the population exposed near four-lane or larger highways range from 57 million within 500 meters, to 10 million within 100 meters.[179] In California, 41% of the population lives within 500 meters of a four-lane road, and 12.9% within 500

[176] *See* "COMMENTS ON PROPOSED HOT SPOT RULE FOR PM-10 AND PM2.5, Exhibit 1-- $PM_{2.5}$ Monitor Data," submitted to EPA docket No. OAR-2003-0049, January 27, 2005.
[177] Gould, Greg, "Near Roadway Emissions: Measurement, Exposure, and Monitoring," Table 7 (June 20, 2012) [submitted to EPA in support of comments by NRDC on the monitoring plans for the South Coast air district.]
[178] Map Showing Highways in the South Coast AQM District with more than 125,000 AADT, and memorandum explaining methodology for estimating population within 300 meters.
[179] Gould, Gregory M., "A Census of the U.S. Near Roadway Population: Particulate Matter Exposure, Environmental Justice, and Coverage of the Air Quality Monitoring Network," Fig. 1. [This report has been submitted for publication, and is submitted to EPA under a claim of copyright with a request that it not be disclosed in the docket pending publication.]

meters of major highways carrying 125,000 trips/day, which EPA determined in the conformity Hot Spot rule are presumptively "projects of air quality concern."[180]

Thus a large portion of the U.S. population is at risk from exposure to the incremental concentrations of $PM_{2.5}$ emitted from highways. As discussed in more detail below, monitoring and modeling data suggest that these incremental exposures range from 1.5 to as much as 5 $\mu g/m^3$ above urban scale background concentrations. EPA estimated in 2007 that in a moderate sized city, reducing the annual concentration of PM2.5 from 15.5 to 15.0 μg/m3 will "result in as many as 25-50 fewer mortalities per year due to air pollution exposure."[181] Assuming a moderate sized city is roughly 500,000 people, then the incremental $PM_{2.5}$ exposures experienced by the millions living within 500 meters of highways throughout the U.S. would cause thousands of additional, preventable deaths annually. The CAA demands that these deaths be prevented. EPA must adopt regulations and policies to ensure that the NAAQS is implemented near highways in order to provide the full measure of protection that the public residing in these near-highway communities are entitled to under the Act.

## The Clean Air Act Requires Implementation Plans that Attain the NAAQS in All Geographic Areas of the State.

Congress requires that "Each State shall have the primary responsibility for assuring air quality within the entire geographic area comprising such State by submitting an implementation plan for such State which will specify the manner in which national primary and secondary ambient air quality standards will be achieved and maintained…." 42 U.S.C. § 7407(a). This mandate requires that areas of the state that violate the NAAQS because of emissions from transportation facilities must be identified, and control measures sufficient to provide for attainment in these areas must be implemented as part of the State Implementation Plan.

This congressional directive that the NAAQS must be achieved and maintained "within the entire geographic area" of each State is affirmed in the directive in the requirement that State implementation plans must "provide[] for implementation, maintenance, and enforcement of such primary standard in each air quality control region (or portion thereof) within such State."[182]

[180] Gould, Greg, "Near Roadway Emissions: Measurement, Exposure, and Monitoring," Table 5 (June 20, 2012).
[181] Clean Air Fine Particle Implementation Rule, 72 Fed. Reg. 20589 (Apr. 25, 2007).
[182] 42 U.S.C. § 7410(a)(1).

The conformity requirement reaffirms this directive by prohibiting federal support, approval or funding for projects that will not conform to a State Implementation Plan.

Conformity to an implementation plan means—

> (A) conformity to an implementation plan's purpose of eliminating or reducing the severity and number of violations of the national ambient air quality standards and achieving expeditious attainment of such standards; and
>
> (B) that such activities will not –
>
> > (i) cause or contribute to any new violation of any standard in any area;
> >
> > (ii) increase the frequency or severity of any existing violation of any standard in any area; or
> >
> > (iii) delay timely attainment of any standard or any required interim emission reductions or other milestone in any area.[183]

In adopting the transportation conformity Hot Spot rule for $PM_{2.5}$, EPA interpreted this mandate to require an emissions analysis for transportation "projects of air quality concern." EPA defined such projects as including "highway projects that have a significant number of or significant increase in diesel vehicles."[184] This criteria does not adopt an explicit numeric test for determining how many diesel vehicles are "significant." EPA provided an example of a highway with an AADT of 125,000 and 8% diesel trucks (i.e., 10,000 trucks/day) as presumptively a project of air quality concern,[185] but did not prescribe highway segments with less than this level of traffic as presumptively conforming. Rather EPA explained that the determination of significance is dependent on the air quality context to which the transportation facility contributes emissions. In the final Hot Spot rule EPA abandoned its proposal to retain the 1993 rule which only required Hot Spot analyses for projects located near monitors showing violations. Instead, EPA substituted this "significance" criteria for identifying projects that must be reviewed for their localized impact on $PM_{2.5}$. EPA explained this result by noting that projects needed to be analyzed in areas where no monitoring data exist, and also areas where monitors show no violations but where project emissions might cause violations.

> While the air quality circumstances at a project's location are an important modeling consideration, these previous regulatory criteria are insufficient to ensure that all projects of air quality concern are analyzed before they receive federal funding or approval. The final rule's criteria will ensure that all projects that have the potential to impact a local air quality violation will be analyzed.[186]

[183] 42 U.S.C. § 7506(c)(1).
[184] 40 C.F.R. § 93.123(b)(1)(i).
[185] 71 Fed. Reg. 12467, 12491 (March 10, 2006).
[186] 71 Fed. Reg. 12467, 12492.

Commenters believe that this comprehensive approach toward identifying transportation facilities that "have the potential to impact a local air quality violation" is a correct application of the statutory mandate that NAAQS be implemented in all geographic areas of a State, including all areas near transportation facilities. We therefore ask EPA to adopt both monitoring requirements and other implementation policies that will be adequate to ensure that all transportation facilities that "have the potential to impact a local air quality violation" will be reviewed and evaluated both for 1) the purpose of determining whether an area that would be attainment based on a regional, urban or neighborhood scale monitor should nonetheless be designated nonattainment because of likely violations associated with vehicle emissions, and 2) the purpose of determining whether a control strategy designed to attain the $PM_{2.5}$ NAAQS at a regional, urban or neighborhood scale monitor will be adequate to provide for attainment in the localized areas affected by direct particles emitted from transportation facilities and significant stationary sources. Below we recommend both monitoring and modeling criteria to achieve this result.

## EPA's Hot Spot Rule Findings Demonstrate the Need to Protect Against NAAQS Violations Linked to Emissions from Transportation Facilities.

EPA first formally acknowledged in the transportation conformity Hot Spot rule[187] that $PM_{2.5}$ emitted from highways causes or contributes to significant concentrations of $PM_{2.5}$ elevated above background levels, and that review of new or expanded transportation projects is necessary to determine whether the proposed transportation facility will increase the severity or frequency of violations of the NAAQS for $PM_{2.5}$ or delay its timely attainment. Commenters believe that these findings in the Hot Spot rule derived from studies of emissions profiles from existing facilities compel the adoption of policies designed to identify existing facilities that emit direct particles in sufficient quantities to cause NAAQS violations when added to urban background levels of $PM_{2.5}$, and will delay or prevent timely attainment if emissions in the vicinity of these facilities are not reduced.

In its original proposal to implement conformity requirements for $PM_{2.5}$, EPA proposed not to require a hot spot analysis for transportation projects because it believed that $PM_{2.5}$ was primarily a regional pollutant dominated by secondary particles. However, after reviewing evidence submitted by commenters and other studies of emissions from transportation facilities, EPA concluded that –

> $PM_{2.5}$ is both a regional and a localized air quality concern in certain circumstances. While it is true that secondary formation from $PM_{2.5}$ precursors is a critical component to the regional

[187] 71 F.R. 12467.

> $PM_{2.5}$ air quality problem, directly emitted $PM_{2.5}$ from certain local sources has the potential to cause or contribute to elevated localized $PM_{2.5}$ concentrations. Such elevated concentrations which exceed applicable standards can have an effect on local communities and populations that the $PM_{2.5}$ standards were designed to protect.[188]

This critical conclusion was based on studies of emissions from existing transportation facilities. The fact that emissions from such existing facilities cause elevated concentrations which exceed the NAAQS establishes the fundamental predicate for Commenters' request that EPA adopt rules and policies adequate to identify the areas where such NAAQS violations are occurring, and to require that they be eliminated by the development of control strategies adequate to provide for attainment.

EPA summarized its key findings from the studies that convinced it to adopt a Hot Spot program for $PM_{2.5.}$

Overall, major conclusions from these studies are:

- Black/elemental carbon (BC or EC) mass concentrations and particle number (e.g., ``ultrafines'') concentrations are consistently associated with proximity to traffic (generally within 150 meters).
- PM2.5 is associated with proximity to traffic in most, but not all cases.
- Both regional background and local sources contribute to site-specific $PM_{2.5}$ concentrations.
- The ``near-roadway increment'' of $PM_{2.5}$ tends to be comprised of approximately 50-80% black or elemental carbon (indicating mobile sources are a key source).[189]

A study that convinced EPA to focus on diesel vehicles identified elemental carbon as the critical component of $PM_{2.5}$ that required regulation. (Cyrys et al., 2003) showed that the difference in long-term average $PM_{2.5}$ mass between traffic sites and background sites was equal to the difference in elemental carbon mass between the two types of sites. Elemental carbon predominantly comes from diesel exhaust, as demonstrated in several source apportionment studies.[190]

EPA found that another study demonstrated that "Trucks were estimated to contribute between 5.0-14.2 [mμ]g/$m^3$ $PM_{2.5}$, depending on the level of truck traffic."[191]

[188] 71 F.R. 12467, 12473.
[189] 71 F.R. 12467, 12472-73.
[190] 71 F.R. 12467, 12492.
[191] 71 F.R. 12467, 12473.

Other studies have become available since EPA's 2006 Hot Spot rulemaking that confirm many of these findings. E.H. Pechan reviewed a number of these for EDF in 2009.[192] Pechan concluded that "[t]hese data demonstrate that emissions from on-road vehicles cause a significant increment in ambient $PM_{2.5}$ concentrations near highways that are not accounted for in areas where air quality monitors have not been sited in the impact zone near highways." The data reviewed by Pechan "demonstrate that highway emissions add from 1 to 17 $\mu g/m^3$ to regional monitored concentrations, depending on the traffic load (AADT), the share of daily traffic contributed by diesel trucks, the distance from the highway, and local factors affecting dispersion from the highway into the atmosphere."[193]

In a dispersion modeling analysis of $PM_{2.5}$ emissions from the highway network in Los Angeles County, Gould found that peak hourly emissions from highways could exceed 36 $\mu g/m^3$ under certain conditions, and average daily emissions could exceed 5 $\mu g/m^3$.[194] This analysis provides more temporally detailed estimates of $PM_{2.5}$ emissions, but the average daily increment of 2-5 $\mu g/m^3$ is similar to the estimate provided by Pechan.

HEI and Karner reviewed studies that show the gradient for $PM_{2.5}$ from the roadside to background sites is smaller than other pollutants such as CO and $NO_2$, but also show that the gradient for elemental carbon (EC) is as large as for other tailpipe pollutants.[195] The gradient for total $PM_{2.5}$ must be smaller than for other pollutants because species contributing to total $PM_{2.5}$ concentrations other that EC are not emitted in noticeable amounts from motor vehicles and will be expected to have more uniform concentrations between the roadside and regional background monitors. However, the increment contributed by EC emitted from vehicles to the near-highway air shed can be the difference between $PM_{2.5}$ attainment and nonattainment. Until such time as EPA adopts a separate NAAQS for EC/BC, the only regulatory approach available to address the incremental exposures caused by EC in the near-highway airshed is to monitor and regulate $PM_{2.5.}$

Unless EPA disowns the findings it made in the Hot Spot rule regarding the contribution that $PM_{2.5}$ emissions from transportation sources make to NAAQS violations, commenters believe that those findings supported by additional studies completed since 2006 compel EPA to adopt requirements designed to identify and remedy NAAQS violations caused by emissions from these sources.

[192] E.H. Pechan, ESTIMATING CONTRIBUTIONS OF ON-ROAD EMISSIONS TO NEAR HIGHWAY $PM_{2.5}$ CONCENTRATIONS (April, 2009).
[193] *Id.*, 20.
[194] Gould, Gregory M., "Regional Scale Dispersion Modeling and Analysis of Directly Emitted Fine Particulate Matter from Highway Vehicles Using AERMOD," 10 (2012).
[195] Karner, "Near-Roadway Air Quality: Synthesizing the Findings from Real-World Data," Env. Sci. & Tech. (2010).

## Health Studies of Traffic Emissions Provide Independent Basis for Attaining the NAAQS Near Highways.

Commenters share EPA's concern that fine particles emitted from highways present significant health risks that must be reduced to achieve the health protection goals underlying the promulgation of NAAQS. These health risks flow from both the dangerous $PM_{2.5}$ species emitted from transportation facilities, and the elevated exposures that occur near highways and other transportation facilities. To prevent or avoid these significant health risks EPA must require States to develop control strategies that will ensure compliance with the revised NAAQS for $PM_{2.5}$.

The EPA has recognized the risk of highway pollution on the surrounding population and environment in:

- The Agency's explanation of the joint research project with the University of Michigan looking at the health effects of roadway pollution: "With more than 45 million Americans living less than 300 feet from a highway, there is growing concern about the health impacts of living near heavily traveled roads."[196]
- EPA's overview of research into near-roadway exposures: "Exposure to near-roadway pollution may increase a person's chances of developing a wide range of health problems, including asthma, hypertension, leukemia, lung cancer, and perhaps even premature mortality."[197]
- the *Charge for Developing Recommendations to Address the Air Quality Impacts of Goods Movement on Communities*, in which EPA acknowledges the following:

> The environmental, public health, and quality of life impacts of goods movement on communities are more pronounced in areas with major transportation hubs and heavily trafficked roads. "Near roadway hotspots" – localized areas with elevated levels of air pollution – is an issue of longstanding concern to EPA and other environmental health agencies. This issue also is a matter of increasing concern to government transportation and planning agencies. Research shows that the many communities, including minority and/or low income communities, living near these transportation hubs and

[196] U.S. EPA, Science in Action: External EPA Fact Sheet. Health Effects of Roadway Pollution, Office of Research and Development. July 2010, Accessed at http://www.epa.gov/nerl/download_files/documents/NearRoadwayTechnical_external_fact_sheet_071910.pdf on August 25, 2012.

[197] U.S. EPA, Along the Road, Research & Development Science Features, February 10, 2011. Accessed at http://www.epa.gov/research/gems/scinews_near-road.htm on August 25, 2012.

thoroughfares, already bear disproportionate environmental impacts because of their close proximity to multiple pollution sources.[198]

- The proposed rule for the nitrogen dioxide NAAQS, which cites the number of people who live near roadways as part of the rationale for proposing near-roadway monitoring of that pollutant. Citing the 2006 American Housing Survey, EPA described that significant increase in the number of households living with 300 feet of highways, railroads or airports, totaling 15.6% of housing units and almost 50 million people.[199].

## Exposure to Near Roadway Pollution Linked to Significant Health Risks.

Strong, repeated reviews of the research shows that $PM_{2.5}$ and the PM species that comprise the dominant fractions of highway emissions (EC/BC and UFP) cause serious health impacts.[200] A growing body of evidence shows that people living near busy roadways where exposures to $PM_{2.5}$ are elevated above regional concentrations face significantly elevated health risks as well. We summarize here some of the studies that provide evidence that the adverse effects associated with exposure to fine particles from highways is as great, if not greater, than the effects linked to a broader mix of particles measured at the regional scale. The Health Effects Institute confirmed this with its expert panel review of the literature on traffic-related air pollution and health impacts in 2010.[201]

*1) Correlation Between Asthma and Attending School Near a Major Roadway.*

Kim et al. (2004) surveying over 1,000 elementary school students in Northern California found higher rates of asthma and bronchitis symptoms in children attending schools near busy roads and freeways.[202] This study evaluated nearby traffic sources and average pollutant concentrations at ten schools. BC concentrations were found to be between 0.7 and 0.8 $\mu g/m^3$ at schools without nearby major traffic sources. Schools within 60 to 360 meters of major traffic sources (90,000 to 210,000 annual average daily traffic) were found to have BC concentrations between 0.8 and 1.1 $\mu g/m^3$. Notably, BC concentrations are above the study average (0.8 $\mu g/m^3$) only for those schools that are near and downwind of heavily traveled freeways. The highest BC concentration, 1.1 $\mu g/m^3$, was 38% higher than the study average for all school sites. Equally important is the finding that the school with the highest BC concentration also measured the highest $PM_{2.5}$ concentration (15 $\mu g/m^3$) which was

[198] EPA, Charge for Developing Recommendations to Address the Air Quality Impacts of Goods Movement on Communities, attached as Exhibit A.
[199] 74 Fed. Reg. 34419
[200] U.S. EPA. ISA, 2009. Chapter 2, Integrative Health and Welfare Effects Overview.
[201] Health Effects Institute, Panel on the Health Effects of Traffic-Related Air Pollution. 2010. Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects. HEI Special Report 17. Health Effects Institute, Boston, MA.
[202] Kim, J. et al., Traffic-related air pollution and respiratory health: East Bay Children's Respiratory Health Study, *American Journal of Respiratory and Critical Care Medicine* 2004; 170: 520-526.

25% above the study average (12 μg/$m^3$). These levels were measured at the school closest (60 meters) to a major freeway. This study demonstrates a link between proximity to highway emissions, increased exposure to both $PM_{2.5}$ and highway-related PM species, and adverse health outcomes.

McConnell et al. (2010) studied thirteen southern California communities where children exposed to traffic-related pollution in school were more likely to develop new onset asthma, irrespective of residential exposure.[203] A study of almost 1,500 children in Dutch schools found a positive relationship between school proximity to freeways and asthma occurrence. Truck traffic intensity and pollutant levels measured in schools were significantly associated with chronic respiratory symptoms.[204]

A recent nationwide study of almost 9,000 U.S. public schools noted that children spend a significant amount of time at school, making exposure to pollution at school an important consideration; the study found that approximately one third of students were likely to be at an increased risk of acute and chronic respiratory disorders due to close proximity of their school to a freeway.[205] *Correlation Between Respiratory Health Problems and Living Near a Major Roadway.*

Proximity of residences to heavy traffic levels has been associated with respiratory impacts such as cough, wheeze, persistent cough, asthma and hospital admissions for asthma in many studies.[206] The California Children's Health Study, which began in 1992, found an 89 percent increase in the likelihood of being diagnosed with asthma for those children living close to freeways versus those living farther away.[207] This study was confirmed by a separate Southern California study finding an 85% higher likelihood for an asthma diagnosis among children living with 75 meters of a major road..[208] Another report from the Children's Health Study showed adverse health impacts of local traffic exposure on children independent of regional air quality, including decreased lung function that is unlikely to be regained and thus predisposes those individuals to cardiovascular illness later in life.[209] A recent

[203] McConnell, R. et al., Childhood Incident Asthma and Traffic-Related Air Pollution at Home and School, *Environmental Health Perspectives* 2010; 118(7): 1021-1026.
[204] Speizer, F. E. and B. G. Ferris, Jr., Exposure to automobile exhaust. I. Prevalence of respiratory symptoms and disease, *Archives of Environmental Health* 1973; 26(6):313-8; Van Vliet, P.et al., Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways, *Environmental Research* 1997; 74(2): 122-32.
[205] Appatova, A.S. et al., Proximal exposure of public schools and students to major roadways: a nationwide US survey, *Journal of Environmental Planning and Management* 2008; 51(5): 631-646.
[206] Nicolai, T. et al., Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children, *Eur Respir J.* 2003;21:956–963; Brunekreef, B. et al., Air pollution from truck traffic and lung function in children living near motor-ways, *Epidemiology* 1997; 8(3): 298-303; Duhme, H. et al., The association between self-reported symptoms of asthma and allergic rhinitis and self-reported traffic density on street of residence in adolescents, *Epidemiology* 1996; 7(6): 578 -582; Edwards, J. et al., Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom, *Archives of Environmental Health* 1994; 49(4): 223-227.
[207] Gauderman, W.J. et al., Childhood Asthma and Exposure to Traffic and Nitrogen Dioxide. *Epidemiology* 2005; 16: 737-743;
[208] McConnell R, et al., Traffic, susceptibility, and childhood, *Environ Health Perspectives* 2006; 114(5):766-772.
[209] Gauderman, W.J., et al., Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study, *Lancet* 2007; 369(19561): 571-7.

review of California Health Interview Survey (CHIS) data revealed a three-fold increase in asthma related hospital visits among children living in high traffic density areas.[210] A similar study based on CHIS data attributes a 92 percent increase in asthma symptoms among those living near the highest traffic densities, and suggests that impacts may be disproportionately worse among those in poverty due to heightened vulnerability.[211] Those in poverty may also be disproportionately exposed to pollution due to older and poorer quality housing. A study in Washington State found that older homes, smaller homes, and homes with fewer renovations were more likely to have a higher infiltration fraction of $PM_{2.5}$.[212]

Narrowing the distance increases the exposure, and the severity of health effects. A study of nearly 10,000 children in England found that wheezing illness, including asthma, was more likely with increasing proximity of a child's home to main roads, with the greatest risk being for children living within 90 meters of the road.[213] A study in rural New York found that children living in neighborhoods with heavy truck traffic within 200 meters of their homes had increased risks of asthma hospitalization.[214] A Dutch study of over 1,000 children found that asthma, wheeze, cough, and runny nose were significantly more common in children living within 100 meters of freeways; and that increasing density of truck traffic was associated with significantly higher asthma levels.[215] Another Dutch study found that traffic-related pollution was associated with increased respiratory infections, as well as some measures of asthma and allergies among four year olds studied from birth.[216] Finally, a landmark study in London demonstrated a strong association between traffic exposure and lung function among asthmatics, finding a marked decline in lung function among subjects during or immediately following a walk along the busy Oxford street compared with observed lung function

[210] Wilhelm et. al., Environmental Public Health Tracking of Childhood Asthma Using California Health Interview Survey, Traffic, and Outdoor Air Pollution Data, *Environmental Health Perspectives* 2008; 116(8): 1254-1260.
[211] Meng et. al., Are Frequent Asthma Symptoms Among Low-Income Individuals Related to Heavy Traffic Near Homes, Vulnerabilities, or Both?, AEP 2008; 18(5):343-350.
[212] Hystad, P.U. et al., Modeling Residential Fine Particulate Matter Infiltration for Exposure Assessment, *Journal of Exposure Science and Environmental Epidemiology* 2009; 19:570-579.
[213] Venn et al., Living Near A Main Road and the Risk of Wheezing Illness in Children, *American Journal of Respiratory and Critical Care Medicine* 2001; 164:2177-2180.
[214] Lin, et al., Childhood Asthma Hospitalization and Residential Exposure to State Route Traffic, *Environmental Research, Section A* 2002; 88:73-81;similarly, a San Diego study found increased medical visits in children living within 550 feet of heavy traffic. English P., et al., Examining Associations Between Childhood Asthma and Traffic Flow Using a Geographic Information System, *Environmental Health Perspectives* 1999; 107(9):761-767.
[215] Van Vliet et al., Motor exhaust and chronic respiratory symptoms in children living near freeways, *Environmental Research* 1997; 74:12-132.
[216] Brauer, M. et al., Air pollution and development of asthma, allergy and infections in a birth cohort, *Eur Respir J* 2007; 29:879-888.

following a walk through London's Hyde Park. $PM_{2.5}$ levels on Oxford Street were more than twice that of Hyde Park.[217]

Looking at these studies and others, the Health Effects Institute Panel concluded that the evidence was "sufficient' to infer a causal association between traffic exposures and exacerbations of asthma." The Panel also found that strong evidence for a causal relationship to incidence of asthma in children, but "in a gray zone between 'sufficient' and 'suggestive but not sufficient'."[218]

Traffic related pollution costs municipal governments significant sums of money in medical treatment. One recent study found that traffic related asthma costs the cities of Long Beach and Riverside, California at least $18 million per year in direct and indirect health care costs.[219]

### *Cancer and Living Near a Roadway.*

The "Multiple Air Toxics Exposure Study-III," a comprehensive study of toxic air pollution in the five-county Los Angeles air basin shows that motor vehicles and other mobile sources of air pollution are the predominant source of cancer-causing air pollution, accounting for roughly 94% of the cancer risk from toxic air pollution, most of which is from diesel exhaust (84% of the cancer risk).[220]

CARB estimates an increased cancer risk of 100 in one million within 90 meters downwind of freeways carrying 10,000 trucks per day.[221] A study in Denver showed that children living within 250 yards of streets or highways with 20,000 vehicles per day are six times more likely to develop all types of cancer and eight times more likely to contract leukemia.[222] A Danish study of several thousand children concluded that a doubling of vehicle pollution increased the risk of lymphomas by 25 percent.[223] An earlier English study found a cancer corridor within three miles of highways, airports, power plants, and other major polluters, showing greater risk of leukemia or other cancers within a few hundred yards from highways or other major pollution sources and decreasing risk of cancer with distance from these roadways and facilities.[224]

---

[217] McCreanor, J. et al., Respiratory Effects of Exposure to Diesel Traffic in Persons with Asthma, N Engl J med 2007; 357:2348-58.
[218] HEI, 2010.
[219] Brandt, S.J. et. al., Costs of childhood asthma due to traffic-related pollution in two California communities. European Respiratory Journal, 2012; DOI: 10.1183/09031936.00157811.
[220] South Coast Air Quality Management District. Multiple Air Toxics Exposure Study-III. September 2008, available at http://www.aqmd.gov/prdas/matesIII/MATESIIIFinalReportSept2008.html.
[221] CARB, 2005.
[222] Pearson et al., Distance-weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers, *Journal of Air and Waste Management Association* 2000; 50:175-180.
[223] Raaschou-Nielsen, O.et al., Air Pollution from traffic at the residence of children with cancer, *Am J Epidemiology* 2001; 153:433-443.
[224] Knox and Gilman, Hazard proximities of childhood cancers in Great Britain from 1953-1980, *Journal of Epidemiology and Community Health* 1997; 51:151-159.

### *Reproductive Impacts and Exposure to Motor Vehicle Pollutants.*

Pre- and post-natal impacts on infants born to mothers with heavy traffic exposure have also been well documented. A Los Angeles study found that pregnant women with high traffic exposure were three times as likely to have a child with certain heart defects as women breathing the cleanest air.[225] A study of California children found an increased risk of autism among children who lived within 300 meters of a freeway during the third trimester and shortly after birth.[226]

### *Cognitive Impairment and Living in High Traffic Areas.*

Studies have shown links between living in high traffic areas and behavioral and learning problems and other impacts on the brain. Cognitive impairment has been associated with proximity to vehicular traffic, most notably among school children.[227] Lower IQs were found in children with prenatal exposure to vehicle-derived polycyclic aromatic hydrocarbons (PAHs) in one long term study.[228] Rodent models also show memory impairment after exposure to vehicle-derived air pollutants.[229]

### *Proximity to Busy Roadways and Other Health Impacts.*

A wide body of research also confirms other adverse health outcomes related to close proximity to busy roadways. Dutch researchers evaluating long term exposure to traffic have found that people who lived near a main road were almost twice as likely to die from heart or lung disease and 1.4 times as likely to die from any cause compared with those who lived in less-trafficked areas.[230] A Canadian study of 5,000 people showed that those living within 50 meters of a major road or within 100 meters of a highway had increased risks of mortality, with an "aging effect" (i.e. years of life lost) of roughly 2.5 years, which is similar to the "aging effect" of having chronic heart disease (3.1 year Rate of Advancement for mortality).[231] Another Canadian study found that people residing within 150 meters of a highway or within 50 meters of a major road were more likely to die of coronary heart diseases. Furthermore, subjects who moved away from a road during the study period showed a decreased risk

---

[225] Ritz B. et al., Ambient air pollution and risk of birth defects in Southern California. *Am J Epidemiology* 2002; 155:17-25.
[226] Volk, H., Residential Proximity to Freeways and Autism in the CHARGE Study. *Environmental Health Perspectives* 2010, doi: 10.1289/ehp.1002835, available at http://dx.doi.org.
[227] Ranft U, Schikowski T, Sugiri D, Krutmann J, Kramer U.. 2009. Long-term exposure to traffic-related particulate matter impairs cognitive function in the elderly. Environ Res 109:1004–1011.
Suglia SF, Gryparis A, Wright RO, Schwartz J, Wright RJ. 2008. Association of black carbon with cognition among children in a prospective birth cohort study. Am J Epidemiol 167:280–286.
[228] Perera FP, Li Z, Whyatt R, Hoepner L, Wang S, Camann D, et al. 2009. Prenatal airborne polycyclic aromatic hydrocarbon exposure and child IQ at age 5 years. Pediatrics 124:e195–e202.doi:10.1542/peds.2008-3506.
[229] Zanchi AC, Fagundes LS, Barbosa F Jr, Bernardi R, Rhoden CR, Saldiva PH, et al. 2010. Pre and post-natal exposure to ambient level of air pollution impairs memory of rats: the role of oxidative stress. Inhal Toxicol 22:910–918.
[230] Hoek, et al., Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study, *Lancet* 2002; 360(9341):1203-9.
[231] Finkelstein et al., Traffic Air Pollution and Mortality Rate Advancement Periods, *Am J Epidemiology* 2004; 160:173-177.

of death from coronary heart disease while those who moved closer to a road were more likely to die of coronary heart disease.[232] Long-term exposure to traffic-related air pollution has also been shown in recent research to contribute to the development of diabetes.[233] Finally, a recent EPA funded study found that of 3,500 people across the U.S., those living within 100 meters of a major roadway experienced a 27% higher mortality rate.[234]

The summary of health impacts above is strongly supported by much of the information in the EPA's Integrated Science Assessment for Particulate Matter. Most noteworthy are the following excerpts linking $PM_{2.5}$ from traffic with serious health impacts:[235]

- Several panel epidemiologic studies have examined the association between PM sources and physiological alterations in cardiovascular function. Lanki et al. (2006) reported positive associations between $PM_{2.5}$ from local traffic and long-range transported $PM_{2.5}$ with ST-segment depression in elderly adults in a study conducted in Helsinki, Finland.[236]
- Yue et al. (2007) found that adult males with coronary artery disease in Erfurt, Germany, demonstrated changes in repolarization parameters associated with traffic-related $PM_{2.5}$, with increased vWF linked to traffic and combustion-generated particles, although the source apportionment was based solely on particle size distribution.[237]
- Positive associations with $PM_{2.5}$ motor vehicle and road dust sources were reported for respiratory symptoms and inhaler use in asthmatic children in New Haven, CT (Gent et al., 2009).[238]
- Cardiovascular mortality in Phoenix (Mar et al., 2000; 2006) [239]and Santiago, Chile, (Cakmak et al., 2009) [240]was associated with $PM_{2.5}$ from traffic.

---

[232] Gan, W. Q., Changes in Residential Proximity to Road Traffic and the Risk of Death from Coronary Heart Disease, *Epidemiology* 2010; 21(5): 642-649.

[233] Anderson, Z.J. et. al, Diabetes Incidence and Long-Term Exposure to Air Pollution, A cohort study, Diabetes Care November 10, 2011; 10.2337/dc11-1155. http://care.diabetesjournals.org/content/early/2011/11/03/dc11-1155.abstract.

[234] Rosenbloom, J.I. et al., Residential Proximity to Major Roadway and 10-Year All-Cause Mortality After Myocardial Infarction, *Circulation* 2012; 125: 2197-2203.

[235] EPA, Integrated Science Assessment for Particulate Matter, Office of Research and Development, December 2009. Sections 6.6.2.1, 6.6.3, and 7.3.7.1.

[236] Lanki T; Hoek G; Timonen K; Peters A; Tiittanen P; Vanninen E; Pekkanen J (2008). Hourly variation in fine particle exposure is associated with transiently increased risk of ST segment depression. *Br Med J*, 65: 782-786.

[237] Yue W; Schneider A; Stolzel M; Ruckerl R; Cyrys J; Pan X; Zareba W; Koenig W; Wichmann HE; Peters A (2007). Ambient source-specific particles are associated with prolonged repolarization and increased levels of inflammation in male coronary artery disease patients. *Mutat Res Fund Mol Mech Mutagen*, 621: 50-60.

[238] Gent JF; Koutrakis P; Belanger K; Triche E; Holford TR; Bracken MB; Leaderer BP (2009). Symptoms and medication use in children with asthma and traffic-related sources of fine particle pollution. *Environ Health Perspect*, 117: 1168-1174.

[239] Mar TF; Norris GA; Koenig JQ; Larson TV (2000). Associations between air pollution and mortality in Phoenix, 1995-1997. *Environ Health Perspect*, 108: 347-353 and Mar TF; Ito K; Koenig JQ; Larson TV; Eatough DJ; Henry RC; Kim E; Laden F; Lall R; Neas L; Stolzel M; Paatero P; Hopke PK; Thurston GD (2006). PM source apportionment and health effects: 3 Investigation of inter-method variations in associations between estimated source contributions of $PM_{2.5}$ and daily mortality in Phoenix, AZ. *J Expo Sci Environ Epidemiol*, 16: 311-320

- Gasoline and diesel sources were associated with ED visits in Atlanta for cardiovascular disease at (Sarnat et al., 2008).[241]
- Because traffic-related pollutants such as UFPs are high near major roadways and then decay exponentially over a short distance, Williams, et al. (2009) assessed exposure according to residential proximity to major roads in a Seattle area study of postmenopausal women. Proximity to major roads was associated with a 21% decrease in natural killer cell function, which is an important defense against viral infection and tumors. This finding was limited to women who reported exercising near traffic.[242]
- In the Puget Sound region of Washington, Karr et al. (2009) reported that there may be a modest increased risk of bronchiolitis related to $PM_{2.5}$ exposure for infants born just before the peak respiratory syncytial virus (RSV) season. Risk estimates were stronger when restricted to cases specifically attributed to RSV and for infants residing closer to highways. Emerging evidence suggests that respiratory infections, particularly infection by viruses such as RSV, can cause asthma or trigger asthma attacks.[243]
- Table 6-18 in the ISA lists study-specific $PM_{2.5}$ factors and source categories associated with health effects. It includes the following health effects associated with traffic: Increased total mortality, increased cardio-vascular mortality, increased respiratory mortality, increased emergency department visits for cardiovascular disease, ST-segment depression, increased blood urea nitrogen, increased mean red cell volume, increased blood PMN percent, decreased blood lymphocytes percent, increased von Willebrand factor, decreased protein C, cytotoxic responses, inflammatory responses, and various other cardiovascular and respiratory impacts.
- Rosenbloom, et al. found a significant increase in all-cause mortality for survivors of acute myocardial infarction depending on residential proximity to major roadways.[244]
- A German study found that chronic exposure to $PM_{10}$ pollution increases deaths from cardiopulmonary causes in a cohort of women. Researchers followed 4,800 women living in industrial and nonindustrial areas of the Ruhr area of Germany who had been included in earlier studies to determine their cause of death relative to long term exposures to air pollution. Living within a 50-meter radius of a major road was associated with an increased risk

[240] Cakmak S; Dales RE; Vida CB (2009). Components of particulate air pollution and mortality in Chile. *Int J Occup Environ Health*, 15: 152-158.

[241] Sarnat JA; Marmur A; Klein M; Kim E; Russell AG; Sarnat SE; Mulholland JA; Hopke PK; Tolbert PE (2008). Fine particle sources and cardiorespiratory morbidity: An application of chemical mass balance and factor analytical source-apportionment methods. *Environ Health Perspect*, 116: 459-466.

[242] Williams L, Ulrich C, Larson T, Wener M, Wood B, Campbell P, Potter J, McTiernan A, De Roos A. 2009. Proximity to Traffic, Inflammation, and Immune Function among Women in the Seattle, Washington, Area. *Environ Health Perspect*. 117: 373.

[243] Karr C, Rudra C, Miller K, Gould T, Larson T, Sathyanarayana S, Koenig J. 2009. Infant exposure to fine particulate matter and traffic and risk of hospitalization for RSV bronchiolitis. *Am J Epidemiol*. 165: 553-560.

[244] Rosenbloom JI, Wilker EH, Mukamal KJ, Schwartz J, Mittleman MA. Residential proximity to major roadway and 10-year all-cause mortality after myocardial infarction. *Circulation*. 2012 May 8;125(18):2197-203.

> of death from cardiopulmonary causes. Exposure to elevated levels of nitrogen dioxide and $PM_{10}$ (estimated from total suspended particulates) increased the risk of total morality and mortality due to cardiopulmonary causes. This study provides additional evidence that long-term exposure to air pollution increases the risk of early death, a finding previously reported in U.S. cohort studies.[245]

EPA has identified highways carrying significant numbers of diesel vehicles as the highways that require attention because of their potential for emitting enough fine particles to cause NAAQS violations. In the Hot Spot rulemaking, EPA determined that new or expanded highway projects require a quantitative analysis to determine whether vehicle emissions are likely to cause new, more frequent, or more severe NAAQS violations for $PM_{2.5}$, and concluded "that highway and transit projects that involve significant levels of diesel vehicle emissions have the potential to increase local $PM_{2.5}$ concentrations."[246] EPA drew this conclusion from

> … studies [that] provide strong evidence of elevated $PM_{2.5}$ concentrations along roadways on a consistent basis from certain types of projects. Based on EPA's review of all studies, studies identified elevated $PM_{2.5}$ concentrations of 8% to 60% for high-traffic roadways to 285% for major truck stops, compared to background concentrations. Variables identified in the studies as key predictors of $PM_{2.5}$ concentrations include: Total traffic volume; volume of heavy-duty trucks; traffic congestion; and proximity to major facilities (within approximately 150 meters). Most studies showed elevation in $PM_{2.5}$, black carbon, or other components associated with major facilities (e.g., truck routes, intermodal or bus terminals).[247]

EPA concluded that the evidence of localized impacts from highways was sufficiently compelling that "it is essential that a quantitative $PM_{2.5}$ or $PM_{10}$ hot-spot analysis be performed for all projects of air quality concern."[248] The Agency concluded that highways "that have a significant number of or significant increase in diesel vehicles" were presumptively "projects of air quality concern."[249]

The EPA points out that there are gradients in near-roadway $PM_{2.5}$ that are most likely associated with heavily travelled roads with significant heavy-duty diesel activity.[250] The localized impacts of pollution from highways have resulted in the recent proposed revisions to the NAAQS rule, including addition of

[245] Gehring U, Heinrich J, Krämer U, Grote V, Hochadel M, Sugiri D, Kraft M, Rauchfuss K, Eberwein HG, Wichmann H-E. Long-term exposure to ambient air pollution and cardiopulmonary mortality in women. *Epidemiology* 2006;17: 545-551.
[246] 71 Fed.Reg. 12468, 12,472 (March 10, 2006)
[247] *Id.*
[248] *Id.*
[249] 71 Fed. Reg. at 12,470; 40 C.F.R. § 93.123(b)(i)
[250] 77 Fed. Reg. 39,009 (Jun. 29, 2012)

a near-road component to the $PM_{2.5}$ Monitoring Network.[251] These proposals, and the additional implementation policies that Commenters request be adopted, are supported by the evidence that exposure to emissions from transportation facilities increases the risk of numerous adverse health effects.

## Highway Emissions Cause the Highest Population Exposures to $PM_{2.5}$

Dozens of studies have shown greatly increased pollutant levels and health impacts in close proximity to freeways, prompting the California Air Resources Board ("CARB") to recommend in 2005 that local governments "[a]void siting new sensitive land uses within 500 feet of a freeway, urban roads with 100,000 vehicles/day, or rural roads with 50,000 vehicles/day."[252] The rationale for that caution is summarized as follows: "In traffic-related studies, the additional non-cancer health risk attributable to proximity was seen within 1,000 feet and was strongest within 300 feet. California freeway studies show about a 70% drop off in particulate pollution levels at 500 feet."

In 2010, the Health Effects Institute published a meta-analysis of the proximity studies and concluded that the exposure zones for traffic-related air pollution ranges from 300 to 500 meters from highways and major roads. Their Panel on the Health Effects of Traffic-Related Air Pollution estimated that in North America, between 35 and 45 percent of the population live in such zones.[253] However, residential proximity may underestimate the population exposed to traffic-related PM, as the Institute's assessment estimates that Americans spend an average of 81 minutes per day in vehicles, although many who work in transportation or law enforcement, for example, may spend even more hours per day exposed to these high levels. [254]

An analysis commissioned by NRDC shows that just 16 percent of counties with population living near high-volume roads also have air quality monitors near these roads, resulting in almost 18 million people living where PM may exceed the federal standards but is not monitored.[255] Many studies provide evidence that poor and minority residents are more likely to live near high-volume roads and suffer disproportionately high exposure to air pollution from motor vehicle emissions.[256] For example,

---

[251] *See generally* 77 Fed. Reg. 38890 (Jun. 29, 2012).
[252] California Air Resources Board, Air Quality and Land Use Handbook: A Community Health Perspective, April 2005, http://www.arb.ca.gov/ch/handbook.pdf.
[253] Health Effects Institute, Panel on the Health Effects of Traffic-Related Air Pollution. 2010. Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects. HEI Special Report 17. Health Effects Institute, Boston, MA. p. ix.
[254] HEI, 2010. P. 3-51.
[255] Greg Gould, Census of the U.S. Near Roadway Population, In Publication; in collaboration with NRDC.
[256] Apelberg BJ, Buckley TJ, White RH. 2005. Socioeconomic and Racial Disparities in Cancer Risk from Air Toxics in Maryland. Environ Health Perspect 113:693–699; doi:10.1289/ehp.7609.

in California, over two percent of public schools (K-12) are within 150 meters of high traffic roads and a disproportionately large percentage of students attending these schools are economically disadvantaged and nonwhite.[257]

## Credible Scientific Evidence Demonstrates High Levels of Particulate Matter and Other Pollutants Near Major Roadways.

Numerous studies have demonstrated that concentrations of many pollutants, including $PM_{2.5}$, near highways are significantly higher than background concentrations measured at regional scale monitors.[258] These data confirm EPA's findings in the conformity hot spot rulemaking that the incremental concentrations of $PM_{2.5}$ observed near highways are higher than regional background levels. [259]Combining these results with EPA's conclusion that higher concentrations are linked to traffic levels and proximity to the roadway, it is clear that air monitors must be placed within the impact zone of highways to satisfy numerous criteria in Part 58, including the requirement to site a monitor at the point of maximum expected concentration in the nonattainment area, and the requirement that the monitoring network be adequate to support a demonstration that the SIP will provide for attainment. Monitoring near roadways will ensure that all people within a geographic area have the benefits of the Clean Air Act protections, and it will address serious environmental justice concerns.

---

Cesaroni G, Badaloni C, Romano V, Donato E, Perucci CA, Forastiere F. 2010. Socioeconomic position and health status of people who live near busy roads: the Rome Longitudinal Study (RoLS). Environ Health 9:41–41; doi:10.1186/1476-069X-9-41. Chakraborty J. 2009. Automobiles, Air Toxics, and Adverse Health Risks: Environmental Inequities in Tampa Bay, Florida. Ann. of the Association of Am. Geographers 99:674–697; doi:10.1080/00045600903066490.Gunier RB, Hertz A, Behren J von, Reynolds P. 2003. Traffic density in California: Socioeconomic and ethnic differences among potentially exposed children. J Expo Anal Environ Epidemiol 13: 240–246. Havard S, Deguen S, Zmirou-Navier D, Schillinger C, Bard D. 2009. Traffic-Related Air Pollution and Socioeconomic Status. Epidemiology 20:223–230; doi:10.1097/EDE.0b013e31819464e1. Houston D, Wu J, Ong P, Winer A. 2004. Structural Disparities of Urban Traffic in Southern California: Implications for Vehicle-Related Air Pollution Exposure in Minority and High-Poverty Neighborhoods. J Urban Affairs 26:565–592; doi:10.1111/j.0735-2166.2004.00215.x. Jerrett M, Buzzelli M. 2007. Geographies of Susceptibility and Exposure in the City: Environmental Inequity of Traffic-Related Air Pollution in Toronto. Canadian Journal of Regional Science 30: 195–210. Kingham S, Pearce J, Zawarreza P. 2007. Driven to injustice? Environmental justice and vehicle pollution in Christchurch, New Zealand. Transportation Research Part D: Transport and Environment 12:254–263; doi:10.1016/j.trd.2007.02.004.

[257] Green, R.S. et. al., Proximity of California Public Schools to Busy Roads, *Environmental Health Perspectives* 2004; 112(1): 61-66.

[258] Gould, Draft Report – Near Roadway Emissions: Measurement, Exposure, and Monitoring; in collaboration with NRDC. Also see: Karner, A.A., D.S. Eisinger, and D.A. Niemeier, *Near-Roadway Air Quality: Synthesizing the Findings from Real-World Data.* Environmental Science & Technology, 2010. **44**(14): 5334-5344.
Zhou Y, Levy J. 2007. Factors influencing the spatial extent of mobile source air pollution impacts: a meta-analysis. BMC Public Health 7:89; doi:10.1186/1471-2458-7-89.

[259] 71 Fed. Reg. 12467, 12472

One recent study in the Los Angeles basin measured elevated air pollutants far downwind, up to 2,000 meters and up to 600 meters upwind of a major freeway.[260] The study, along Interstate10, documented high concentrations of ultra-fine particulates, polycyclic aromatic hydrocarbons and nitric oxide at distances of 1,200 meters (roughly 4,000 feet) and farther downwind, especially during pre-sunrise hours when winds were low, humidity was high and there was a surface temperature inversion.

As noted earlier, numerous studies reviewed by the HEI and others provide the strongest evidence that elevated pollutant concentrations can be found within up to 500 meters (1,600 feet) of freeways and busy roadways, although evidence of effect extends up to 1,500 meters.[261] Furthermore, the HEI review also concluded that $PM_{2.5}$ does not decay over a sharp distance as do other traffic-related pollutants. Homes affected by traffic had concentrations similar to those measured at roadside settings. By contrast, ultrafine particles did drop off significantly from the exhaust plumes. [262]

The summary above is supported by the following noteworthy descriptions of elevated pollutant levels near freeways from EPA's Integrated Science Assessment for Particulate Matter:[263]

- In a mobile platform sampling study, Westerdahl et al. (2005)[264] and Fruin et al. (2008)[265] also reported substantial peaks in [Ultra-fine Particles] UFP concentration when sampling at highways in comparison with a background site (the University of Southern California) using the same data set. Near roadway environments can exhibit high concentration gradients, particularly for UFPs.

- Ntziachristos et al. (2007) observed that the near-road particle size distribution was substantially higher in the UF mobility diameter range. [266]
- Baldauf et al. (2008) reported elevated UFP number concentrations downwind of a highway in Raleigh, NC, when compared to measurements approximately 100 meter upwind of the road. [267]

[260] Hu, S. et.al., A wide area of air pollutant impact downwind of a freeway during pre-sunrise hours, *Atmospheric Environment* 2009; 43:2541-2549.
[261] Karner et al., 2010; also HEI, 2010.
[262] HEI, 2010. pp 3-20 to 3-22
[263] EPA, Integrated Science Assessment for Particulate Matter, Office of Research and Development, December 2009; Sections 3.5.1.3 and 3.8.4.1.
[264] Westerdahl D, Fruin S, Sax T, Fine PM, Sioutas C (2005). Mobile platform measurements of ultrafine particles and associated pollutant concentrations on freeways and residential streets in Los Angeles. *Atmos Environ*, 39: 3597-3610.
[265] Fruin S, Westerdahl D, Sax T, Sioutas C, Fine PM (2008). Measurements and predictors of on-road ultrafine particle concentrations and associated pollutants in Los Angeles. *Atmos Environ*, 42: 207-219.
[266] Ntziachristos L; Ning Z; Geller MD; Sioutas C (2007). Particle concentration and characteristics near a major freeway with heavy-duty diesel traffic. *Environ Sci Technol*, 41: 2223-2230.
[267] Baldauf R; Thoma E; Hays M; Shores R; Kinsey J; Gullett B; Kimbrough S; Isakov V; Long T; Snow R; Khlystov A; Weinstein J; Chen FL; Seila R; Olson D; Gilmour I; Cho SH; Watkins N; Rowley P; Bang J (2008). Traffic and meteorological impacts on

- Hagler et al. (2009) noted a 5-12% decrease in number concentrations per 10-meter distance from the road for a number of studies in the U.S. with unobstructed air flow.[268]
- Sharp gradients in black carbon mass have been observed along roadways with high diesel traffic (Zhu et al., 2002).[269]
- Gutiérrez-Dabán et al. (2005) examined the mass distribution of various [Polycyclic Aromatic Hydrocarbons] PAHs under different traffic and urban density conditions. Concentrations were nearly an order of magnitude lower for the low traffic urban periphery location when compared with the high traffic or industrial locations.[270]
- Olson and McDow (2009) reported decreases by a factor of 1.04-2.37 in select PAH and organic source marker concentrations when comparing measurements 10 meters and 275 meters from a highway in Raleigh, North Carolina.[271]
- Morwaska et al. (2008) stated that UFP number concentrations in the near-road environment were roughly 18 times higher than in a non-urban background environment, while measured concentrations in street canyons and tunnels were 27 and 64 times higher, respectively than background. [272]
- By sampling UFP number concentrations at multiple sites in Los Angeles, Moore et al. (2009) demonstrated five- to seven-fold differences between concentrations measured directly next to a freeway and an ocean-side site.[273]
- Zhou and Levy (2007) noted in a meta-analysis of near-road studies that the concentrations are generally elevated within 300-400 meters of a roadway for EC and UFPs.[274]
- Kinney et al. (2000) showed EC to increase linearly with increasing traffic counts and large spatial variations in two sites that had concentrations significantly higher than ambient measurements.[275]

---

near-road air quality: Summary of methods and trends from the Raleigh near-road study. *J Air Waste Manag Assoc*, 58: 865-878.

[268] Hagler GSW; Baldauf RW; Thoma ED; Long TR; Snow RF; Kinsey JS; Oudejans L; Gullett BK (2009). Ultrafine particles near a major roadway in Raleigh, North Carolina: Downwind attenuation and correlation with traffic-related pollutants. *Atmos Environ*, 43: 1229-1234.

[269] Zhu Y; Hinds WC; Kim S; Shen S; Sioutas C (2002). Study of ultrafine particles near a major highway with heavy-duty diesel traffic. *Atmos Environ*, 36: 4323-4335.

[270] Gutiérrez-Dabán A; Fernández-Espinosa AJ; Ternero-Rodríguez M; Fernández-Álvarez F (2005). Particle-size distribution of polycyclic aromatic hydrocarbons in urban air in southern Spain. *Anal Bioanal Chem*, 381: 721-736.

[271] Olson DA; McDow SR (2009). Near roadway concentrations of organic source markers. *Atmos Environ*, 43: 2862-2867.

[272] Morawska L; Thomas S; Jamriska M; Johnson G (1999). The modality of particle size distributions of environmental aerosols. *Atmos Environ*, 33: 4401-4411.

[273] Moore K; Krudysz M; Pakbin P; Hudda N; Sioutas C (2009). Intra-community variability in total particle number concentrations in the San Pedro Harbor Area (Los Angeles, California). *Aerosol Sci Technol*, 43: 587-603.

[274] Zhou Y; Levy JI (2007). Factors influencing the spatial extent of mobile source air pollution impacts: a meta-analysis. *BMC Public Health*, 7: 89.

*These observations support the conclusion that averaged monitoring data, or data from monitoring sites intended to represent regional, urban or neighborhood scale concentrations, do not represent exposures at locations within the zone where emissions from transportation facilities are expected to contribute incremental concentrations of* $PM_{2.5}$.[276]

## Assessing $PM_{2.5}$ Concentrations Near Highways and Other Transportation Facilities to Make Nonattainment Designations.

The legacy of EPA's existing policies for monitoring $PM_{2.5}$ from highways and other transportation facilities is that virtually no monitors exist that are providing data to show where $PM_{2.5}$ emitted from such facilities are causing or contributing to concentrations that violate the revised NAAQS.[277] In the absence of monitored data from near-highway sites, actual air quality data is not available to identify and designate areas where emissions from transportation facilities are causing or contributing to concentrations that will make the area nonattainment for the revised NAAQS.

To resolve this data gap, EPA has two options that could be implemented to meet the statutory deadline for making § 107(d) designations for the revised NAAQS:

1) require $PM_{2.5}$ monitors to be installed within a few months of final promulgation of the revised NAAQS, and use 1 year of data to determine if an area affected by emissions from a major transportation facility are causing the area to exceed the revised NAAQS; or

2) require the states to use dispersion modeling to estimate the incremental contribution of $PM_{2.5}$ emissions from major transportation facilities and add those modeled estimates to regional, urban or neighborhood scale monitored concentrations in the area affected by emissions from the major transportation facility to determine if the resulting cumulative concentration would violate the revised NAAQS.

Commenters urge EPA to adopt the second option for numerous reasons.

First, one year of air quality data is not a reliable measure of whether an area is likely to violate the NAAQS. EPA's traditional requirement in Part 50, Appendix N, that not less than 3 years of data be used to determine whether an area is in attainment is well-grounded in sound science. The effect of

---

[275] Kinney PL; Aggarwal M; Northridge ME; Janssen NAH; Shepard P (2000). Airborne concentrations of $PM_{2.5}$ and diesel exhaust particles on Harlem sidewalks: a community-based pilot study. *Environ Health Perspect,* 108: 213-218

[276] U.S.EPA, Integrated Science Assessment for Particulate Matter, Office of Research and Development, December 2009; Section 3.8.4.1.

[277] Gould, Gregory M., "A Census of the U.S. Near Roadway Population: Particulate Matter Exposure, Environmental Justice, and Coverage of the Air Quality Monitoring Network," Tables 1 and 2, pp. 11-13. [This report has been submitted for publication, and is submitted to EPA under a claim of copyright with a request that it not be disclosed in the docket pending publication.]

using one year of monitor data could often be to designate an area as attainment when that one year of data is not representative of longer term air quality. If EPA were to begin to use one year of data to designate areas as attainment for purposes of making initial designations under § 107(d)(1), EPA would soon come under pressure to allow states to use only one year of data to make redesigations to attainment under § 107(d)(3). Commenters believe that the inherent uncertainty associated with one year of air quality data is not acceptable for making attainment determinations, regardless of whether they are initial designations or subsequent redesignations.

Second, it will be difficult, and possibly impractical, to identify monitoring sites that meet EPA's siting criteria for measuring $PM_{2.5}$ near highways (see section VI, below), install monitoring equipment, and begin operation of sites within the time that will be necessary to ensure that one year of quality assured data is available to make timely designation decisions. These obstacles to establishing monitoring sites is one of the reasons why EPA proposed to delay commencement of monitoring until 2017. While 5 years is likely much longer than needed to establish such sites, a few months is probably not realistic.

Third, the data available within a few weeks of promulgation to identify prospective monitoring sites where the contribution of transportation facility emissions to peak concentrations is greatest will be limited largely to traffic data. While total traffic (AADT) is an important variable, EPA's review of studies for the Hot Spot rule shows that other variables such as truck share of total AADT, time of day when truck share is greatest, meteorology, and background concentrations of $PM_{2.5}$ are important variables too. It may not be possible to fully integrate these variables into the site selection process without modeling.

Fourth, the uncertainty inherent in using one year of data and the impracticality of obtaining air quality data to meet the statutory two-year schedule for designations can be avoided by modeling emissions from transportation facilities of air quality concern to identify the area most likely to have the peak concentration of $PM_{2.5}$. In addition, the multiple variables that play a role in identifying the site with greatest concentrations can be, and typically are, integrated into the modeling of transportation facility emissions.

Fifth, EPA has developed the modeling protocols for estimating the incremental contribution of emissions from transportation facilities to concentrations of $PM_{2.5.}$ EPA issued guidance in 2010 requiring that transportation project sponsors use the dispersion modeling techniques outlined in EPA's conformity guidance for the quantitative analysis of PM emissions from a transportation

project.[278] EPA's quantitative guidance prescribes the assumptions and inputs for modeling emissions from proposed transportation facilities to determine the impact emissions from the facility will have on the NAAQS, and require that control measures be adopted where modeling evidence indicates that emissions will cause or contribute to NAAQS violations. These protocols are available and suitable for application to existing transportation facilities to estimate emissions and associated incremental ambient concentrations attributable to such facilities.

To test the use of these modeling protocols for the purpose of estimating the contribution of emissions from such facilities to ambient concentrations, and to use the outputs of such modeling protocols to identify the locations of expected greatest concentrations, Gould et al. applied EPA's modeling guidance for quantitative analysis of $PM_{2.5}$ emissions to the highway network in Los Angeles County, CA.[279] This application of EPA's modeling guidance for the quantitative assessment of emissions from transportation facilities demonstrates the feasibility of this approach for the purpose of identifying both the expected incremental contribution of $PM_{2.5}$ from a large highway network, and the areas of peak concentrations. This work demonstrates the practicality of using EPA's existing conformity guidance to develop the data relevant to making nonattainment designations under § 107(d)(1).

Obviously, the need to apply this modeling protocol for designation purposes will be limited to areas that are not nonattainment based on concentrations of $PM_{2.5}$ reported by regional, urban or neighborhood scale monitors. Where existing monitoring stations demonstrate that the area is nonattainment for the revised NAAQS, no additional analysis of the contribution from transportation facilities will be required for the purpose of making designations under § 107(d)(1). However, this modeling tool will be relevant to the development of control strategies and attainment demonstrations. See section VII, below.

However, in metropolitan areas where existing regional, urban and neighborhood scale monitors show attainment of the revised NAAQS, these data cannot be relied upon to make attainment designations without considering the incremental contribution of emissions from transportation facilities. The current monitoring network is clearly deficient because of its inability to account for the incremental contribution of emissions from transportation facilities. HEI identifies this deficiency.

[278] "Transportation Conformity Guidance for Quantitative Hot-spot Analyses in PM2.5 and PM10 Nonattainment and Maintenance Areas," (OTAQ, U.S. EPA, December 2010) Accessed at http://www.epa.gov/otaq/stateresources/transconf/policy/420b10040.pdf August 31, 2012.

[279] Gould, Gregory M., et al., "Regional Scale Dispersion Modeling and Analysis of Directly Emitted Fine Particulate Matter from Highway Vehicles Using AERMOD" (July 2012). [This report has been submitted for publication, and is submitted to EPA under a claim of copyright with a request that it not be disclosed in the docket pending publication.]

> Monitoring locations are often selected to maximize regional representation and minimize the influence of local sources (e.g., traffic or point sources). Although the air quality in urban areas is often dominated by contributions from mobile sources, the sites in urban networks are too far apart to assess variations in intra-urban exposures from traffic in small areas. [280]

Zhou, Y., and Levy also demonstrate that air quality reported from existing monitors located outside the 300 to 400 meter zone adjacent to highways is not representative of concentrations in the near-highway atmosphere. For this reason, they conclude that "the observation that the spatial extent is generally within a few hundred meters for highway or city roads demonstrates the need for high resolution modeling near the source."[281] Gould's analysis of the location of $PM_{2.5}$ monitors finds only 9 are located within 100 M of highways with 100,000 AADT, and only 3 are located within 100 M of highways with more than 200,000 AADT. [282] This analysis demonstrates conclusively that the monitors in the current $PM_{2.5}$ network are not located near transportation facilities in accordance with EPA's siting criteria in Part 58, Appendix E, and in all but a few cases are not located in sufficient proximity to such facilities to even detect some incremental contribution of $PM_{2.5}$ emitted from such facilities.

The use of modeling to determine initial attainment status for designation purposes is not novel. Commenters believe that this approach closely follows the modeling approach adopted for $SO_2$.

> [I]n areas without currently operating monitors but with sources that might have the potential to cause or contribute to violations of the NAAQS, we anticipate that the identification of NAAQS violations and compliance with the 1-hour SO2 NAAQS would primarily be done through refined, source-oriented air quality dispersion modeling analyses, supplemented with a new, limited network of ambient air quality monitors.[283]

The policy reasons that convinced EPA to adopt modeling as the preferred method for designating nonattainment areas where large $SO_2$ sources exist, but monitoring data is not available, provide support for using a similar approach for designating areas with unmonitored, major transportation facilities.

Therefore EPA must consider additional sources of data to determine whether areas where regional, urban and neighborhood monitors demonstrate attainment are actually in attainment when the

[280] HEI, 2010.
[281] Zhou, Y, J.I. Levy, "Factors influencing the spatial extent of mobile source air pollution impacts: a meta-analysis," BMC Public Health (2007),
[282] Gould, Gregory M., "A Census of the U.S. Near Roadway Population: Particulate Matter Exposure, Environmental Justice, and Coverage of the Air Quality Monitoring Network," Table 1. [This report has been submitted for publication, and is submitted to EPA under a claim of copyright with a request that it not be disclosed in the docket pending publication.]
[283] 75 Fed. Reg. 35551 (June 22, 2010).

incremental contribution of emissions from transportation facilities are added to background concentrations. In the absence of spatially relevant monitoring data, we agree with Zhou and Levy that this data gap should be filled with high resolution modeling of transportation facility emissions. EPA's quantitative conformity modeling guidance provides an appropriate protocol for undertaking this analysis to supplement background monitoring data for the purpose of making § 107(d)(1) designations. Commenters ask EPA to require this approach to ensure that areas where incremental concentrations of $PM_{2.5}$ associated with emissions from major transportation facilities cause or contribute to levels that violate the revised NAAQS are identified and designated nonattainment.

To implement this approach Commenters believe it would be appropriate to exclude entirely from this analysis areas with monitored concentrations sufficiently below the NAAQS to accommodate emissions from a transportation facility with an expected large incremental contribution to local $PM_{2.5}$ concentrations, or where the transportation facility with the highest expected incremental contribution to local $PM_{2.5}$ concentrations is less than the margin between monitored concentrations and the NAAQS. However, commenters are not prepared at this time to suggest what these exclusion criteria should be. Rather we ask that EPA conduct a separate rulemaking for the purpose of developing these criteria to govern the designations decision process.

## Monitor Siting Criteria.

Assuming that modeling is used to identify and designate areas where emissions from transportation facilities are causing or contributing to violations of the revised $PM_{2.5}$ NAAQS, the results of modeling the transportation network in large metropolitan areas would be available during 2014 to select monitor locations that would commence operation in 2017. Commenters do not oppose commencing operation of the monitors in 2017 provided EPA does not rely on those monitors to commence the designation and SIP development schedules in the Act.

### *Transportation-Focused Monitors Should not be limited to CBSAs with One Million Residents.*

EPA's proposal to site near-roadway monitors only in CBSA's with a population of one million persons of greater must be rejected. EPA's approach would exempt from the near-highway monitoring requirement areas like the San Joaquin Valley that suffer from extreme elevated PM2.5 pollution levels AND significant diesel truck traffic volumes on I-5 and US 99. Using modeling to determine siting would better targets those areas likely to see the peak concentrations resulting from the combination of background and traffic-related emissions. Commenters understand EPA's desire to prioritize resources, but population is not a reasonable surrogate for traffic volumes and truck share to identify the areas at greatest risk.

The evidence EPA reviewed in the transportation hot spot rulemaking convinced the Agency that the critical variables associated with the near-highway increment are primarily diesel traffic and proximity to the source.

- Black/elemental carbon (BC or EC) mass concentrations and particle number (e.g., ``ultrafines'') concentrations are consistently associated with proximity to traffic (generally within 150 meters).
- PM2.5 is associated with proximity to traffic in most, but not all cases.
- Both regional background and local sources contribute to site-specific PM2.5 concentrations.
- The ``near-roadway increment'' of PM2.5 tends to be comprised of approximately 50-80% black or elemental carbon (indicating mobile sources are a key source).[284]
- EPA found that "Trucks were estimated to contribute between 5.0-14.2 [mu]g/m3 PM2.5, depending on the level of truck traffic."[285]

EPA's conclusions from the data establish that the variables associated with the $PM_{2.5}$ *increment* are related to traffic density (AADT) and truck share, not surrounding population density or the size of the metropolitan airshed. *Total* $PM_{2.5}$
concentrations for a location will include the increment added by emissions from the transportation facility to the urban background. Monitors need to be cited where they will detect the maximum concentration that will result from adding the increment caused transportation facilities to the regional background. But the potential for NAAQS violations near transportation facilities will not be limited to large urbanized areas with more than 1 million population. The risk of NAAQS violations will exist wherever emissions from a transportation facility are large enough to make up the difference between the urban background and the NAAQS. Many moderately sized cities with urban background concentrations below the NAAQS will exceed the NAAQS if a contribution of 2-5 $\mu g/m^3$ is added to the urban background. All of these areas where the highway increment, when added to the urban background, will cause NAAQS violations need to be identified.

As discussed in section V, Commenters ask that until monitors provide three-years of quality data at the nonattainment area's peak, EPA require modeling to identify likely nonattainment areas wherever an urbanized area contains a highway or other transportation facility that meets the Agency's criteria for identifying "projects of air quality concern."[286] Thus the problem EPA seeks to avoid, i.e., not using limited monitoring resources in areas where monitoring may not be needed to determine whether the nonattainment status has actually been eliminated, can be avoided. Using modeling to make the first round of designations, will also ensure the monitors are sited only in areas where emissions from

[284] 71 Fed. Reg. 12472-73.
[285] *Id.,* 12473.
[286] 71 Fed. Reg.

transportation facilities are shown to contribute to the nonattainment peak, and one monitor can be located at the expected peak.

### *Current $PM_{2.5}$ Monitoring Siting Criteria Should be Retained.*

Commenters ask that EPA retain its current rule governing the distance criteria for siting $PM_{2.5}$ monitors near highways or other transportation facilities.[287] We believe that these criteria for siting $PM_{2.5}$ monitors when "the maximum concentration area(s) is judged to be a traffic corridor or street canyon" were appropriate when first adopted, and remain appropriate for the stated purpose of monitoring "localized hot-spot sites in areas of highest concentrations." As discussed above, we believe this policy is not only appropriate to ensure that no health sacrifice areas are created, but it is required by the statutory mandate to attain the NAAQS in all geographic areas of the State.

The longstanding requirement that monitors sited to detect the "highest concentrations" "must be between 5 and 15 meters from the major roadway" is potentially at odds with the current proposal to co-locate $PM_{2.5}$ monitors with $NO_2$ monitors if the $NO_2$ monitoring stations have been sited more than 15 meters from the major roadway. EPA has not provided any justification for abandoning its longstanding policy of siting monitors to detect the "localized hot-spot sites in areas of highest concentrations." The primary rationales offered by the Agency for proposing to co-locate $PM_{2.5}$ monitors with $NO_2$ monitors are 1) to reduce cost, 2) make it more convenient for agency staff to service both monitors at a single location, 3) avoid the need for finding multiple sites with access to power and security, and 4) to facilitate health research by collecting multi-pollutant highway-related data at sites where different locations do not introduce significant variables related to distance, traffic and meteorological conditions.

Commenters agree that these reasons for the proposal provide a rational basis for a policy to co-locate $NO_2$ and $PM_{2.5}$ monitors, but they do not provide a rational basis for co-locating both monitors at sites more than 15 meters from the traffic corridor which is the source of air quality concern. EPA does not propose to abandon the Agency's stated intent underlying the 15-meter rule for $PM_{2.5}$ monitors, i.e., "The intent is to locate localized hot-spot sites in areas of highest concentrations."[288] Therefore there is no rational basis for abandoning the 15-meter rule. To allow $PM_{2.5}$ monitors to be sited 50 meters from

[287] 40 C.F.R. Part 58, Appdx E, § 6.3(b).
"The intent is to locate localized hot-spot sites in areas of highest concentrations whether it be from mobile or multiple stationary sources. If the area is primarily affected by mobile sources and the maximum concentration area(s) is judged to be a traffic corridor or street canyon location, then the monitors should be located near roadways with the highest traffic volume and at separation distances most likely to produce the highest concentrations. For the microscale traffic corridor site, the location must be between 5 and 15 meters from the major roadway. For the microscale street canyon site the location must be between 2 and 10 meters from the roadway. For the middle scale site, a range of acceptable distances from the roadway is shown in figure E-1 of this appendix."
[288] 40 C.F.R. Part 58, Appdx E, § 6.3(b).

the roadside, as EPA's siting guidance for $NO_2$ monitors allows, would have the effect of not locating the site with the highest $PM_{2.5}$ concentrations because all of the evidence in the record shows that concentrations of $PM_{2.5}$ decrease with distance from the roadside. The consequence of this policy would be to create 50-meter zones between the highway and the monitor where $PM_{2.5}$ concentrations would clearly be expected to exceed the levels monitored 50 meters away, and that would inevitably violate the NAAQS in cases where the monitor barely measured attainment.

The population within this 50-meter zone is not trivial. Gould's analysis demonstrates that roughly 2 million American's reside within 100 meters of major highways (> 100,000 AADT).[289] The population within 50 meters of the same highways can reasonably be estimated to be roughly 1 million. These residents should not be consigned to a 50-meter health sacrifice zone. Therefore Commenters urge EPA to retain the current siting criteria in Part 58, Appdx E § 6.3, for locating $PM_{2.5}$ monitors near highways that are determined to contribute to the highest concentrations in a $PM_{2.5}$ nonattainment area.

### *Species Analyzed at Transportation-Oriented Monitors Should Include EC/BC, Organic Carbon and Ultra-Fines.*

Monitoring data collected at sites located to measure the incremental contribution from transportation facilities should include analysis of the species that are primarily associated with transportation sources. To support future control strategy development in cases where initial SIPs developed from modeling data fail to attain the NAAQS, measurement of the contribution of transportation-related species will provide important information about the cause of SIP failure. To correct a SIP failure, it will be important to know whether the excess concentrations are attributable primarily to emissions from local transportation facilities or from regional sources.

In addition, to support research related to the assessment of health risks associated with ultrafine particles emitted from transportation facilities, particle counts in the ultrafine range should also be obtained in addition to particle mass.

## Modeling Guidance for SIP Attainment Demonstrations Must be Revised to Require High Resolution Modeling of Highway Emissions.

[289] Gould, Gregory M., "A Census of the U.S. Near Roadway Population: Particulate Matter Exposure, Environmental Justice, and Coverage of the Air Quality Monitoring Network," Fig. 1 (July 2012).

### *Defining the Design Value in the Absence of Monitors Located to Measure the Incremental Contribution to Ambient Concentrations from Transportation Sources.*

EPA's Implementation Rule for $PM_{2.5}$ and guidance for the development of control strategies designed to implement the PM NAAQS focuses on urban and regional scale modeling to demonstrate that proposed control measures will provide for attainment at the "design value" monitor reporting the concentrations that violate the NAAQS.

> PM2.5 design value for a nonattainment area is the highest of the three-year average concentrations calculated for the monitors in the area, in accordance with 40 CFR part 50, appendix N.[290]

Given the data "black hole" around transportation facilities created by the absence of monitoring within the 300 to 400-meter zone of impact for $PM_{2.5}$, EPA needs to recognize and account for the fact that, unless the monitor with "the highest of the three-year average concentrations calculated for the monitors in the area" is in fact located near the transportation facility expected to contribute the greatest increment to regional background concentrations, then the concentration measured at the highest regional, urban or neighborhood scale monitor not representative of the increment contributed by transportation emissions does not represent the highest $PM_{2.5}$ concentration for the nonattainment area. To ensure that the NAAQS is attained near transportation facilities as well as at regional monitoring sites, the control strategy must be designed to reduce the incremental concentrations caused by emissions from transportation facilities. The highest concentration among the regional, urban or neighborhood scale monitor cannot be used as the design value for the entire nonattainment area because a control strategy demonstrated to attain at that monitor will not demonstrate attainment at locations where direct particles emitted from transportation facilities locally add pollution to regional levels.

To develop a design value for use in the development of a control strategy that accounts for the incremental concentrations contributed by emissions from transportation facilities, Commenters recommend that EPA address this gap in the monitoring record by requiring the air quality planning agency for each nonattainment area to apply the same dispersion modeling methods recommended above, see section V, to determine whether areas with monitored attainment, but with unmonitored high traffic transportation facilities, should be designated nonattainment. The fact that these modeling methods and procedures have been developed with extensive input from the regulated community, and are now in use to estimate emissions under the transportation conformity Hot Spot rule, should provide confidence that these methods will also serve to inform the SIP development process, and not impose excessive burdens on planning agencies.

[290] 40 C.F.R. §51.1000 Definitions.

The modeling analysis of the highway network in Los Angeles County performed by Gould, et al., demonstrates how the outputs from EPA's conformity modeling protocol provide useful information for determining an area's design value. EPA's protocol produces high resolution concentrations at the 20-meter scale which can be combined with the concentrations measured at regional, urban or neighborhood scale monitors to determine the expected peak value in a nonattainment area. This approach also provides important source information useful in the design of control strategies. Monitors properly sited near transportation facilities would not likely provide as clear an understanding of the relative contribution of regional emissions sources versus local transportation sources as the combination of modeling transportation sources separately from regional source emissions which are reflected in large scale monitoring results.

### *Revising Modeling Guidelines for Attainment Demonstrations.*

EPA's Implementation Rule requires that "The attainment demonstration and supporting air quality modeling should be consistent with EPA's PM2.5 modeling guidance."[291]

Currently, EPA's modeling guidance for SIP demonstrations requires a large-scale gridded model to demonstrate that average concentrations in the grid where the design value monitor is located will attain the NAAQS.[292] But EPA's Implementation Rule also recognizes the need to identify areas that are not monitored where ambient violations may be anticipated, and to model these locations to determine if additional control measures are needed to attain the NAAQS:

> In addition [to predicted concentrations at monitors], we describe an "unmonitored area analysis" which uses interpolated ambient data combined with gridded model outputs to examine whether potential violations of the NAAQS may occur in unmonitored areas. If potential violations are indicated, we recommend further analysis of the problem through additional local modeling. Options for State action to address such a situation could include imposition of reasonably available control technology to reduce emissions, or the deployment of an air quality monitor to further characterize the problem.
>
> We believe that the combination of these model-based tests will adequately determine whether attainment of the standards is likely by the attainment date. We also believe

[291] 40 C.F.R. §51.1007.
[292] "Guidance on the Use of Models and Other Analyses for Demonstrating Attainment of Air Quality Goals for Ozone, PM2.5, and Regional Haze," US EPA (April 2007).

> that these tests address the issue of hotspots by recommending a combination of photochemical modeling, dispersion modeling of local sources, and additional monitoring and/or emissions controls.[293]

The monitoring guidance "recommends" supplemental modeling when there is reason to believe that high to local $PM_{2.5}$ concentrations of $PM_{2.5}$ are occurring in unmonitored areas.

> To address the issue of PM2.5 concentrations in unmonitored area, we have recommended an 'unmonitored area analysis' (see section 3.4). The unmonitored area analysis is intended to be the primary means for identifying high PM2.5 concentrations outside of traditionally monitored locations. The spatial resolution of the modeling that is the underlying basis of the unmonitored area analysis will determine how well it addresses primary PM hotspot issues.[294]

This process is appropriate for demonstrating that the control strategy will reduce the incremental contribution to ambient concentrations of $PM_{2.5}$ that result from transportation facility emissions. But this approach can no longer be simply a recommended policy. One or more of the commenters have asked EPA to require that air agencies perform such supplemental modeling for transportation facilities to support their claims that control strategies will provide for attainment near such facilities. EPA has declined to do so, arguing that the Guidance merely recommends an approach to the states, but does not require it.

Now that EPA is formally recognizing the significance of the health risks associated with exposure to $PM_{2.5}$ from transportation facilities, and the importance of monitoring these emissions and preventing NAAQS violations associated with these emissions, EPA must also make clear to the states that they must perform the supplemental modeling required to demonstrate that the control strategy will provide for local air quality near transportation facilities that will attain the NAAQS. We therefore ask that this section of the Modeling Guidance require supplemental modeling when the nonattainment area includes major transportation facilities.

In addition, we ask that the Guidance require supplemental modeling that provides outputs for receptors at the 20-meter scale to parallel the protection offered by EPA's monitor siting criteria in Part 58, Appendix E, § 6.3.

[293] 72 Fed. Reg. 20607 (April 25, 2007).
[294] "Guidance on the Use of Models and Other Analyses for Demonstrating Attainment of Air Quality Goals for Ozone, PM2.5, and Regional Haze," US EPA (April 2007), ¶5.3.2.

## Petition for Rulemaking.

Commenters' request in section VII of these comments that EPA revise the Modeling Guidance incorporated by reference into the Implementation Rule for $PM_{2.5}$ , 40 C.F.R. § 51.1007, raises an issue involving the Implementation Rule that EPA did not explicitly propose to address in the NAAQS rulemaking. Nonetheless, we believe that the proposed changes to regulations 1) to require compliance with both the revised annual and 24-hour NAAQS at micro- and middle scale monitors, 2) to eliminate the requirement that such monitors be "population-oriented," and 3) to require monitoring at the location where emissions from transportation facilities are expected to cause or contribute to the highest concentration in an area, implicates the provisions of the Implementation Rule that establish the requirements for SIPs to demonstrate attainment of the revised NAAQS. The new monitoring requirements are based on the need to demonstrate attainment in localized areas where emissions from transportation facilities are expected to cause or contribute to peak concentrations that would not previously have been the focus of the development of control strategies for SIPs. To the extent that these local areas near transportation facilities where $PM_{2.5}$ has not been previously monitored are now a focus of regulatory attention, that attention needs to be reflected in EPA's guidance for control strategies and the modeling required to demonstrate that the strategy will be adequate to attain the NAAQS at these locations.

Should the Agency decide that policies in the Implementation Rule and Modeling Guidance are not necessarily implicated by the changes in the applicability of the revised NAAQS, and new monitoring requirements to demonstrate final attainment of the revised NAAQS near transportation facilities, Commenters hereby submit this Petition for Rulemaking to open the Implementation Rule for the purpose of making mandatory the modeling procedures that EPA had previously only recommended to demonstrate that the control strategy for a nonattainment area is adequate to provide for attainment in areas near transportation facilities that are expected to experience locally elevated concentrations of $PM_{2.5}$ above the levels reported at regional, urban and neighborhood scale monitors. This Petition requests that EPA open a rulemaking to make the changes proposed by Commenters in section VII of these comments.

## Prepared by

Diane Bailey, M.S.
Senior Scientist
Natural Resources Defense Council

Paul Cort
Staff Attorney
Earthjustice

Elena Craft, Ph.D.
Health Scientist
Environmental Defense Fund

Emily Davis
Staff Attorney
Natural Resources Defense Council

John Graham, Ph.D.
Senior Scientist
Clean Air Task Force

Will Margrabe
Staff Attorney
Clean Air Council

Adriano Martinez
Staff Attorney
Natural Resources Defense Council

Jennifer Nearhold
Legal Intern
Natural Resources Defense Council

Janice E. Nolen
Assistant Vice President, National Policy & Advocacy
American Lung Association

Deborah Shprentz
Consultant to the American Lung Association

Joanne Spaulding
Senior Managing Attorney
Sierra Club

Robert Yuhnke
Transportation Air Quality Consultant