Comments of

American Lung Association
Appalachian Mountain Club
Center for Biological Diversity
Clean Air Council
Earthjustice
Environmental Defense Fund
National Parks Conservation Association
Natural Resources Defense Council
Sierra Club

on EPA's Proposed Revisions to the National Ambient Air Quality Standards for Ozone

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I. EXECUTIVE SUMMARY

The Clean Air Act requires the U.S. Environmental Protection Agency to set a primary National Ambient Air Quality Standard (NAAQS) for ozone pollution at a level that protects public health with an adequate margin of safety, and it requires the nation to then achieve compliance with that level of air pollution. The Environmental Protection Agency (EPA) has appropriately recognized that the existing ozone NAAQS of 75 ppb standard falls far short of providing this level of assurance, even for healthy individuals.

An extensive body of scientific literature unequivocally documents the need for a 60 ppb standard. Chamber studies based on exposure of healthy individuals to concentrations of 60 ppb for less than 8 hours identified the onset of adverse health effects as defined by the American Thoracic Society, the Clean Air Scientific Advisory Council (CASAC), and even the EPA in prior iterations of the ozone NAAQS review, including statistically significant lung function decrements and airway inflammation. Sensitive populations, including children, asthmatics, the elderly, and outdoor workers, exposed to similar concentrations for similar durations are likely to experience even more significant impacts. Epidemiological studies bolster the conclusions of the controlled human studies and link ozone exposures at levels below 65 ppb to a wide range of serious clinical effects including respiratory morbidity and mortality.

The undersigned groups urge EPA to instead fulfill its unambiguous mandate under the Clean Air Act to protect public health by revising the primary ozone NAAQS to the level clearly dictated by the science: 60 parts per billion. By EPA's own assessment, achieving a standard of 60 ppb, even exclusive of the massive health benefits that would accrue in California, would result in as many as 7,900 lives saved, 1.8 million fewer asthma exacerbations for children, and 1.9 million fewer lost school days each year.[1] By contrast, a standard of 65 ppb would achieve approximately half of these benefits, and a standard of 70 ppb is projected to save thousands fewer lives each year, and avoid only one-sixth of the asthma exacerbations and lost school days of a 60 ppb standard.

Moreover, in adopting a new standard, it is critical that EPA incorporate and provide for the fact that the "form" of the standard that EPA proposes to adopt will greatly undermine the protective value of the nominal numerical (e.g., 60, 65 or 70 ppb) level of the standard. Under the current and proposed form of the standard, areas currently attaining levels of 70 and 65 ppb routinely record numerous occurrences of 8-hour daily maximum ozone concentrations of 70 ppb, 75 ppb, and even 80 ppb and above.[2]

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[1] U.S. EPA Regulatory Impact Analysis of the Proposed Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone (EPA-452/P-14-006), ES-14-ES-15, Tbl. ES-7 (2014).

[2] Seven years after the 75 ppb standard was established, areas currently meeting the 75 ppb NAAQS routinely record numerous exceedances of increments 5 ppb higher (i.e., 75, 80, and 85); there is no basis to conclude that an analogous distribution of ozone levels would not occur under a lower NAAQS.

We identify technical flaws in EPA's exposure assessment that lead to underestimating exposures. But more importantly, EPA's proposed range standard cannot be squared with the Clean Air Act because it necessarily requires people—especially sensitive populations such as children--to stay indoors to avoid exposures to levels of air quality known to be unhealthful. This is all the more problematic because some people do not have an option to limit outdoor activity, such as the nearly 9 million workers whose jobs require them to be out of doors all day.

In addition, a weak ozone standard poses a unique injustice to disadvantaged and minority communities. Across the nation, minorities are consistently overrepresented in areas with higher ozone levels and that are in nonattainment of ozone NAAQS. Furthermore, across the nation the asthma burden of minorities—particularly among blacks—is far higher than that of whites. EPA must adopt a 60 ppb standard if it is to protect these communities

Ultimately, the Clean Air Act promises Americans that anyone can go outside whenever they want, for as long as they want, and where air quality is attaining the NAAQS it will be safe for them to breathe. EPA must adopt a level no higher than 60 ppb if EPA is to fulfill this promise. The high end of EPA's proposal—70 ppb—would be a fundamental betrayal of this promise. It would wholly fail to eliminate occurrences of multiple occurrences of levels of 60, 70, and even 80 ppb. And it is inconsistent with the scientific advice of CASAC, which concluded that "[a]t 70 ppb, there is substantial scientific evidence of adverse effects . . . including decrease in lung function, increase in respiratory symptoms, and increase in airway inflammation."[3]

As for the secondary, welfare-protective, standard, EPA's proposal to set it identical to the primary standard is unlawful and arbitrary. CASAC and the National Park Service have strongly endorsed setting a distinct secondary standard to protect vegetation. Thus, to protect vegetation and ecosystems and to mitigate anthropogenic climate change, we call for EPA to follow the science and set a single-year W126 standard that is no higher than 7 ppm-hrs.

II. LEGAL STANDARD

A. Overview of Clean Air Act Legal Mandates Governing National Ambient Air Quality Standards

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. 42,381 (December 18, 1970).

[3] Letter from CASAC Chair Dr. H. Christopher Frey to U.S. EPA Administrator Gina McCarthy re: Second Draft Policy Assessment for the Review of the Ozone National Ambient Air Quality Standards (EPA-CASAC-14-004), ii, June 26, 2014 [hereinafter CASAC Letter 2014a]

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 pollutant to below the level of the NAAQS and to keep it there.

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." *Id.* § 7408(a)(1)(A), (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." *Id.* § 7409(b)(1). To ensure that the NAAQS continue to provide the necessary protection, at least every five years, EPA must review and revise as appropriate the underlying air quality criteria and the NAAQS themselves to keep pace with scientific understanding. *Id.* § 7409(d)(1). Any primary NAAQS 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.

The Act delegates to the Administrator the responsibility to review and revise NAAQS. Thus, neither the Office of Management and Budget (OMB) nor any other agency has authority to alter or override the Administrator's decisions in the review and revision process. Further, technical judgments or opinions that appear to come from OMB, and not EPA, are not entitled to

judicial deference. *See, e.g.*, *Pub. Citizen Health Research Group v. Tyson*, 796 F.2d 1479, 1505 (D.C. Cir. 1986).

B. EPA's Prior Implementation of the Ozone NAAQS

One of the first pollutants for which EPA adopted NAAQS was ozone, a principal component of urban smog, and a severe lung irritant even to healthy adults. *See* 66 Fed. Reg. 5002, 5012/3 (Jan. 18, 2001). The first predecessor to the current primary ozone NAAQS was promulgated in 1971 at 0.08 ppm, averaged over one hour. 36 Fed. Reg. 8187 (April 30, 1971). *See Am. Petroleum Inst. v. Costle*, 665 F.2d 1176, 1182 (D.C. Cir. 1981) (though the 1971 standard was nominally addressed to photochemical oxidants, compliance was gauged by measuring only ozone). In 1979, EPA relaxed this standard to 0.12 ppm, also averaged over one hour. 44 Fed. Reg. 8220 (Feb. 8, 1979).

Subsequently, a growing body of peer-reviewed scientific evidence emerged, documenting the inadequacy of the 1979 standard to protect public health with an adequate margin of safety. However, despite the Act's express mandate to review and (as appropriate) revise NAAQS at intervals of no greater than five years, 42 U.S.C. § 7409(d)(1), EPA failed to consider the new evidence, or to revise the NAAQS to reflect it. 58 Fed. Reg. 13,008, 13,013 (Mar. 9, 1993) (EPA "missed both the 1985 and 1990 deadlines for completion of [ozone NAAQS] review cycles under section 109(d)"). Even after being sued by American Lung Association and ordered to complete a review of the NAAQS, EPA issued a final decision that still refused to consider the new evidence—and declined to revise the NAAQS. 58 Fed. Reg. at 13,008, 13,013-14, 13,016. When that decision was challenged in the D.C. Circuit, EPA sought and received a voluntary remand to consider the new science. Order of June 27, 1994 in *American Lung Association v. Browner*, D.C. Cir. No. 93-1305.

Finally, many years after the new evidence started to emerge, EPA completed a NAAQS review considering that evidence. That review produced the 1997 eight-hour NAAQS, at 0.08 ppm (equivalent to 0.084 ppm). 62 Fed. Reg. 38,856 (July 18, 1997). After several years of litigation, the D.C. Circuit upheld the standard against industry challenge. *Am. Trucking Ass'ns v. EPA*, 283 F.3d 355 (D.C. Cir. 2002); *see also Am. Trucking Ass'ns v. EPA*, 175 F.3d 1027 (D.C. Cir. 1999), *reh'g granted in part and denied in part*, 195 F.3d 4 (D.C. Cir. 1999), *aff'd in part and rev'd in part sub nom. Whitman v. Am. Trucking Ass'ns*, 531 U.S. 457 (2001).

EPA then failed again to timely review and revise the 1997 NAAQS, leading to another suit forcing it to carry out its mandatory duty under 42 U.S.C. § 7409(d). *Am. Lung Ass'n v. Whitman*, No. 03-CV-778 (D.D.C.). In the review process, the Clean Air Scientific Advisory Committee (CASAC), which is charged with reviewing the air quality criteria and NAAQS and making scientific recommendations on them, unanimously found that the primary NAAQS should be revised to a level between 0.060 and 0.070 ppm. In 2008, EPA disagreed with CASAC and set the primary standard at 0.075 ppm. 73 Fed. Reg. 16,436 (Mar. 27, 2008).

Soon thereafter, EPA itself raised concerns about whether its 2008 standards complied with the Act and began a reconsideration of the NAAQS. In early 2010, based solely on the information already before it, EPA proposed to strengthen the primary NAAQS to somewhere

within the CASAC-recommended range of 0.060-0.070 ppm. 75 Fed. Reg. 2938 (Jan. 19, 2010). The proposal stated that the Administrator "judge[d] that a standard level of 0.075 ppm is not sufficient to provide [health] protection with an adequate margin of safety." *Id.* at 2996/2. EPA ultimately declined to finalize its proposed reconsideration, instead assuring the D.C. Circuit that it would address the reconsideration when it completed its next review and revision of the ozone NAAQS. *See Mississippi v. EPA*, 744 F.3d 1334, 1341-42 (D.C. Cir. 2013). Ultimately, the D.C. Circuit upheld the 2008 standard. *Id.* at 1342.

The 2008 standard was due for review and revision in 2013. Because EPA missed the deadline, American Lung Association, Sierra Club, and others again sued EPA and obtained an order requiring EPA to complete this review by October 1, 2015. Order, *Sierra Club v. EPA*, No. 13-CV-2809 (N.D. Cal. Apr. 30, 2014).

C. The Clean Air Act Requires EPA to Set the 2015 Ozone NAAQS at a Level That Protects the Public Health of All Americans and That Provides for an Adequate Margin of Safety Where There Is Uncertainty

In setting or revising a primary NAAQS, section 109 of the Clean Air Act requires that EPA assure the protection of public health with an adequate margin of safety. As noted above, 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. 42,381 (Dec. 18, 1970) (remarks of Senator Muskie, floor manager of the conference agreement). Thus:

> 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. at 8210. Thus, as EPA has acknowledged, it cannot deny Americans protection from the effects of air pollution 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.[4]

Likewise, in implementing the mandates in section 109(b) and setting the NAAQS, 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.

[4] *See also* 116 Cong. Rec. 32,901 (Sept. 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 33,114 (Sept. 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 33,116 (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 42,392 (Dec. 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 42,523 (remarks of Congressman Vanik) ("Human health and comfort has been placed in the priority in which it belongs -- first place.").

See, e.g., *Nat'l Envtl. Dev't Ass'n's Clean Air Project v. EPA*, 686 F.3d 803, 810 (D.C. Cir. 2012). 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 that given pollution concentration and the health effects that result from that level of concentration of pollution, while providing 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. 95-294, at 43-51 (1977); *Ethyl Corp. v. EPA*, 541 F.2d 1 (D.C. Cir. 1976) (*en banc*). That is all the more true where the effects involved include highly serious ones like death and hospitalization. See *Ethyl Corp.*, 541 F.2d at 18 ("the public health may properly be found endangered … by a lesser risk of a greater harm").

D. In Setting the Ozone NAAQS EPA Must Err on the Side of Protecting Public Health When There Is Scientific Uncertainty

The D.C. Circuit has characterized the NAAQS as "preventative in nature." *E.g.*, *Ethyl Corp.*, 541 F.2d at 15; *see also* H. Rep. No. 95-294, at 49-51 (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"). 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: "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.'" *E.g.*, *Am. Trucking Ass'ns*, 283 F.3d at 369, 378.

Thus, in keeping with the precautionary and preventative nature of the NAAQS, EPA must set a new ozone standard that protects against potential adverse health effects—not just those impacts that have been well established by science. *See Am. Trucking Ass'ns*, 283 F.3d at 369 (citing 1997 Ozone NAAQS, 62 Fed. Reg. 38,857 (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 this same "margin of safety" requirement, 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. Am. 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"). Rather, as discussed above, EPA must take a protective and precautionary approach that errs on the side of caution in interpreting uncertainty.

E. EPA Must Also Set the At A Level That Protects Vulnerable Subpopulations

Importantly, 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, the D.C. Circuit has 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 Coal. of Battery Recyclers Ass'n v. EPA*, 604 F.3d 613, 618 (D.C. Cir. 2010); *Am. Farm Bureau Fed'n v. EPA*, 559 F.3d 512, 524 (D.C. Cir. 2009). EPA must also build into the NAAQS an adequate margin of safety for these sensitive subpopulations. *See Am. Farm Bureau Fed'n*, 559 F.3d at 526.

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. 91-1196, 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*, 684 F.3d at 810. 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, over 85 million Americans have cardiovascular disease;[5] 12.7 million have been diagnosed with Chronic Obstructive Pulmonary Disease (COPD), which includes both emphysema and chronic bronchitis;[6] and 25.9 million Americans, including 7.1 million children, have chronic asthma.[7] Considering that these disease categories alone encompass one-third of the population of the United States, the public health implications are enormous. Children and the elderly are additional populations at increased risk from ozone air pollution, according to the Integrated Science Assessment (ISA).[8] There are 74 million children under 18 and 40 million adults over 65 in the United States. Proposed Rule at 75,269/2. Socioeconomic status—living in poverty—may also be risk factor, per the ISA. According to the Census Bureau, 45.3 million people live in poverty.[9]

F. EPA Cannot Consider the Economic Cost of Meeting the 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.

G. EPA Must Give Due Deference to the Advice of CASAC

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), 7607(d)(3). When CASAC makes a scientific finding, it is not enough for EPA merely to "disagree" with CASAC's findings on policy grounds: "to the extent that CASAC has exercised scientific judgment, EPA must respond in kind." *Mississippi*, 744 F.3d at 1358. Nor can EPA rotely invoke "uncertainty" to justify disagreeing with CASAC's scientific judgment. *Id.* at 1357.

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[5] D. Mozaffarian et al., on behalf of the American Heart Association Statistics Committee and Stroke Statistics Subcommittee . Heart disease and stroke statistics—2015 update: a report from the American Heart Association, *Circulation*, 131: e29-e322, e156 (2015).

[6] CDC, National Center for Health Statistics. National Health Interview Survey Raw Data (2011). Calculations by the American Lung Association Research and Health Education Division using SPSS and SUDAAN software.

[7] CDC. Behavioral Risk Factor Surveillance System (2011); *accord* 79 Fed. Reg. 75,269/2 (Dec.17, 2014)[hereinafter Proposed Rule] ("more than 25 million people" have asthma, and, specifically, 9.5% of the 74 million children in the United States—more than 7 million—have asthma).

[8] U.S. EPA, Integrated Science Assessment for Ozone and Related Photochemical Oxidants (EPA/600/R-10/076F) (2013).

[9] C. DeNavas-Walt & D.B. Bernadette, U.S. Census Bureau, Current Population Reports, *Income and Poverty in the United States: 2013* (P60-249) 12 (2014), *available at* http://www.census.gov/content/dam/Census/library/publications/2014/demo/p60-249.pdf.

Instead, "EPA must explain why the evidence on which CASAC relied cannot support the degree of confidence CASAC placed in it. This is especially true given the added layer of stringency imposed by EPA's obligations under section 307(d)(6)." *Id.*

Even if CASAC makes a policy, rather than scientific, recommendation that EPA departs from, 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).

III. PROCEDURAL BACKGROUND

In 2009, after consultation with CASAC and the public, EPA revised the procedure for the NAAQS review process. The new, clearly defined process was used for the first time in this review cycle for the ozone NAAQS. The review was conducted over a period of five years, with public participation and peer review by CASAC at every step of the process. The chronology below demonstrates that the NAAQS review process undertaken for ozone was extraordinarily thorough, with many checks and balances.

One of the procedural changes incorporated into the revised review process is the convening of a scientific workshop at the beginning of the review to help frame the most significant scientific issues. More specifically, in October 2008, EPA issued a public call for new information regarding ozone. Later that month, EPA convened a workshop on the ozone NAAQS, inviting experts in the field to participate panel presentations on new scientific developments relevant to the review of the ozone NAAQS.

Based on the workshop discussions, EPA developed a draft Integrated Review Plan (IRP), in February 2009, laying out a plan and a schedule for the ozone NAAQS review, which was reviewed by CASAC and the public. EPA then developed an Integrated Science Assessment (ISA) as the next step in the review process. This entailed a thorough literature search and a rigorous assessment of the scientific literature. Prior to the publication of the first draft of the document, in August 2010, EPA convened a workshop with invited experts and the public to review the preliminary draft chapters.

EPA then published a first draft of the ISA in February 2011, and invited public comments on the document. CASAC likewise convened a two-day meeting in May 2011 to hear comments from the public and to review the draft document. The Committee developed extensive written comments to the Agency, recommending revisions to the draft document. EPA released a second draft ISA in September 2011. As usual, the agency received written and oral comments on the document from the public, and a detailed review by CASAC. During its in-depth peer review, CASAC recommended many changes in the document which necessitated development of a third draft ISA, which was issued in June 2012. Although not part of the original protocol, this additional step demonstrates that this was a fully open, public process that recognized and addressed the concerns of both independent scientists and public commenters. The final ISA of 1,250 pages was published in February 2013. More than two thousand studies

are cited in the bibliography for the ISA, including one thousand studies new to this review.

While EPA was in the process of drafting the ISA, it was also undertaking the Health Risk and Exposure Assessment (HREA) and Welfare Risk and Exposure Assessment (WREA). More specifically, after publication of the first draft ISA, in April 2011, EPA released a detailed plan for the development of the HREA and WREA. This plan was also subject to public comments and peer review by CASAC. In August 2012, EPA then released first drafts of the HREA, WREA, and the Policy Assessment (PA). These documents underwent extensive review by CASAC and the public. They were followed by a second draft HREA, WREA, and PA in January 2014, which were also subject to public comment and review by CASAC. The final HREA, WREA, and PA were finalized in August and published in September 2014.

All told, CASAC met 15 days through public meetings and public teleconferences to review draft versions of the various documents, including the IRP, the ISA, the HREA, the WREA, and the PA. The CASAC panel submitted hundreds of pages of comments, individually, and as a whole.

The review of the ozone NAAQS led to recommendations by CASAC and EPA staff scientists for revisions to the standards. For the primary, public health standard, the EPA scientific staff recommended a range of 60 to 70 ppb, while CASAC endorsed a range of 60 to somewhat below 70 ppb. Given the conclusions of CASAC, the Policy Assessment and the evidence before EPA, there is no reasoned basis for EPA's failure to specify 60 ppb as the low end of its proposed range. Indeed, as discussed below, all of the evidence indicates that the standard must be set at a level at least as protective as 60 ppb.

IV. THE EXTENSIVE AND ROBUST BODY OF SCIENTIFIC EVIDENCE ESTABLISHES A CLEAR NEED FOR A STANDARD OF 60 PPB IN ORDER TO PROTECT THE HEALTH OF ALL AMERICANS WITH AN ADEQUATE MARGIN OF SAFETY

A. Ozone is Recognized to Cause a Wide Range of Adverse Impacts to Human Health at Levels of 60 ppb

Exposure to ozone is connected to a wide range of significant human health impacts. Serious physiological effects result from both single incidents of exposure at high concentrations and from repeat exposures over time, even for healthy individuals and at relatively low concentrations. Adverse health effects including respiratory and cardiovascular morbidity, premature mortality, and central nervous system and developmental impacts have been demonstrated through controlled human exposure, epidemiologic, and toxicological studies.[10] While the impacts of acute ozone exposure are better understood, there is a growing body of scientific evidence showing long-lasting adverse impacts of chronic ozone exposure, which may be more severe and less reversible.

Exposure to ozone, both in the short-term (acute) and long-term (chronic), is known to

[10] *See generally* U.S. EPA (2013). Integrated Science Assessment for Ozone and Related Photochemical Oxidants (EPA/600/R-10/076F) [hereinafter ISA].

cause or exacerbate respiratory impacts such as breathing discomfort (e.g., coughing, wheezing, shortness of breath, pain upon inspiration), decreasing lung function and capacity, and lung inflammation and injury. Research on the relationship between ozone exposure and respiratory effects is well-documented and in fact, EPA's ISA made a conclusive determination that short-term exposure to ozone is responsible for adverse respiratory effects.[11] Studies have consistently demonstrated that exposure to relatively low concentrations of ozone is associated with lung function decrements, increases in respiratory symptoms, pulmonary inflammation in children with asthma, increases in respiratory-related hospital admissions and emergency department visits, and respiratory mortality. In addition, the ISA concludes there is a "likely causal" relationship between long-term exposure and adverse respiratory effects such as pulmonary inflammation and injury, new onset asthma, and respiratory mortality, and EPA finds an "overall strong body of evidence of adverse health effects."[12]

Ozone exposure is shown to result in respiratory tract inflammation and epithelial permeability. Inflammation can be considered evidence that injury has occurred.[13] Acute ozone exposure initiates an inflammatory response throughout the respiratory tract that has been observed to persist for at least 18-24 hours following the exposure.[14] This inflammation can evolve into a chronic inflammatory state and repeat episodes can alter the structure and function of tissues, leading to a "scarring" or "stiffening" of the lung tissue, such as pulmonary fibrosis. Lung tissue (epithelium or lining) may thus experience damage from chronic exposure to even relatively low levels of ozone. Inflammation can also alter the body's host defense response to inhaled microorganisms, particularly in sensitive groups, and responses to agents like allergens or toxins. Studies suggest that acute ozone exposure might impair lung host defense capability, resulting in a predisposition to bacterial infections in the lower respiratory tract.[15]

Short-term exposure to ozone results in bronchoconstriction—the tightening or narrowing of airways in the lungs—and in airway obstruction, causing coughing, wheezing, and shortness of breath. Ozone exposure has been shown to cause an increase in airway hyperresponsiveness, a condition in which the airways undergo enhanced bronchoconstriction.[16] Ozone-induced airway hyperresponsiveness results in a predisposition for bronchial narrowing upon inhalation of a variety of ambient stimuli. Symptoms have been demonstrated in both asthmatics and healthy individuals, although asthmatics are at higher risk due to already having greater airway inflammation and bronchial reactivity.

Ozone exposure harms lung function. As controlled human exposure studies and panel

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[11] *Id*. sec. 6.2.
[12] *Id*. at 1-5; U.S. EPA (2014). Policy Assessment for the Review of the Ozone National Ambient Air Quality Standards (EPA-452/R-14-006) 3-40 [hereinafter Policy Assessment].
[13] ISA at 6-76.
[14] *See* ISA sec. 6.2.3; A. Torres et al. (1997). Airway inflammation in smokers and nonsmokers with varying responsiveness to ozone, *Am. J. Respir. Crit. Care Med*., 156(3): 728-736; I.S. Mudway & F.J. Kelly (2004). An investigation of inhaled ozone dose and the magnitude of airway inflammation in healthy adults, *Am. J. Respir. Crit. Care Med.*, 169(10): 1089-1095.
[15] *See* ISA sec. 6.2.5.5.
[16] *See* ISA sec. 6.2.2; *see also* D.H. Horstman et al. (1990). Ozone concentration and pulmonary response relationships for 6.6-hour exposures with five hours of moderate exercise to 0.08, 0.10, and 0.12 ppm, *Am. J. Respir. Crit. Care Med*., 142(5): 1158-1163; R. Jörres, D. Nowak, & H. Magnussen (1996).The effect of ozone exposure on allergen responsiveness in subjects with asthma or rhinitis, *Am. J. Respir. Crit. Care Med.*, 153(1); 56-64.

studies demonstrate, respiratory responses to acute ozone exposure include decreased breathing capacity, rapid and shallow breathing, and painful inhalation. These changes are reported following exposures to relatively low ambient ozone concentrations, particularly in sensitive groups such as children and outdoor workers. Studies examining lung function decrements following outdoor activity show robust associations with ozone concentrations at 60 ppb and below[17] and even down to 40 ppb.[18] Early lung function deficits in children may lead to lower maximum lung function later in life, as well as to increased risk of respiratory disease, cardiovascular morbidity, and mortality.[19] For adults, chronic ozone exposure is tied to lasting declines in lung function and other respiratory effects.[20]

Not only is ozone exposure linked to the exacerbation of existing asthma, but also to new cases of the disease. Individuals with asthma are at greater risk for experiencing ozone-related health effects, especially children. Children living in areas with high ambient ozone concentrations were found in one study to be more likely to either have asthma or to experience asthma attacks compared to children living in areas with lower concentrations.[21] The relationship between asthma and ozone exposure is supported by evidence of increases in respiratory asthma medication use and asthma-related hospital and emergency room visits following exposure. Evidence also points to long-term exposure causing new-onset asthma. For adults, studies show increased risk for developing asthma with each 10 ppb increase in annual mean ozone or 8-hour average.[22] Not surprisingly, ozone is also connected to new onset asthma in children.[23]

An expanding body of research reveals causal relationships between ozone exposure and cardiovascular health.[24] Controlled human exposure studies document negative cardiovascular effects in response to short-term ozone exposure, including changes in heart rate variability and blood markers of systemic inflammation and oxidative stress, further supporting effects observed

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[17] B. Brunekreef, et al. (1994). Respiratory effects of low-level photochemical air pollution in amateur cyclists, *Am. J. of Resp. and Crit. Care Med.*, 150(4): 962-966; D.M. Spektor et al. (1988). Effects of ambient ozone on respiratory function in active, normal children, *Am. Rev. of Resp. Disease*, 137(2): 313-320; M.H. Gielen, S.C. van der Zee, J.H. van Wijnen, C.J. van Stehen, & B. Brunekreef (1997). Acute effects of summer air pollution on respiratory health of asthmatic children, *Am. J. Respir. Crit. Care Med*., 155(6): 2105-2108.

[18] M. Brauer, J. Blair, & S. Vedal (1996). Effect of ambient ozone exposure on lung function in farm workers, *Am. J. of Resp. and Crit. Care Med.*, 154(4): 981-987.

[19] R. Rojas-Martinez, et al. (2007). Lung Function Growth in Children with Long-Term Exposure to Air Pollutants in Mexico City, *Am. J. Respir. Crit. Care Med.*, 176(4): 377-384.

[20] A. Galizia & P.L. Kinney, Long-Term Residence in Areas of High Ozone: Associations with Respiratory Health in a Nationwide Sample of Nonsmoking Young Adults (1999). *Environ. Health Perspect*., 107(8): 675-679; N. Künzli et al. (1997). Association between Lifetime Ambient Ozone Exposure and Pulmonary Function in College Freshmen: Results of a Pilot Study, *Environ Res*., 72(1), 8-23; I.B. Tager, et al. (2005). Chronic Exposure to Ambient Ozone and Lung Function in Young Adults, *Epidemiology*, 16(6): 751-759.

[21] L.J. Akinbami, C.D. Lynch, J.D. Parker, & T.J. Woodruff (2010). The association between childhood asthma prevalence and monitored air pollutants in metropolitan areas, United States, 2001-2004, *Environ Res*., 110(3): 294-301.

[22] W.F. McDonnell, D.E. Abbey, N. Nishino, & M.D. Lebowitz (1999). Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the AHSMOG study, *Environ. Res*., 80(2): 110-121; J. Greer, D.E. Abbey, & R.J. Burchette (1993). Asthma related to occupational and ambient air pollutants in nonsmokers, *J. Occup. Environ. Med.*, 35(9): 909-915.

[23] *See e.g.*, R. McConnell et al. (2002). Asthma in exercising children exposed to ozone: A cohort study, *Lancet*, 359(9304): 386-391.

[24] *See* ISA sec. 6.3 and 7.3.

in toxicological studies.[25] Ozone exposure is shown to be associated with increased risks of heart attacks, coronary atherosclerosis, stroke, and heart disease, even at very low concentrations; one study showed positive correlations between these impacts and mean concentrations of 25 ppb and maximum concentrations of only 40.2 ppb.[26] More new studies link ozone exposure to increased risk for heart attacks[27] and stroke incidents.[28] Chronic ozone exposure may put children at risk for cardiovascular disease later in life and young adults growing up in areas with higher ozone concentrations have shown a tendency towards early atherosclerotic (hardening of the arteries).[29]

Short- and long-term ozone exposure has been linked to premature mortality.[30] Epidemiological studies show a strong relationship between short-term ozone exposure and premature mortality. The ISA describes numerous studies across the U.S., Canada, and Europe that link ambient ozone concentrations with respiratory mortality, finding that on average it occurs at mean 8-hour maximum concentrations of less than 63 ppb.[31] In one key study of 98 U.S. cities, mean concentrations of only 26.8 ppb were associated with mortality and a 10 ppb increase in the prior week's ozone level increased mortality by 0.52 percent.[32] Higher risks were also associated with factors such as race and socioeconomic status. Another large U.S. cohort study showed a significant increase in the risk of death from respiratory causes in association with long-term exposure to ozone.[33] Some studies have shown relationships between long-term ozone exposures and cardiopulmonary mortality as well.[34]

Other health impacts linked to ozone exposure are related to newborns and developing

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[25] R.B. Devlin et al. (2012). Controlled exposure of healthy young volunteers to ozone causes cardiovascular effects, *Circulation*, 126(1):104-111; H. Gong et al. (1998). Cardiovascular effects of ozone exposure in human volunteers, *Am. J. Respir. Crit. Care Med.*, 158(2): 538-546; L. Liu (1999). A comparison of biomarkers of ozone exposure in human plasma, nasal lavage, and sputum, *Inhalation Toxicology*, 11(8), 657-674.

[26] P. Koken et al. (2003). Temperature, air pollution, and hospitalization for cardiovascular diseases among elderly people in Denver, *Environ Health Perspec*. 111(10): 1312-1317.

[27] K.B. Ensor, L.H. Raun, & D. Persse (2013). A Case-Crossover Analysis of Out-of-Hospital Cardiac Arrest and Air Pollution, *Circulation*, 127(11):1192-1199; S. von Klot et al. (2005). Ambient Air Pollution is Associated with Increased Risk of Hospital Cardiac Readmissions of Myocardial Infarction Survivors in Five European Cities, *Circulation* , 112(5): 3073-3079; J. Ruidavets et al. (2005). Ozone Air Pollution is Associated with Acute Myocardial Infarction, *Circulation*, 111(5): 563-569.

[28] J.B. Henrotin, J.P. Besancenot, Y. Bejot, & M. Giroud (2007). Short-term effects of ozone air pollution on ischaemic stroke occurrence: a case-crossover analysis form a 10-year population-based study in Dijon, France, *Occup. Environ. Med*., 64(7):4439-445; D.Q. Rich et al. (2006). Increased Risk of Paroxysmal Atrial Fibrillation Episodes Associated with Acute Increases in Ambient Air Pollution, *Environ. Health Perspec*., 114(1):120-123.

[29] C.V. Breton et al. (2012). Childhood air pollutant exposure and carotid artery intima-media thickness in young adults, *Circulation*, 126(13): 1614 –1620; S.D. Adar (2012). Childhood exposures to ozone: the fast track to cardiovascular disease?, *Circulation,* 126(13):1570-1572.

[30] The ISA concludes that there is a "likely causal" relationship between short-term ozone increases and total mortality. Chronic ozone exposure was "suggestive of a causal relationship" with premature mortality.

[31] ISA at 2-22.

[32] M.L. Bell & F. Dominici (2008). Effect modification by community characteristics on the short-term effects of ozone exposure and mortality in 98 US communities, *Am. J. of Epidem.*, 167(8): 986-997.

[33] M. Jerrett et al. (2009). Long-term ozone exposure and mortality, *New Eng. J. of Med.*, 360(11): 1085-1095.

[34] K.R. Smith et al. (2009). Public health benefits of strategies to reduce greenhouse-gas emissions: Health implications of short-lived greenhouse pollutants, *Lancet*, 374(9707): 2091-2103; A. Zanobetti & J. Schwartz (2011). Ozone and Survival in Four Cohorts with Potentially Predisposing Diseases, *Am. J. Respir. Crit. Care Med*., 184: 836-841.

fetuses.[35] Although research on these effects is less developed, existing evidence presents serious public health concerns and additional support for enacting a lower primary standard that protects public health and provides a precautionary margin of safety. Prenatal ozone exposure has been linked to reduced birth weight, premature delivery, and birth defects. Studies have observed associations between lower birth weight and intrauterine growth retardation and prenatal ozone exposure.[36] Prenatal exposure to elevated ozone concentrations has also been associated with premature birth[37] and with birth defects in some studies.[38] The effects of pre-natal ozone exposure may persist after birth. One study of eight U.S. cities with mean 8-hour concentrations of only 48 ppb showed that children born prematurely or with low birth weight are more susceptible to ozone-related health impacts.[39] Another recent study demonstrated that prenatal ozone exposure leads to increased need for health care, with the costs of health care in the first days after birth increasing by $964 per unit (ppm).[40]

Finally, new research is suggestive of a relationship between ozone exposure and effects on the central nervous system. [41] Studies show that acute ozone exposure may be linked to alterations in neurotransmitters, motor activity, short- and long-term memory, sleep patterns, and signs of neurodegeneration.

B. A Number of Subpopulations Are Particularly Susceptible to Exposure to Ozone, Including Children

As noted above, people with asthma and other respiratory or pulmonary health conditions are particularly susceptible to exposure to ozone and can have more severe reactions to lower levels of ozone than the general population. However, other especially sensitive subpopulations exist as well. These are discussed separately below.

1. Scientific Studies Establish Children Are Highly Susceptible to Ozone And Clear Risks to Children's Health Occur at Ozone Levels of 60 ppb

[35] Finding greater evidence than in the last review, the ISA concludes that research is "suggestive of a causal relationship" between long-term exposures to ozone and reproductive and developmental effects.
[36] M.T. Salam et al. (2005). Birth Outcomes and Prenatal Exposure to Ozone, Carbon Monoxide, and Particulate Matter: Results from the Children's Health Study, *Environ. Health Perspec*. 113(11): 1638-1644; R. Morello-Frosch, B.M. Jesdale, J.L. Sadd, & M. Pastor (2010). Ambient air pollution exposure and full-term birth weight in California, *Environ. Health*, 9(44).
[37] C. Jansen, A. Neller, G. Williams, & R. Simpson (2006). Maternal exposure to low levels of ambient air pollution and preterm birth in Brisbane, Australia, *BJOG*, 113(8): 935-941; B. Jalaludin et al. (2007). Impact of ambient air pollution on gestational age is modified by season in Sydney, Australia, *Environ. Health*, 6, 16 (2007).
[38] B. Ritz et al. (2002). Ambient Air Pollution and Risk of Birth Defects in Southern California, *Am. J. Epidem.*, 155(1): 17-25; S.M. Gilboa et al. (2005). Relation between ambient air quality and selected birth defects, seven county study, Texas, 1997-2000, *Am. J. Epidemiol* .,162(3): 238-252; B. Hwang, & J. Jaakkola (2008). Ozone and other air pollutants and the risk of oral clefts, *Environ. Health Perspect.*, 116(10): 1411-1415.
[39] K.M. Mortimer, I.B. Tager, D.W. Dockery, L.M. Neas, & S. Redline (2000). The effect of ozone on inner-city children with asthma: identification of susceptible subgroups, *Am. J. Respir. Crit. Care Med.*, 162(5): 1838-1845.
[40] L. Trasande et al. (2013). Exploring prenatal outdoor air pollution, birth outcomes and neonatal health care utilization in a nationally representative sample, *J. Expo. Sci. Environ. Epidemiol*., 23(3): 315-21.
[41] *See* ISA sec. 7.5.

One of the subpopulations most susceptible to ozone is children. The more than 73 million children under age 18 comprise nearly one-fourth of the U.S. population--23.2 percent. Of these, nearly 20 million children are under age 5.[42] Children's singular vulnerability to air pollution stems from their developmental stage and their behavior. Ongoing lung growth and development, higher relative ventilation rates, and high levels of outdoor activity mean that children face unique health risks from air pollution exposure.[43] It is well established that children cannot simply be treated as small adults and require extra protection from the harmful effects of air pollution[44] - beyond what EPA has proposed.

Children's respiratory systems are undergoing critical stages of development that places them at greater risk for ozone-induced damage.[45] In fact, most of a child's lungs will develop after birth until adolescence, including 80% of alveoli, the air sacs that transfer oxygen to the blood.[46] Children's lungs have larger surface area per kilogram of body weight compared to adults.[47] As described by EPA, "[c]hildren are considered to be at greater risk from O3 exposure because their respiratory systems undergo lung growth until about 18-20 years of age and are therefore thought to be intrinsically more at risk for O3-induced damage."[48] In other words, children's lungs continue to grow until they reach adulthood, during which time they are more vulnerable to damage. Children's immune systems are also still developing, making them more susceptible to infection and respiratory illness than adults.[49]

Children have higher baseline ventilation rates relative to lung volume and breathe in more air per pound of body weight.[50] As a result, they take in higher relative doses of air pollutants than adults do. Children also tend to have a greater oral breathing contribution than adults, i.e., are mouth breathers, further increasing pollutant intake.[51]

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[42] U.S. Census Bureau (2014). Population Division, Annual Estimates of the Resident Population for Selected Age Groups by Sex for the United States, States, Counties, and Puerto Rico Commonwealth and Municipios: April 1, 2010 to July 1, 2013.

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

[44] World Health Organization (2005). The Effects of Air Pollution on Children's Health and Development: a review of the evidence E86575, *available at* http://www.euro.who.int/document/E86575.pdf.

[45] *See e.g.*, T.F. Bateson & J. Schwartz (2007). Children's response to air pollutant, *J. Toxicol. Environ. Health,* 71(3): 238-243; C.G. Plopper & M.V. Fanucchi (2000). Do urban environmental pollutants exacerbate childhood lung diseases?, *Environ. Health Perspect.*, 108(6), A252–A253; L. Trasande & G.D.Thurston (2005). The role of air pollution in asthma and other pediatric morbidities, *J Allergy Clin. Immunol.*. 115(4): 689-699.

[46] R.R. Dietert et al. (2000). Workshop to Identify Critical Windows of Exposure for Children's Health: immune and respiratory systems workgroup summary, *Environ Health Perspect.*, 108(supp 3), 483-490; *see also* J. Schwartz (2004). Air pollution and children's health, *Pediatrics*, 113(4): 1037-1043; M. Dunnil (1962). Postnatal growth of the lung, *Thorax*, 17, 329-333.

[47] Schwartz 2004, *supra* note 45.

[48] Policy Assessment at 3-81.

[49] Dietert et al. 2000, *supra* note 45; Bateson & Schwartz 2007, *supra* note 44.

[50] U.S. EPA (2011). Exposure Factors Handbook: 2011 Edition (EPA/600/R-090/052F); Wolf (ed.) (2000). Indoor Air Pollutants Affecting Child Health. American College of Medical Toxicology; ISA Table 4-5.

[51] W.D. Bennett, K.L. Zeman, & A.M. Jarabek (2008). Nasal contribution to breathing and fine particle deposition in children versus adults, *J. Toxicol. Environ. Health A.*, 71(3): 227-237; U.S. EPA 2011, *supra* note 49; Wolf 2000, *supra* note 49.

In addition to all of these physiological factors that make children more vulnerable to ozone exposure, they are more likely to be active outdoors. [52] Through sports, school, and play, children's outdoor activities increase their exposure to ozone pollution and, correspondingly, increase the likelihood that they will suffer adverse ozone-related health impacts.[53] Children spend more time outdoors during midday and afternoons when pollutant levels tend to be higher. High intensity activities increase ventilation rates and pollution inhalation.[54] This extra vulnerability is not lost on EPA, which found that "the percentages of children estimated to experience exposures of concern is considerably larger than the percentages estimated for adult populations (i.e., approximately 3-*fold larger across urban study areas*)."[55] Playing outside and participating in sports and activities are important parts of growing up and encouraging healthy, active, and well-balanced lifestyles. Instead of forcing them to be shuttered indoors to avoid damage to their sensitive, developing bodies, we should be creating a safe, clean environment where our kids can be kids.

The evidence showing children's physiological vulnerability to ozone exposure is strong. A sizeable and growing body of scientific studies demonstrates serious health harms from exposure to ozone concentrations at or below 60 ppb, including respiratory impacts, decreased pulmonary function, impaired lung development, new asthma onset, and increased respiratory-related hospitalization and emergency room visits. Looking at infants, a Virginia study found that they were at greater risk for respiratory symptoms when exposed to ozone, even with mean 8-hour maximum concentration as low as 54.5 ppb and only exceeding 80 ppb twice during the study period.

These panel studies demonstrate health impacts to children at ambient concentrations below what EPA proposes for the new standard. Additional studies of children at summer camp, where a great deal of time is spent outdoors and being active, consistently show that increasing levels of ozone are associated with diminished lung function. [56] Children ages 8-15 at a summer camp in New Jersey showed decreases in lung function associated with ozone exposure, even when excluding concentrations above 60 ppb.[57]

Evidence indicates that, for some children, ozone may contribute to their development of incident asthma. A significant cohort study in California showed that healthy, active children playing three or more sports and growing up in communities with ambient ozone levels ranging from 55.8 to 69 ppb were over three times more likely to develop asthma than their peers in

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[52] Proposed Rule at 75,267: "It is generally recognized that children spend more time outdoors than adults, and, therefore, would be expected to have higher exposure to O3 than adults"; *see also* U.S. EPA (2011). Health Risk and Exposure Assessment for Ozone (EPA-452/R-14-004a), 2-18 [hereinafter HREA].

[53] HREA at 5-11:"Due to the increased amount of time spent outdoors engaged in relatively high levels of physical activity (which increases intake), school-age children as a group are particularly at risk for experiencing O3-related health effects."

[54] *See* ISA at 4-31.

[55] Proposed Rule at 75,285 (emphasis added).

[56] *See* P.L. Kinney, G.D. Thurston, & M. Raizenne (1996).The effects of ambient ozone on lung function in children: A reanalysis of six summer camp studies, *Env. Health Perspect.*, 104(2): 170-174; G.D. Thurston, M. Lippmann, M.B. Scott, & J.M. Fine (1997). Summertime haze air pollution and children with asthma, *Am. J. of Resp. and Crit. Care Med.*, 155(2): 654-660.

[57] Spektor et al. 1988, *supra* note 16.

communities with ozone levels ranging from 30.6 to 50.9 ppb.[58] None of the high ozone communities would have violated a primary standard in the upper range of what EPA now proposes. Similarly, another noteworthy California study found that a 5 ppb increase in annual average 8-hour ozone concentrations resulted in positive associations with children having asthma and having asthma attacks, with maximum average levels of only 59.5 ppb.[59] A study of Medicaid children in Harris County, Texas investigated the effect of increased short-term ozone, $PM_{2.5}$ and nitrogen dioxide concentrations on the timing of asthma onset. Each 10 ppb increase in ozone was significantly associated with new-onset asthma during the warm season. The results indicated that among children who developed asthma, their initial date of diagnosis was more likely to occur following periods of higher short-term ambient pollutant levels.[60] These studies suggest that even if communities are able to meet ozone standards within the range and form that EPA is proposing, more children would face greater risk for developing asthma compared to communities meeting a standard set at 60 ppb or below.

Children with asthma have repeatedly been shown to suffer from both lung function decrements and respiratory symptoms.[61] Even children without asthma are suggested to be at higher risk for of respiratory-related pulmonary impacts than adults.[62]

An Atlanta study looked at emergency department visits for children under 4-years-old and found that each 30 ppb increase in the 3-day average of ozone was associated with an 8 percent higher risk of pneumonia and 4 percent higher risk of upper respiratory infection.[63] Another Atlanta study of children ages 5-17 found that children are particularly at risk for ozone-induced respiratory impacts and saw a 6.4 percent increase in emergency room visits for a 30 ppb increase in 8-hour concentrations. Mean annual 8-hour concentrations were only 47.3 ppb and a dose-response relationship between ozone exposure and emergency room visits for children was evident at concentrations as low as 30 ppb.[64]

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[58] R. McConnell et al. (2002). Asthma in exercising children exposed to ozone: A cohort study, *Lancet*, 359(9304): 386-391.

[59] L.J. Akinbami, C.D. Lynch, J.D. Parker, & T.J. Woodruff (2010). The association between childhood asthma prevalence and monitored air pollutants in metropolitan areas, United States, 2001-2004, *Environ Res*., 110(3): 294-301.

[60] J.K. Wendt, E. Symanski , T.H. Stock, W. Chan, & X.L. Du (2014). Association of short-term increases in ambient air pollution and timing of initial asthma diagnosis among Medicaid-enrolled children in a metropolitan area. *Environ. Res.*, 131: 50-8.

[61] J. Just et al., Short-term health effects of particulate and photochemical air pollution in asthmatic children, *Eur. Resp. J.*, 20(4), 899-906 (2002); Mortimer et al. 2000, *supra* note 38; M.A. Rosset al (2002). Effect of ozone and aeroallergens on the respiratory health of asthmatics, *Arch. of Env. Health*, 57(6): 568-578; Thurston et al. 1997, *supra* note 55; I. Romieu et al. (1997). Effects of intermittent ozone exposure on peak expiratory flow and respiratory symptoms among asthmatic children in Mexico City, *Arch. of Env. Health*, 52(5): 368-376.

[62] *See e.g.*, P. Höppe et al. (2003). Environmental ozone effects in different population subgroups, *Int. J. of Hygiene and Env. Health*, 206(6): 505-516; *see also* ISA at 6-61: "evidence suggests that the ambient O3-associated lung function decrements found in children overall were not solely due to effects in children with asthma, and that increases in ambient O3 exposure may decrease lung function in healthy children."

[63] L.A. Darrow, M. Klein, W.D. Flanders, J.A. Mulholland, P.E. Tolbert, & M.J. Strickland (2014). Air Pollution and Acute Respiratory Infections Among Children 0-4 Years of Age: An 18-Year Time-Series Study, *Am. J. Epidemiol.*, 180(10): 968-77.

[64] M.J. Strickland et al. (2010). Short-term associations between ambient air pollutants and pediatric asthma emergency department visits, *Am. J. of Resp. and Crit. Care Med.*, *182*(3): 307-316.

A number of new studies released since EPA's ISA was published show that rising ambient ozone concentrations are associated with increased hospitalizations and emergency room visits for children.[65] Indeed, the *ISA* already concluded that chronic ozone exposure is associated with childhood asthma hospital admissions in multiple studies with mean annual 8-hour maximum ozone concentrations of less than 41 ppb.[66] There is also evidence that chronic exposure to ozone may be damaging to children's health later in life. For instance, a study of freshman university students in California found that lifetime exposure to high ambient ozone levels was associated with decreased airway function.[67]

For ethical reasons, controlled human exposure studies are generally performed on healthy adults, not children – thus resulting in a lack of exposure study data for children. That understandable lack of controlled exposure data has led to undervaluing risks to children by treating them like adults in considering the available controlled exposure data. EPA largely relies on a single study from 1985 with a limited sample size of 22 children, along with modeling and opaque references to summer camps studies, to assert that "children exhibit the same lung function responses following O3 exposures as healthy 18 year olds."[68] At the same time, EPA, agreeing with CASAC, says that"[c]ompared to the healthy individuals included in controlled human exposure studies, members of at-risk populations (e.g., asthmatics, children) could be more likely to experience adverse effects, could experience larger and/or more serious effects, and/or could experience effects following exposures to lower O3 concentrations."[69] Given CASAC's and EPA's recognition of the clear evidence that children experience greater sensitivity to and impacts from exposure to ozone, EPA's assessments need to place greater relative value on epidemiological studies of children.

As multiple studies cited above demonstrate, reducing ozone concentrations can reduce respiratory morbidity for children. CASAC has stated that exposures of 70 ppb "are of significant concern, especially for children, asthmatics, the elderly and other at risk populations."[70] Given the substantial evidence of impacts to health resulting at 8-hour concentrations below 65 ppb, it is inconceivable that a standard of 70 ppb or even 65 ppb would be protective of children. Indeed, it is questionable whether even a 60 ppb standard would protect children. Given the requirement to err on the side of caution, EPA should set a primary standard that does not exceed 60 ppb.

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[65] *See e.g.*, J.A. Gleason, L. Bielory, & J.A. Fagliano (2014). Associations between ozone, PM2.5, and four pollen types on emergency department pediatric asthma events during the warm season in New Jersey: a case-crossover study, *Environ. Res.*, 32: 421-429; Darrow, *supra* note 62; M.J. Strickland, M. Klein, W.D. Flanders, H.H. Chang, J.A. Mulholland, P.E. Tolbert, & L.A. Darrow (2014). Modification of the effect of ambient air pollution on pediatric asthma emergency visits: susceptible subpopulations, *Epidemiology*, 25(6): 843-850.

[66] ISA at 2-23.

[67] Tager et al. 2005, *supra* note 19.

[68] Proposed Rule at 75,248, n. 28; *see also id.* at 75,275 ("In the near absence of controlled human exposure data for children, risk estimates are based on the assumption that children exhibit the same lung function response following O3 exposures as healthy 18 year olds (i.e., the youngest age for which controlled human exposure data is available).").

[69] *Id.* at 75,273.

[70] Letter from CASAC Chair Dr. H. Christopher Frey to U.S. EPA Administrator Gina McCarthy re: *Second Draft Policy Assessment for the Review of the Ozone National Ambient Air Quality Standards* (EPA-CASAC-14-004), 7, June 26, 2014 [hereinafter CASAC Letter 2014a].

A focus on children is particularly appropriate because, as EPA acknowledges, "the percentages of children estimated to experience exposures of concern is considerably larger than the percentages estimated for adult populations (i.e., approximately 3-fold larger across urban study areas)."[71] Thus, not only are children physiologically more sensitive, but they also more often experience levels of ozone pollution that health science indicates is likely to result in adverse health impacts.

2. Asthmatics Are a Large Subpopulation Highly Sensitive to Ozone, Particularly Asthmatic Children

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Asthma is a highly prevalent disease in the United States. About 1 in 12 people, or about 25 million people, have asthma.[72] That represents approximately 8 percent of adults, or 18.7 million adults, and 9.3 percent of children, or 6.8 million children. [73] The Centers for Disease Control and Prevention (CDC) estimate that each year there are 14.2 million visits to physician offices, 1.3 million visits to hospital outpatient departments, and 1.8 million visits to emergency departments with asthma as primary diagnosis, and an estimated 3,600 deaths each year from asthma.[74]

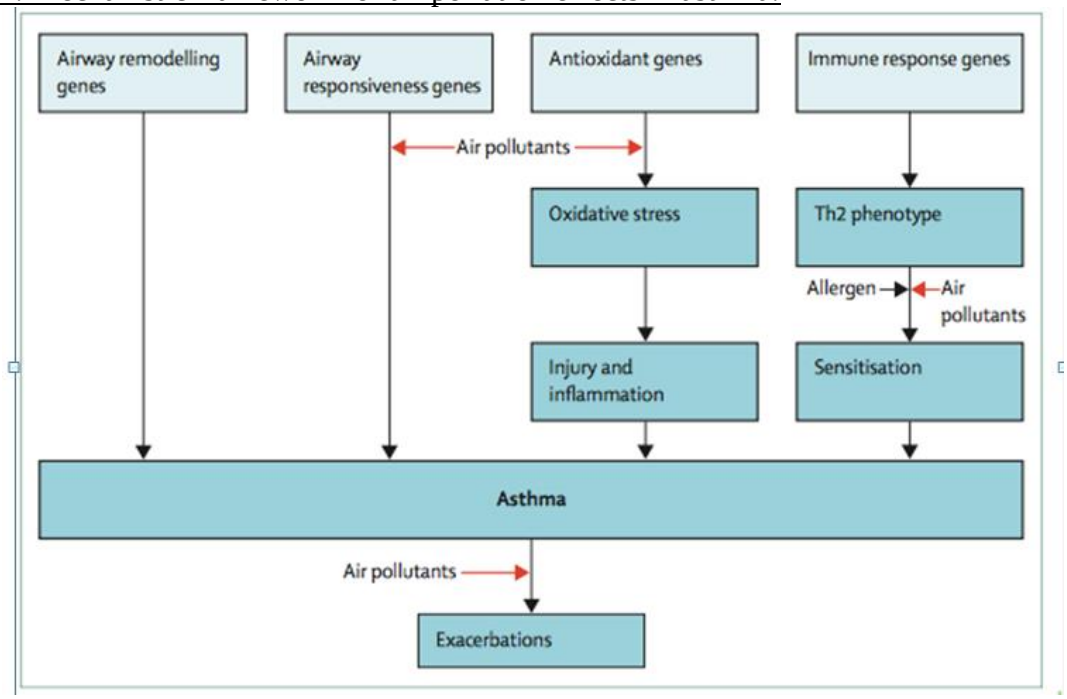
Air pollution is known to trigger asthma exacerbations and is associated with asthma symptoms, airway inflammation, airway hyperresponsiveness, decreased lung function, and reduced response to asthma rescue medications. Controlled studies have found that asthmatics experience twice the decrement in lung function of healthy people when exposed to the same level of ozone.[75] As a 2014 review article explained in summarizing the evidence:

> The idea that outdoor air pollution can cause exacerbations of pre-existing asthma is supported by an evidence base that has been accumulating for several decades, with several studies suggesting a contribution to new-onset asthma as well.[76]

The article presented a mechanistic framework, shown below, for the effects of air pollution, including ozone, in asthma:

[71] Proposed Rule at 75,272.
[72] CDC, Asthma in the US, *available at* http://www.cdc.gov/VitalSigns/asthma (accessed Mar. 15, 2015).
[73] CDC (2012). National Health Interview Survey.
[74] *Id.*
[75] D.H. Horstman, B.A. Ball, J. Brown, T. Gerrity, & L.J. Folinsbee (1995). Comparison of pulmonary responses of asthmatic and nonasthmatic subjects performing light exercise while exposed to a low level of ozone. *Toxicol Ind Health*, 11 (4): 369-85.
[76] M. Guarnieri & J.R. Balmes (2014). Outdoor air pollution and asthma, *Lancet*, 383 (9928): 1581-92.

Figure 1. Mechanistic framework for air pollution effects in asthma.[77]



The sensitivity of asthmatics to ozone exposure is particularly true for children. Asthma is the most common chronic disorder in children. As noted earlier, in the most recent national survey by the CDC, in 2012, 6.8 million children reported having asthma.[78] Younger children are particularly affected. Almost half of all children have at least one episode of wheeze before age six. 48 percent of preschool children with asthma have suffered an asthma attack in the preceding year -- a rate higher than for any other age group.[79]

Young children with asthma have long been regarded as a group who are very susceptible to adverse effects from air pollution because of their developing lungs, immature metabolic pathways, high ventilation rates per body weight, and increased times exercising outdoors. Even exposures in utero might affect postnatal risk of asthma and asthma exacerbations. Low birthweight, which might be associated with narrow airways during early childhood, is a risk factor from symptoms of asthma related to air pollution.[80]

[77] *Id.*
[78] CDC 2012, *supra* note 72.
[79] Guarnieri & Balmes 2014, *supra* note 75.
[80] *Id.*

In part, asthmatic children are more highly sensitive to ozone because of the early stage of the physiological development of their lungs. As explained by Guamieri and Balmes:

> Children appear to be most vulnerable to the harmful effects of ambient air pollutants. As their lungs are not completely developed, children may experience greater exposure to environmental pollutants than adults and the higher doses of varied composition may remain in their lungs for a greater duration. Altogether, the negative effects of air pollutants on pulmonary function place children at a greater risk of air pollutant-induced exacerbation of asthma for the duration of their lives. [81]

Indeed, there numerous studies tying increased air pollution to asthma onset and to triggering attacks among asthmatics. Children with asthma have repeatedly been shown to suffer from both lung function decrements and respiratory symptoms.[82] Even children without asthma are suggested to be at higher risk for of respiratory-related pulmonary impacts than adults.[83]

Studies have documented respiratory symptoms in children with asthma such as chest tightness, persistent cough, and shortness of breath, even at prior-day levels of only 52.1 ppb.[84] An eight-city U.S. study of inner-city children with asthma found that adverse respiratory effects were experienced by children across all cities, which together had a mean 8-hour average ozone concentration of only 48 ppb.[85] Ozone was found to be associated with significant declines in pulmonary function and asthma symptoms in the children examined. Another analysis of the same group found that even when excluding the 5 percent of days where ambient concentrations exceeded 80 ppb, results were nearly identical.[86] Another key study showed lung decrements in children ages 7-13 following exposure to 8-hour concentrations of less than 51 ppb.[87] Still another study of French children with asthma revealed both decreases in lung function and respiratory symptoms (asthma attacks and respiratory infections) at maximum concentrations of 61.7 ppb.[88]"

There is also evidence that long-term, chronic ozone exposure may be associated with children's hospital visits. For example, a New York State study showed that long-term ozone

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[81] L. Tzivian (2011). Outdoor air pollution and asthma in children, *J. Asthma,* 48 (5): 470-81.

[82] Just et al. 2002, *supra* note 60; Mortimer et al. 2000, *supra* note 38; M.A. Ross. et al. (2002). Effect of ozone and aeroallergens on the respiratory health of asthmatics, *Arch. of Env. Health*, 57(6): 568-578; Thurston, G.D., Lippmann, M., Scott M.B., & Fine, J.M. (1997). Summertime haze air pollution and children with asthma, *Am. J. of Resp. and Crit. Care Med.*, *155*(2), 654-660; I. Romieu et al. 1997, *supra* note 60.

[83] *See e.g.,* P. Höppe et al., *supra* note 66; *see also* ISA at 6-61: "evidence suggests that the ambient O3-associated lung function decrements found in children overall were not solely due to effects in children with asthma, and that increases in ambient O3 exposure may decrease lung function in healthy children."

[84] J.F. Gent et al. (2003). Association of Low-Level Ozone and Fine Particles with Respiratory Symptoms in Children with Asthma. *JAMA*, *290*(1): 1859-1867.

[85] Mortimer et al. 2000, *supra* note 38.

[86] K.M. Mortimer, L.M. Neas, D.W. Dockery, S. Redline, & I.B. Tager (2002). The effect of air pollution on inner-city children with asthma. *Eur. Respir. J.*, 19(4): 699–705.

[87] M.H. Gielen, S.C. van der Zee, J.H. van Wijnen, C.J. van Stehen, & B. Brunekreef (1997). Acute effects of summer air pollution on respiratory health of asthmatic children, *Am. J. Respir. Crit. Care Med*., 155(6): 2105-2108.

[88] Just et al. 2002, *supra* note 60.

exposure was associated with first asthma hospital admissions for children 1-to 6-years-old. [89] Thus, children exposed to chronically high ozone levels were more likely to develop asthma exacerbations so severe as to require hospitalization. Just a 1 percent increase in mean ozone concentrations during the ozone season increased the risk of a child's hospital admission by 22 percent. Notably, ozone levels weren't actually all that high - annual mean 8-hour concentrations were only 41.06 ppb and only 50.62 ppb during the ozone season. Younger children and those in low socioeconomic groups showed a greater risk of asthma hospitalization than did other children at the same ozone levels.

A study of primarily African-American, Latino, and lower-income children with asthma in Detroit, Michigan explored the question of how ambient air pollution affects susceptible populations. Corticosteroid use was used as a marker of more severe asthma. Daily one-hour maximum ozone concentrations were associated with increased odds of respiratory symptoms, particularly among children using corticosteroid medication and among children living in the southwest community of Detroit. Similar patterns of associations were not seen with PM. Researchers concluded that ozone at levels near or below annual standard levels are associated with negative health impact in this population of asthmatic children.[90] Researchers in Pittsburgh, Pennsylvania used a case-crossover approach to investigate the relationship of air pollution and emergency department visits in an urban population. A 2.5 percent increase was observed in asthma emergency department visits for each 10 ppb increase in the 1-hour maximum ozone level on day two. [91]

The relationship between ozone and respiratory impacts in children is further supported by connections between ozone exposure and respiratory-related hospital admissions and emergency room visits, as well as increased asthma medication use. Epidemiological studies linking ozone exposure to respiratory-related hospitalizations and emergency room visits for children offer important evidence of some of the more severe impacts that ozone has on children's health and well-being.

For instance, a New York City study showed stronger associations between ozone exposure and asthma-related hospital admissions for 6- to 18-year-olds, whose lungs are still developing, compared with other age groups.[1] Another study in Seattle demonstrated higher asthma hospitalizations for children under age 18 than for adults, which the authors found "suggests that children are more immediately responsive to adverse effects of O3 exposure."[1] In it, a 30 ppb increase in 8-hour maximum ozone concentrations was associated with a 19.1-36.8 percent increase in asthma emergency-department visits for children. A Canadian study also showed stronger associations between asthma-related emergency room visits and ozone exposure in 5- to 14-year-olds than for any other age group.[92] A study of children with asthma under age 12 in Connecticut and Massachusetts found that ozone levels were significantly associated with

[89] S. Lin, X. Liu, L.H. Le, & S. Hwang (2008). Chronic exposure to ambient ozone and asthma hospital admissions among children, *Env. Health Perspect.*, 116(12): 1725-1730.
[90] T.C. Lewis et al. (2013). Air pollution and respiratory symptoms among children with asthma: vulnerability by corticosteroid use and residence area, *Sci.Total Environ.*, 448: 48-55.
[91] J.A. Glad et al. (2012). The relationship of ambient ozone and PM(2.5) levels and asthma emergency department visits: possible influence of gender and ethnicity, *Arch Environ Occup Health*, 67(2), 103-8.
[92] Villeneuve et al. (2007). Outdoor air pollution and emergency department visits for asthma among children and adults: A case-crossover study in northern Alberta, Canada, *Env. Health*, 6(40).

respiratory symptoms and rescue medication use among children.[93] Maximum eight-hour concentrations of only 63.3 ppb were associated with a 30 percent increase in chest tightness, while previous day levels of 52.1 ppb or higher were associated with increased chest tightness, persistent cough, and shortness of breath.

New studies provide extensive further evidence that people with respiratory disease are at increased risk, above that faced by the general population. Critical new evidence since the last review correlates exposure to ozone with respiratory symptoms, increased airway responsiveness, school absenteeism, and increased medication use in people with asthma.

A study sought to establish the prevalence of "responders" in four different population subgroups: children, asthmatics, the elderly, and athletes, by assessing symptoms and measuring respiratory function.[94] The study found higher rates of ozone responders in asthmatics (21 percent) and children (18 percent), as compared to the elderly and athletes (both 5 percent). This means that children and asthmatics have a higher risk of being ozone sensitive and experiencing more acute lung function decrements than these other population groups. This study indicates that individuals with asthma are more sensitive to the effects of low-level ozone exposures than healthy persons.

Important new evidence of the increased sensitivity of children with asthma comes from two studies by Mortimer et al.[95] The effects of daily ambient air pollution were examined in a cohort of 864 asthmatic children in 8 urban areas of the U.S. in a longitudinal study. The cities studied were Baltimore, Chicago, Cleveland, Detroit, Bronx/East Harlem, St. Louis, and Washington D.C. Eight-hour average ozone concentrations from 10 a.m. to 6 p.m. were 48 ppb. Median concentrations across cities ranged from 34 to 58 ppb. Researchers found that summertime air pollution at levels below the current air quality standards was significantly related to symptoms and decreased pulmonary function in children with asthma. Ozone was most influential on peak expiratory flow rate. Adverse respiratory effects were observed in all cities. This compelling study provides strong support for an 8-hour ozone standard of 60 ppb or below.

Another study of the same cohort found that asthmatic children born prematurely or with low birth weight have the greatest response to ozone.[96] Scientists sought to ascertain which subgroups in a cohort of 846 inner-city asthmatic children aged 4-9 years old were most susceptible to the effects of summertime ozone. Children were recruited from emergency departments and primary care clinics the eight U.S. cities. The mean 8-hour ozone concentrations from 10 a.m. to 6 p.m. across these cities were 48 ppb. The study reported that "children of low birth weight or of premature birth are at greater risk for respiratory problems, and appear to be substantially more susceptible to the effects of summer air pollution than children of normal birth weight or full-term gestation."

[93] Gent, J.F., et al. (2003). Association of Low-Level Ozone and Fine Particles with Respiratory Symptoms in Children with Asthma. *JAMA*, *290*(1)*:* 1859-1867

[94] P. Höppe et al. (2003). Environmental ozone effects in different population subgroups, *Int. J. of Hygiene and Env. Health*, 206(6): 505-516.

[95] Mortimer et al. 2000, *supra* note 38.

[96] Mortimer et al. 2002, *supra* note 85.

3. Adult "Responders" Are a Sensitive Population that Must Be Protected Under the Revised Standard

It has long been known that among healthy adults, some people are especially sensitive to ozone exposures. These people, historically known as responders, experience a markedly increased decline in pulmonary lung function in response to exposure to ozone, and their elevated responses are consistent over time. The controlled human exposure studies demonstrate that, within even a small sample of the population, some of those individuals respond with greater adverse effect to the ozone exposure.[97] A high level of inter-individual variability is also evidenced by the 3- to 20-fold difference in airway inflammation following ozone exposure.[98] In this review, for the first time, EPA recognizes that a certain genetic makeup predisposes some individuals to be especially responsive to ozone exposures, although other characteristics may also play a role that is not currently clear. It is estimated that between 5 and 20 percent of the healthy population are responders.[99] It is imperative that the standards be set at levels that will protect these responders, and not merely at levels that will protect the population on average.

4. Outdoor Workers, Due to Their Prolonged Exposures to Ambient Air, Are a Sensitive Population Requiring Heightened Protection

Another large and important subpopulation with heightened vulnerability to ozone is outdoor workers. Outdoor workers experience more frequent exposure to ozone than the general population, due to the time spent outdoors, and the increased breathing rate under physical exertion. Several studies have examined the association between ozone exposure and health outcomes in outdoor workers, including farm workers,[100] mail carriers,[101] and others.[102] In the United States, this population constitutes more than 9 million people. Outdoor workers include a diverse set of occupations, ranging from construction workers to farm workers. Table 1 lists some categories of outdoor workers and provides estimates of population size though this tabulation does not include members of the military forces.

Table 1. Worker Counts for Occupations likely to Involve Outdoor Work.[103]

Occupations	Number of workers

[97] C.S. Kim et al. (2011). Lung function and inflammatory responses in healthy young adults exposed to 0.06 ppm ozone for 6.6 hours, *Am. J. Respir. Crit. Care Med.*,183(9): 1215-21.
[98] Proposed Rule at 75,247.
[99] U.S. Environmental Protection Agency. Office of Air Quality Planning and Standards. Review of the National Ambient Air Quality Standards for Ozone Preliminary Assessment of Scientific and Technical Information. Draft Staff Paper November 1988.
[100] Brauer et al. 1996, *supra* note 17.
[101] C. Chan & T. Wu (2005). Effects of ambient ozone exposure on mail carriers' peak expiratory flow rates, *Environ Health Perspect*, 113: 735-738.
[102] Tovalin, et al. (2006). DNA damage in outdoor workers occupationally exposed to environmental air pollutants, *Occup. Environ. Med.*, 63: 230-236; M.S. O'Neill et al. (2003). Ozone exposure among Mexico City outdoor workers, *J. Air Waste Manag. Assoc.*, 53: 339-346.
[103] Derived from Census 2000 EEO Data Tool, *available at* http://www.census.gov/eeo2000/index.html. The Census Bureau tabulation excludes the four military categories and 35 occupational categories falling below a 10,000 person threshold.

Farm, Ranch, and Other Agricultural Managers	201,980
Farmers and Ranchers	587,015
Construction Managers	651,400
Surveyors, Cartographers, and Photogrammetrists	35,640
Surveying and Mapping Technicians	82,180
Conservation Scientists and Foresters	28,340
Athletes, Coaches, Umpires, and Related Workers	194,120
Emergency Medical Technicians and Paramedics	112,885
Fire Fighters	242,395
Miscellaneous Law Enforcement Workers	9,250
Police Officers	597,925
Crossing Guards	55,070
Lifeguards and Other Protective Service Workers	98,560
First-Line Supervisors/Managers of Landscaping, Lawn Service, and Groundskeeping Workers	134,200
Grounds Maintenance Workers	1,014,820
Door-To-Door Sales Workers, News and Street Vendors, and Related Workers	195,650
Couriers and Messengers	203,545
Meter Readers, Utilities	43,400
Postal Service Mail Carriers	354,395
Miscellaneous Agricultural Workers, Including Animal Breeders	806,075
Fishing and Hunting Workers	51,100
Forest and Conservation Workers	18,980
Logging Workers	105,675
Brickmasons, Blockmasons, and Stonemasons	212,210
Cement Masons, Concrete Finishers, and Terrazzo Workers	94,500
Construction Laborers	1,266,235
Miscellaneous Construction Equipment Operators	357,330
Roofers	222,995
Fence Erectors	29,835
Hazardous Materials Removal Workers	22,425
Highway Maintenance Workers	96,185
Rail-Track Laying and Maintenance Equipment Operators	12,200
Septic Tank Servicers and Sewer Pipe Cleaners	8,175
Miscellaneous Construction and Related Workers	33,505
Derrick, Rotary Drill, and Service Unit Operators, and Roustabouts, Oil, Gas, and Mining	15,545
Earth Drillers, Except Oil and Gas	29,140
Explosives Workers, Ordnance Handling Experts, and Blasters	9,590
Aircraft Mechanics and Service Technicians	183,075
Electrical Power-Line Installers and Repairers	106,285
Railroad Brake, Signal, and Switch Operators	10,070
Railroad Conductors and Yardmasters	48,330

Parking Lot Attendants	62,420
Service Station Attendants	126,575
Transportation Inspectors	39,945
Miscellaneous Transportation Workers, Including Bridge and Lock Tenders and Traffic Technicians	20,650
Pumping Station Operators	19,395
Refuse and Recyclable Material Collectors	88,455
TOTAL NUMBER OF WORKERS	**8,939,670**

In addition to the outdoor workers discussed above, recreational exercising adults and children will experience increased ozone exposure due to increased breathing rates.[104] Because participation in some sports can result in a child drawing up to 17 times the "normal" amount of air into the lungs, young athletes may be more likely to develop asthma.[105] Hikers, bikers, walkers, runners, and those that play outdoor sports are also at risk because their increased ventilation rate exposes them to a higher dose of ozone.

5. The Elderly Are Also a Large Sensitive Subpopulation At Risk From Ozone

The elderly are another sensitive subpopulation at risk from ozone exposure, as identified by EPA. More than 14.1 percent of the U.S. population is age 65 or older, equal to nearly 45 million elderly people.[106] The elderly are vulnerable to ozone exposure due to decreased physiological, metabolic, and compensatory processes, and a greater incidence of cardiovascular and respiratory disease, compared to younger adults. For example, one meta-analysis of epidemiologic evidence regarding sensitivity to mortality or hospital admission from short-term ozone exposure found persuasive evidence of increased mortality risk for elderly populations.[107] Similarly, studies from other countries report a pronounced relationship between daily mortality and ozone exposure in elderly[108] and that ozone exposure increases hospital admission rates in the elderly.

6. Recent Evidence Also Suggests New Subpopulations with Heightened Sensitivity to Ozone

The 2013 ISA includes new categories of populations at risk that were not identified in previous reviews. For example, two new categories are individuals with reduced intake of certain nutrients (i.e., vitamins C and E), and individuals with certain genetic makeup, including variations in genes related to oxidative metabolism or inflammation. While there are no ready estimates of the numbers of people that fall within these two categories, the expansion of EPA's

[104] Brunekreef et al. 1994, *supra* note 16; Spektor et al. 1998, *supra* note 16; Kinney et al. 1996, *supra* note 55.
[105] McConnell et al. 2002, *supra* note 22.
[106] U.S. Census Bureau, Population Division (2014). Annual Estimates of the Resident Population for Selected Age Groups by Sex for the United States, States, Counties, and Puerto Rico Commonwealth and Municipios: April 1, 2010 to July 1, 2013.
[107] M.L. Bell, A. Zanobetti, & F. Dominici (2014). Who is more affected by ozone pollution? A systematic review and meta-analysis. *Am. J. Epidemiol.*, 180 (1): 15-28.
[108] S. Cakmak, R.E. Dales, & C.B. Vidal (2007). Air pollution and mortality in Chile: susceptibility among the elderly. *Environ. Health Perspect.*, 115: 524-527.

populations at risk indicates that more people may be at risk of ozone and need stronger standards than were adopted in 2008.

C. Ozone Can Pose Significant Health Risks for Populations with Pre-Existing Health Conditions

Substantial and growing evidence warns that people with an array of pre-existing conditions such as obesity, diabetes, heart disease, COPD, and cystic fibrosis may be at increased risk from exposure to ozone. The ISA now recognizes a likely causal relationship between short-term exposures to ozone and cardiovascular effects. It necessarily follows that people with heart disease and high blood pressure already are at increased risk from ozone exposure. The ISA also concludes that there is suggestive evidence that ozone causes reproductive and developmental effects. This being the case, pregnant women should be considered among those with increased susceptibility to ozone air pollution.

1. Obesity is a Pre-Existing Condition Requiring Additional Protection Under the Revised NAAQS

There is growing information identifying obesity as a potential risk factor for increased susceptibility to ozone air pollution. More than 1 in 3 adults are considered to be obese, while 1 in 20 adults (6.3 percent) are classified as having extreme obesity.[109] Among children ages 2 to 19, 16.9 percent are considered to be obese.[110] All told, there are 80 million obese adults, and 12 million obese children. This demographic must be carefully considered and a precautionary approach taken to ensure they are adequately protected.

Obese individuals have higher breathing rates, which can increase their exposure to ozone and other air pollutants.[111] Scientific studies have recognized obesity as a risk factor in asthma.[112] Obesity may modify airway inflammation, which affects the sensitivity of the lung to ozone and obese individuals may have lower baseline lung function.[113]

The recently published results from the Framingham Heart Study, report that obese people had a larger decrease in FEV_1[114] in association with previous-day ozone exposure than non-obese participants. The association per 10 ppb of ozone was more than twice as large for

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[109] K.M. Flegal, M.D. Carroll, B.K. Kit, & C.L. Ogden (2012). Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. *J. Am. Med. Assn.* 301(5): 491-497; Weight Control Information Network, National Institute of Diabetes and Digestive and Kidney Diseases, Overweight and Obesity Statistics, *available* at http://win.niddk.nih.gov/statistics (accessed Mar. 15, 2015).

[110] C.L. Ogden, M.D. Carroll, B.K. Kit, & K.M. Flegal (2012). Prevalence of obesity and trends in body mass index among US children and adolescents, 1999-2010, *J. Am. Med. Assn.* 301(5): 483-90.

[111] P.D. Koman (2012). How Does the Obesity Epidemic Affect Risk from Air Pollution? Risk Science Center Occasional Papers, University of Michigan School of Public Health; P. Brochu, M. Bouchard, & S. Haddad (2013). Physiological Daily Inhalation Rates for Health Risk Assessment in Overweight/Obese Children, Adults, and Elderly, *Risk Analysis*.

[112] O. Sideleva & A.E. Dixon (2014). The many faces of asthma in obesity, *J. Cell. Biochem*., 115 (3): 421-6.

[113] A.E. Dixon et al. (2010). An official American Thoracic Society Workshop report: obesity and asthma, *Proc. Am. Thorac. Soc.*, 7(5): 325-35.

[114] FEV_1 (forced expiratory volume in one second) is a commonly-used metric for assessing respiratory health.

obese subjects, compared with the non-obese.[115] Similar results were reported in the Normative Aging Study, which found that obesity worsened the effect of ozone on lung function in the elderly.[116] These findings are backed up by experimental studies which have found that the inhaled dose of ozone was greater in obese mice and that airway hyperresponsiveness and airway inflammatory responses to ozone were enhanced in obese mice compared to lean or normal-weight mice.[117] More recently, a multi-city study in China reported that the effects of ozone air pollution on hypertension were greatest among obese men,[118] while a study of Chinese children reported that respiratory health effects are enhanced among obese children.[119]

2. Diabetes is a Pre-Existing Condition Requiring Additional Protection

Another growing segment of the population, individuals suffering from diabetes may also be at greater risk of adverse health impacts from ozone exposure. According the American Diabetes Association, in 2012, 29.1 million Americans, or 9.3 percent of the population had been diagnosed with diabetes, including 208,000 people under the age of twenty.[120]

A study in Santiago, Chile tested the association between daily air pollution concentrations and hospitalizations for serious complications of diabetes. Ozone was associated with increased risk of acute complications of diabetes requiring hospitalization, suggesting that improvements in air quality may reduce morbidity from diabetes.[121] Similarly, a panel study in Chapel Hill, North Carolina examined the short-term effects of temperature and ozone on endothelial dysfunction in people with type II diabetes. Investigators reported an association between temperature decreases and ozone increases on endothelial dysfunction in individuals with diabetes.[122]

Laboratory toxicology studies have explored the mechanisms by which ozone might induce glucose intolerance and potentially lead to diabetes, though this is in the early stages of research. A laboratory study in rats, to be published in the journal *Diabetes*, reported that ozone plays a causative role in the development of insulin resistance, suggesting that it could boost the development of diabetes.[123] A 2013 publication reported "acute ozone exposure induces marked

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[115] M.B. Rice, et al. (2013). Short-term exposure to air pollution and lung function in the Framingham Heart Study, *Am. J. Respir. Crit. Care Med.*, 188(11): 1351-7.

[116] S.E. Alexeeff, et al. (2007). Ozone exposure and lung function: effect modified by obesity and airways hyperresponsiveness in the VA normative aging study, *Chest*, 132 (6): 1890-7.

[117] R.A. Johnston et al. (2008). Diet-induced obesity causes innate airway hyperresponsiveness to methacholine and enhances ozone-induced pulmonary inflammation, *J. Appl. Physiol.*, 104: 1727–1735.

[118] Y. Zhao et al. (2013). Does obesity amplify the association between ambient air pollution and increased blood pressure and hypertension in adults? Findings from the 33 Communities Chinese Health Study, *Int. J. Cardiol*., 168 (5): e148-50.

[119] G.H. Dong et al. (2013). Obesity enhanced respiratory health effects of ambient air pollution in Chinese children: the Seven Northeastern Cities study, *Int. J. Obes.* (Lond), 37 (1): 94-100.

[120] American Diabetes Association, Statistics About Diabetes-Statistics About Diabetes Data from the *National Diabetes Statistics Report, available at* http://www.diabetes.org/diabetes-basics/statistics/#sthash.1JKw9Pf7.dpufhttp://www.diabetes.org/diabetes-basics/statistics/#sthash.1JKw9Pf7.dpuf.

[121] R.E. Dales, S. Cakmak, C.B. Vidal, & M.A. Rubio (2012). Air pollution and hospitalization for acute complications of diabetes in Chile, *Environ. Int*., 46: 1-5.

[122] S. Lanzinger et al. (2014). The impact of decreases in air temperature and increases in ozone on markers of endothelial function in individuals having type-2 diabetes, *Environ. Res.*, 134: 331-8.

[123] R.E. Vella et al. (2014). Ozone exposure triggers insulin resistance through muscle c-Jun N-terminal Kinases

systemic metabolic impairments in Brown Norway rats of all ages, likely through sympathetic stimulation."[124] These and other studies indicate that people with diabetes should be among those considered especially vulnerable to ozone exposures.

3. COPD is a Pre-Existing Condition Necessitating Additional Protection under the Revised Standard

The ISA indicates that ozone causes adverse respiratory health effects, but it finds evidence inadequate to classify people with chronic obstructive pulmonary disease (COPD), a disease that includes both chronic bronchitis and emphysema, at increased risk from ozone. This is counterintuitive, because the causal finding is based in part on evidence of increased risk of hospital admissions, emergency room visits, and increased risk of premature death. COPD is the third leading cause of death in America, claiming the lives of 134,676 Americans in 2010.[125] In 2011, 12.7 million U.S. adults were estimated to have COPD.[126] However, close to 24 million U.S. adults have evidence of impaired lung function, indicating an under-diagnosis of COPD.[127]

Studies show that people with COPD are especially susceptible to ozone. A very large case-crossover study of Medicare recipients in 36 U.S. cities evaluated the effect of ozone and PM_{10} on respiratory hospital admissions in the elderly over a 13-year period. The study found that the risk of daily hospital admissions for COPD and pneumonia increased with short-term increases in ozone concentrations during the warm season, but not during the cold season. Importantly, 8-hour mean warm season ozone concentrations in this study ranged from 15 ppb in Honolulu to 63 ppb in Los Angeles. As indicated in Table 2 below, ozone concentrations in most cities were in the 40-55 ppb range.[128] This study provides powerful evidence for a standard of 60 ppb or below.

Table 2: Mean ozone levels in U.S. cities during 1986-1999.[129]

(JNKs) activation, *Diabetes*. Epub ahead of print.

[124] V. Bass et al. (2014). Ozone induces glucose intolerance and systemic metabolic effects in young and aged Brown Norway rats, *Toxicol. Appl .Pharmacol.*, 273(3): 551-60.

[125] Centers for Disease Control and Prevention (2013). National Center for Health Statistics. National Vital Statistics Report. Deaths: Final Data for 2010; 61(04).

[126] CDC (2011). National Center for Health Statistics. National Health Interview Survey Raw Data, 2011. Analysis performed by the American Lung Association Research and Health Education Division using SPSS and SUDAAN software.

[127] CDC (2002). Chronic Obstructive Pulmonary Disease Surveillance – United States, 1971-2000. Morbidity and Mortality Weekly Report. 51(SS06):1-16.

[128] M. Medina-Ramón, A. Zanobetti, & J. Schwartz (2006). The Effect of Ozone and PM_{10} on Hospital Admissions for Pneumonia and Chronic Obstructive Pulmonary Disease: A National Multicity Study, *Am. J. of Epid*, 163: 579-588.

[129] *Id.*

TABLE 1. Environmental variables and respiratory hospital admissions in 36 US cities during 1986–1999

City, state	Mean (SD*) ozone level (ppb)		Mean (SD) PM_{10}* level ($\mu g/m^3$)	Mean (SD) apparent temperature (°C)	Total population aged ≥65 years (no.)	COPD* admissions (no.)	Pneumonia admissions (no.)
	Warm season	Cold season					
Albuquerque, New Mexico	50.5 (9.3)	34.5 (10.2)	27.9 (16.5)	12.2 (8.9)	50,379	3,115	9,035
Atlanta, Georgia	55.9 (21.4)		33.0 (16.4)	17.1 (10.2)	155,955	15,503	36,488
Baltimore, Maryland	52.3 (20.2)	26.8 (13.0)	32.4 (17.1)	13.0 (11.1)	197,438	19,950	40,858
Birmingham, Alabama	49.7 (17.0)		36.1 (21.0)	17.4 (10.5)	119,809	13,134	33,011
Boston, Massachusetts	42.3 (17.8)	28.3 (11.3)	25.4 (11.7)	10.0 (10.3)	342,322	34,700	88,936
Boulder, Colorado	51.3 (14.2)		24.2 (15.5)	8.5 (9.7)	17,048	1,678	3,427
Canton, Ohio	52.6 (17.8)		26.1 (12.6)	9.3 (11.2)	53,216	7,534	12,965
Chicago, Illinois	40.0 (16.1)	22.7 (9.8)	33.6 (17.4)	9.5 (11.9)	631,826	49,581	142,576
Cincinnati, Ohio	50.0 (17.8)		32.2 (15.6)	11.9 (11.5)	115,000	10,797	33,323
Cleveland, Ohio	44.6 (17.6)		37.1 (19.1)	9.8 (11.3)	220,659	29,947	50,262
Colorado Springs, Colorado	45.5 (11.3)	30.4 (11.6)	23.3 (13.4)	7.8 (9.0)	31,674	2,497	5,729
Columbus, Ohio	49.8 (18.1)		30.5 (14.6)	11.1 (11.5)	92,485	12,571	21,900
Denver, Colorado	44.0 (14.0)	22.1 (12.7)	33.2 (18.8)	8.5 (9.7)	64,152	4,219	11,820
Detroit, Michigan	41.7 (17.2)		33.7 (19.7)	9.3 (11.5)	263,997	5,751	12,393
Honolulu, Hawaii	15.0 (8.4)		15.9 (6.2)	27.5 (2.9)	91,485	28,404	57,682
Houston, Texas	44.9 (22.1)	32.9 (17.1)	30.3 (16.0)	22.2 (10.1)	196,474	3,798	14,463
Jersey City, New Jersey	50.3 (23.4)		32.2 (17.0)	12.4 (11.1)	70,014	18,863	41,754
Los Angeles, California	63.0 (23.4)	31.4 (20.2)	44.0 (19.3)	16.5 (4.3)	855,666	9,211	12,645
Minneapolis, Minnesota			27.3 (14.6)	7.4 (12.5)	175,854	63,316	174,241
Nashville, Tennessee	44.9 (16.8)	23.9 (13.5)	32.2 (14.9)	15.5 (11.3)	59,235	9,805	26,923
New Haven, Connecticut	45.4 (19.5)		26.0 (16.1)	9.6 (10.8)	117,863	5,962	14,719
New York City, New York	41.0 (19.5)	19.7 (10.0)	28.9 (13.9)	12.5 (10.8)	952,731	8,082	22,954
Palm Beach, Florida	28.6 (12.7)	33.7 (12.0)	20.0 (8.1)	27.1 (6.3)	210,389	70,181	187,043
Philadelphia, Pennsylvania	47.8 (21.0)	23.0 (13.0)	32.1 (15.8)	12.9 (11.1)	241,206	10,626	22,170
Pittsburgh, Pennsylvania	48.4 (19.9)		30.3 (20.0)	10.3 (10.9)	232,505	26,604	47,126
Provo, Utah	54.6 (10.9)		35.1 (26.7)	9.6 (10.4)	18,429	33,408	52,148
Sacramento, California	55.6 (15.7)	32.7 (14.2)	31.1 (19.7)	14.4 (7.0)	109,674	718	4,081
Salt Lake City, Utah	54.0 (12.5)		35.7 (23.9)	9.6 (10.4)	61,079	8,680	21,840
San Diego, California	47.6 (12.1)	40.4 (15.2)	33.3 (13.1)	17.0 (4.4)	272,348	2,090	9,348
San Francisco, California	22.8 (8.1)	19.3 (10.2)	27.7 (16.8)	12.6 (3.8)	105,263	17,632	43,446
Seattle, Washington	35.0 (14.2)		28.8 (18.6)	9.5 (6.3)	167,328	4,711	18,139
Steubenville, Ohio	46.1 (17.3)		34.7 (19.9)	10.3 (10.9)	23,878	9,334	23,732
St. Louis, Missouri	48.4 (17.1)		27.7 (12.7)	13.7 (12.3)	214,492	4,039	9,412
Spokane, Washington	44.6 (10.4)		32.2 (28.3)	6.5 (9.0)	47,877	5,633	8,976
Washington, DC	48.4 (20.2)	20.1 (12.3)	27.7 (13.4)	14.2 (11.2)	77,672	17,665	54,386
Youngstown, Ohio	47.1 (20.3)		31.2 (15.6)	8.9 (11.0)	61,122	8,267	14,862

* SD, standard deviation; PM_{10}, particulate matter with an aerodynamic diameter of ≤10 μm; COPD, chronic obstructive pulmonary disease.

A study in Hong Kong examined the relationship between levels of ambient air pollutants and the hospitalization rate due to COPD in Hong Kong. Significant effects were found between hospital admissions for COPD and all five ambient air pollutants examined, but ozone was the most important of the air pollutants studied. This study provides further evidence of the special susceptibility of people with COPD to ozone.[130]

[130] Ko et al. (2007). The temporal relationship between air pollutants and hospital admissions for chronic obstructive pulmonary disease in Hong Kong, 62(9): 780-5.

Likewise, a study in Taipei, Taiwan also reported positive associations between ozone and hospital admissions for COPD in single- and two-pollutant models, where mean ozone concentrations were 20.52 ppb, and maximum ozone concentrations were 62.79 ppb.[131] A French study reported that ozone exacerbates symptoms in COPD patients. Thirty-nine senior adults with severe COPD were followed by their physicians in Paris, France, during a 14-month period. Daily levels of PM_{10}, ozone, sulfur dioxide, and nitrogen dioxide were monitored. Only the 8-hour average ozone concentration was associated with exacerbation of COPD symptoms.[132] According to the researchers:

> Our results are consistent with those of toxicological studies that have shown the inflammatory mechanisms of O_3. The recruitment of inflammatory cells into the lung presents a risk of tissue damage through the release of toxic mediators by activated inflammatory cells. Perhaps this phenomenon would be more serious among patients suffering from COPD, in whom a pre-existent inflammation of the small or large airways would be constant.[133]

Another just published study, this one of 1,200 people with emphysema who had undergone lung volume reduction surgery, assessed the association between short- and long-term pollutant concentrations and changes in pulmonary function.[134] Air pollution exposure ($PM_{2.5}$ and ozone) was strongly associated with worsened respiratory function and symptoms. Researchers concluded that "exposures even below those of air quality standards may still pose significant risks to severe chronic obstructive pulmonary disease (COPD) subjects."[135]

4. Heart Disease is a Pre-Existing Condition that Poses Additional Risks When Exacerbated by Ozone Exposure

EPA must also assure that the primary standard prevents adverse effects to cardiovascular health from ozone exposure. Heart disease is the leading cause of death for both men and women in the U.S. Approximately 85.6 million people in this country suffer from some form of cardiovascular disease, including high blood pressure.[136] The ISA found that that the relationships between short-term exposures to ozone and both total mortality and cardiovascular effects are likely to be causal. ISA 1-7 to -8. There is substantial support for this finding.

Just this year, an expert consensus document prepared on behalf of the European Society of Cardiology explored the mechanisms of interactions and relationships between ambient air pollution and cardiovascular disease. As illustrated in Figure 2 below, the paper identifies

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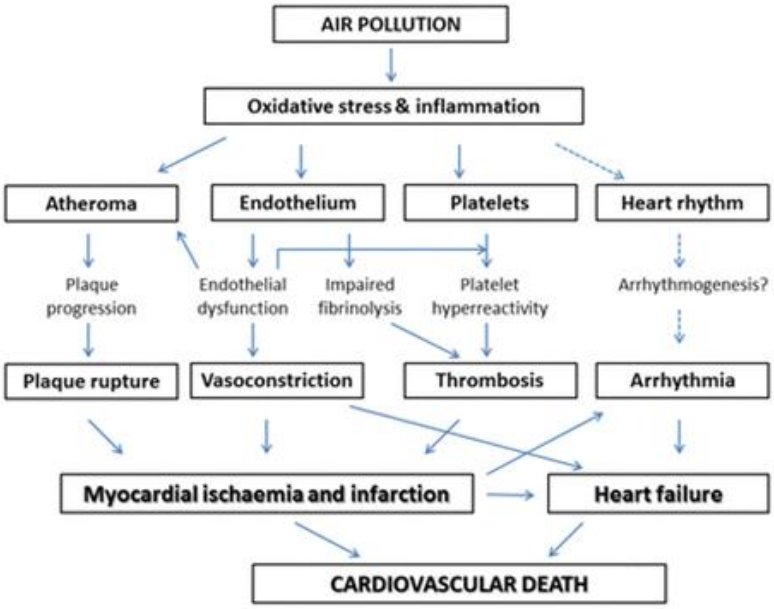
[131] C.Y. Yang & C.J. Chen (2007). Air pollution and hospital admissions for chronic obstructive pulmonary disease in a subtropical city: Taipei, Taiwan, *J. Toxicol. Environ. Health*, 70, 1214-1219.
[132] H. Desqueyroux, J.C. Pujet, M. Prosper, Y. Le Moullec, & I. Momas (2002). Effects of Air Pollution on Adults With Chronic Obstructive Pulmonary Disease, *Arch. of Environ. Health*, 57: 554-560.
[133] *Id*.
[134] M. Kariisa et al. (2015). Short- and long-term effects of ambient ozone and fine particulate matter on the respiratory health of chronic obstructive pulmonary disease subjects, *Arch. Environ. Occup. Health*, 70 (1): 56-62.
[135] *Id.*
[136] John Hopkins Medicine, Cardiovascular Disease Statistics, *available at* http://www.hopkinsmedicine.org/healthlibrary/conditions/cardiovascular_diseases/cardiovascular_disease_statistics_85,P00243 (accessed Mar. 15, 2015).

multiple pathways by which air pollution is linked to cardiovascular morbidity and mortality, including the induction of oxidative stress, systemic inflammation, endothelial dysfunction, atherothrombosis, and arrhythmogenesis.[137]

Figure 2. Mechanistic effects of air pollution on cardiovascular morbidity and mortality.[138]



The authors concluded that "[a]ir pollution should be viewed as one of several major modifiable risk factors in the prevention and management of cardiovascular disease."[139] Similarly, Zanobetti and Schwartz examined associations between "long-term" ozone exposures and survival of Medicare participants in 105 major U.S. cities who have been hospitalized for COPD, diabetes, congestive heart failure, and myocardial infarction. The analysis found significant associations between annual summer-average levels of ozone and deaths for all of these four groups. It finds a 6 to 8 percent increase in risk per 5 ppb increase in the summer average of daily maximum 8-hour ozone levels. This study provides additional support for considering patients with COPD, diabetes, and heart disease as susceptible populations.[140]

Several recent studies published since the ISA have reported that ozone pollution increases risk of cardiac arrest. A case-crossover study in Houston examined emergency medical services data on out-of-hospital cardiac events relative to air pollution concentrations. Investigators reported consistent evidence of an association between out-of-hospital cardiac events and exposure to ozone. A 20 ppb ozone increase in the 8-hour average daily maximum ozone concentrations was associated with an increased risk of a cardiac event on the same day.

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[137] D.E. Newby et al. (2015). Expert position paper on air pollution and cardiovascular disease. *Eur. Heart. J.*, 36 (2): 83-93.
[138] *Id.*
[139] *Id*.
[140] A. Zanobetti & J. Schwartz (2011). Ozone and survival in four cohorts with potentially predisposing diseases, *Am. J. Respir. Crit. Care. Med.*, 184(7): 836-41; Teng et al. (2014). A systematic review of air pollution and incidence of out-of-hospital cardiac arrest, *J. Epidemiol. Comm Health*, 68 (1): 37-43.

Similarly, a 20 ppb increase in ozone in the previous 1 to 3 hours was also associated with an increased risk of a cardiac event on the same day. Mean hourly ozone concentrations in this study were 25.5 ppb.[141] A recent study examined over 2,000 cases of out-of-hospital cardiac arrest in Helsinki, Finland, for the period of 1998 to 2006. Ozone pollution was linked with a large increase in the risk of cardiac arrest due to arrhythmia, occurring two to three days after exposure to ozone.[142]

A 2014 study in Stockholm, Sweden used ten years of data from the Swedish cardiac arrest register with a time-stratified case-crossover design to analyze exposure to air pollution and the risk of out-of-hospital cardiac arrest. Exposure to ozone, $PM_{2.5}$, PM_{10}, NO_2, and NOx was defined as the mean urban background level during 0–2, 0–24, and 0–72 hours before the event and control time points. The study, which adjusted for temperature and relative humidity, found that ozone in urban background was associated with an increased risk of out-of-hospital cardiac arrest for all time windows analyzed.[143] The mean 8-hour maximum ozone concentration in urban areas during the warm season was 34 ppb. The study suggested that short-term elevations of ozone urban background levels are associated with an increased risk of out-of-hospital cardiac arrest with no indication of a threshold, in a region with ~50 $\mu g/m^3$ [23.4 ppb] annual ozone levels.

5. Evidence Connects Ozone Exposure to Strokes

There is also increasing evidence of a relationship between ozone exposure and stroke. A time-stratified case-crossover analysis evaluated the relationships between stroke hospital admissions and ozone among patients aged 65 years and older in Allegheny County, Pennsylvania. The study found that same day exposures to ozone may increase the risk of hospitalization for stroke, and that effects were greater in males than in females. Researchers concluded that "results suggest that O3 has an adverse effect on stroke hospitalization. Specific patient subgroups, such as males, may be at increased risk."[144] Given the growing evidence, EPA needs to follow the requirement of including a margin of safety to protect heart patients from increased risk of cardiac arrest and stroke. Indeed, such patients should be considered a sensitive population.

6. Transplant Recipients Are a Sensitive Subpopulation for Ozone Exposure

Similarly, two studies indicate that transplant recipients may be a sensitive subpopulation. Not surprisingly, research suggests lung transplant patients are susceptible to harm from pollution. For example, a retrospective cohort study of kidney transplant recipients with numerous known risk factors explored whether they may constitute a sensitive subgroup as

[141] K.B. Ensor, l.H. Raun, & D. Persse (2013), A case-crossover analysis of out-of-hospital cardiac arrest and air pollution. *Circulation*, 127 (11): 1192-9.
[142] F.S. Rosenthal et al. (2013). Association of ozone and particulate air pollution with out-of-hospital cardiac arrest in Helsinki, Finland: evidence for two different etiologies, *J. Expo. Sci. Environ. Epidemiol.*, 23 (3): 281-8.
[143] A. Raza et al. (2014). Short-term effects of air pollution on out-of-hospital cardiac arrest in Stockholm, *Eur. Heart J.*, 35 (13): 861-8.
[144] X. Xu, Y. Sun, S. Ha, E.O. Talbott, & C.T. Lissaker (2013). Association between ozone exposure and onset of stroke in Allegheny County, Pennsylvania, USA, 1994-2000, *Neuroepidemiology*, 41(1): 2-6.

far as air pollution is concerned. Monthly concentrations of ozone and PM_{10} were calculated from ambient monitoring data and interpolated to zip code centroids according to patients' residence. For the entire transplant cohort, average pollutant levels for ozone were 25.5 ± 4.4 ppb. For each 10 ppb increase in ozone, the risk of fatal coronary heart disease increased by about 34 percent in both the single and the two pollutant model. Researchers concluded that for kidney transplant recipients, ambient ozone levels are potentially associated with higher risk of fatal coronary heart disease.[145]

A retrospective cohort study of 397 bilateral lung recipients examined the relation between ambient air pollution, chronic lung allograft dysfunction, and mortality.[146] These studies suggest that transplant recipients may be at increased risk for chronic lung allograft dysfunction from ozone exposures and should be considered a susceptible population.

7. Cystic Fibrosis is a Pre-Existing Condition Requiring a More Protective Ozone Standard

People with cystic fibrosis, a chronic lung disease, may also be at risk. Cystic fibrosis is a disease that causes thick, sticky mucus to form in the lungs, pancreas, and other organs. This mucus clogs the lungs causing wheezing, shortness of breath, and frequent lung infections.[147] Cystic fibrosis involves significant inflammation and oxidative stress, and exposure to air pollutants can worsen the lung damage. Deficits in innate immunity, chronic infection, oxidative stress, and inflammation due to cystic fibrosis may indicate a population disproportionately vulnerable to the harms of air pollution. The Cystic Fibrosis Foundation estimates that 30,000 children and adults in the U.S. have cystic fibrosis.[148]

A study in São Paulo, Brazil investigated the association between the short-term variation in the concentration of air pollutants and the occurrence of respiratory exacerbations in children and adolescents with cystic fibrosis.[149] Researchers report that a 22.2 ppb (interquartile range) increase in ozone concentration can lead to a 52 percent increase in the risk of respiratory exacerbation in children and adolescents with cystic fibrosis 48 hours after exposure, after controlling for other risk factors. Researchers indicated that the WHO Air Quality Guidelines, already more stringent that the NAAQS, should be lowered by at least 25 percent to minimize risks of pulmonary exacerbations in this high-risk population, concluding:

> The interaction between ozone and lung epithelial lining fluid in patients with CF most likely produces oxidized species that may be responsible for triggering lung inflammation

[145] R. Spencer-Hwang et al. (2011). Ambient air pollutants and risk of fatal coronary heart disease among kidney transplant recipients, *Am. J. Kidney Dis.*, 58(4): 608-16.
[146] Bhinder S, Chen H, Sato M, Copes R, Evans GJ, Chow CW, Singer LG. Air pollution and the development of post-transplant chronic lung allograft dysfunction. *Am J Transplant* 2014; 14 (12): 2749-57.
[147] Cystic Fibrosis Foundation, About CF, *available at* http://www.cff.org/AboutCF (accessed Mar. 15, 2015).
[148] *Id*.
[149] S. Farhat, et al. (2014). Ozone is associated with an increased risk of respiratory exacerbations in patients with cystic fibrosis, *Chest*, 144(4): 1186-1192.

> and contribute to acute bronchoconstriction and airway hyperresponsiveness, similar to the changes observed in asthma.[150]

An editorial in the journal *Chest* reviewing study commented:

> These results show that ground-level ozone air pollution has substantial adverse effects on the respiratory health of patients with cystic fibrosis and that the current US Environmental Protection Agency standards are not sufficient to protect the respiratory health of these patients. [151]

An earlier, much larger study of 11,484 cystic fibrosis patients aged 6 years and older, who lived within 30 miles of an air quality monitor, found that a 10 ppb rise in ozone was associated with increased risk of pulmonary exacerbations.[152] Collectively these studies suggest that cystic fibrosis patients have been overlooked as a susceptible population.

8. Due to the Risk of Reproductive and Developmental Effects, Pregnant Women Should Be Considered a Sensitive Group

Pregnant women and their fetuses are another population that should be considered especially susceptible to air pollution. A number of recent studies have reported associations between exposure to air pollution and adverse pregnancy outcomes such as pre-eclampsia, preterm birth, and low birth weight babies. For example, a prospective register-based cohort study in Stockholm, Sweden observed an increased risk with ozone exposure in the first trimester for preterm birth and pre-eclampsia. Researchers estimated one in twenty cases of pre-eclampsia to be associated with ozone exposure.[153] A study published in 2012 assessed the association between ambient pollutant concentrations and term birth weight for 1.5 million births in Texas from 1998 to 2004. Lower birth weight was associated with ozone exposure in the first and second trimester.[154] Another study in North Carolina reported that ozone concentrations in both urban and rural areas may be associated with an increased risk of term low birth weight and small for gestational age births.[155]

These are just a few of the studies look at air pollution concentrations and adverse pregnancy outcomes and which strongly suggest that pregnant women are especially vulnerable to exposure to ozone pollution. To comply with its Clean Air Act mandate, EPA must set standards to protect pregnant women and their fetuses.

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[150] *Id.*
[151] H.J. Farber (2013). Public policy, air quality, and protecting the most vulnerable, *Chest*, 144(4): 1093-4.
[152] Goss, et al. (2004). Effect of ambient air pollution on pulmonary exacerbations and lung function in cystic fibrosis, *Am. J. Respir. Crit. Care Med.*, 169 (7): 816-21.
[153] D. Olsson, I. Mogren, & B. Forsberg (2013). Air pollution exposure in early pregnancy and adverse pregnancy outcomes: a register-based cohort study, *BMJ Open*, 3(2).
[154] L.A. Geer, J. Weedon, & M.L. Bell (2012). Ambient air pollution and term birth weight in Texas from 1998 to 2004, *J. Air Waste Manag. Assoc.*, 62 (11): 1285-95.
[155] L.C. Vinikoor-Imler (2014). Associations between prenatal exposure to air pollution, small for gestational age, and term low birthweight in a state-wide birth cohort, *Environ. Res.*, 132: 132-9.

D. Based on the Demonstrated Impacts to Healthy Individuals at 60 ppb, the Impacts to Sensitive Populations at 60 ppb Are Properly Characterized as Adverse

In making judgments as to when various ozone-related effects become regarded as adverse to the health of individuals, EPA has explained that it looks to guidelines published by the American Thoracic Society (ATS)[156], as well as the advice of CASAC.[157] Effects may be adverse at the individual or population level. For example, at the individual level, ATS guidance concludes that "transient, reversible loss of lung function in combination with respiratory symptoms should be considered adverse."[158] ATS has also identified as adverse "medically significant physiologic changes generally evidenced by" (among other things) "[i]nterference with the normal activity of the affected person or persons."[159] Additionally, effects may be adverse at the population level. As EPA explains, "[e]xposure to air pollution that increases the risk of an adverse effect to the entire population is adverse, even though it may not increase the risk of any individual to an unacceptable level."[160] Based on the ATS guidance and CASAC's advice to EPA regarding the proper characterization of adverse impacts, it is clear that known impacts to sensitive populations at levels of 60 ppb are properly characterized as adverse.

1. Children and Adults with Lung Disease Will Experience Impacts Properly Characterized as Adverse Based on Exposures to 60 ppb

While CASAC advised that "[e]stimation of FEV1 decrements of ≥15 percent is appropriate as a scientifically relevant surrogate for adverse health outcomes in active healthy adults,"[161] CASAC also concluded that lung function decrement greater than 10 percent in sensitive populations including children and adults with lung disease would interfere with normal activity for many individuals and would likely result in additional and more frequent use of medication.[162] As CASAC advised,

> for children and adults with lung disease, even moderate functional (e.g., FEV_1 decrements ≥ 10 percent but < 20 percent, lasting up to 24 hours) or symptomatic responses (e.g., frequent spontaneous cough, marked discomfort on exercise or with deep breath, wheeze accompanied by shortness of breath, lasting up to 24 hours) would likely interfere with normal activity for many individuals, and would likely result in additional and more frequent use of medication.[163]

As ATS explains in its 2000 guidance on adverse effects, a change in medication constitutes a change in clinical status and an adverse health impact at the individual level:

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[156] American Thoracic Society (2000). What Constitutes an Adverse Health Effect of Air Pollution? *Am. J. Respir. Crit. Care Med*., 161:665-673 [hereinafter ATS 2000].
[157] Proposed Rule at 75,263.
[158] Proposed Rule at 75,263.
[159] *Id*. at 75263/1.
[160] *Id*. at 75263.
[161] *Id*., quoting CASAC Letter 2014a at 3.
[162] *Id*.
[163] *Id.* at 75,263-4, *citing* U.S. EPA, 2007, p.3-72; 72 FR 37849, July 11, 2007.

> The committee judged that air pollution-related symptoms associated with diminished quality of life or with a change in clinical status should be considered as adverse at the individual level. Characterizing the degree of symptomatology associated with diminished quality of life is an appropriate focus for research and a topic that could be investigated using new approaches for assessing quality of life. A change in clinical status can be appropriately set in a medical framework as one requiring medical care or a change in medication.[164]

Recent controlled human exposure studies conducted with healthy adults show that 60 ppb for 6.6 hours results in lung function decrement >10 percent for 10 percent of individuals.[165] As discussed extensively in section IV.B.2.a above, children are more sensitive to ozone pollution than adults.[166] Consequently, exposures of 60 ppb for 6.6 hours for children and for adults with lung disease are anticipated to result in additional and more frequent use of medication, a change in clinical status that ATS and CASAC consider to be adverse at the individual level.

2. Individuals with Pre-existing Pulmonary or Cardiac Disease Will Experience Impacts Properly Characterized as Adverse Based on Exposures to 60 ppb

Similarly, CASAC advises that 10 percent decrements in FEV_1 can lead to respiratory symptoms in individuals with pre-existing pulmonary or cardiac disease.[167] These populations are at least as sensitive as healthy adults who were in the controlled human exposure studies.[168] As ATS explained, "by definition, susceptible individuals cannot have the same margin of safety as the non-susceptible groups within the population."[169] ATS recommends, and EPA acknowledges, that a combination of lung functions decrements of 10 percent in FEV_1 and respiratory symptoms is adverse.[170] Consequently, it is clear that individuals with pre-existing pulmonary or cardiac disease are likely to experience impacts properly characterized as adverse based on exposures to 60 ppb.

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[164] American Thoracic Society. What constitutes an adverse health effect of air pollution? Official statement of the American Thoracic Society. *Am J Respir Crit Care Med* 2000; 161 (2 Pt 1): 665-73.

[165] Proposed Rule at 75,250/2 & n.37 (noting that percentage is an underestimate).

[166] *See also* Proposed Rule at 75,246 ("[A]t-risk populations or lifestages, such as people with asthma or children, are expected to be affected more by [exposures of concern] than healthy adults.") EPA in a footnote in the proposed rule observes that the HREA's estimates of lung function decrements are "based on the assumption that children exhibit the same lung function responses following O_3 exposures as healthy 18 year olds," citing a 30-year old study of 8-11 year old children. *Id*. at 75,248, n.28. Given the differing physiology of children discussed in section IV.B.2.a, reliance on a single study involving 8- to 11-year old children to conclude they experience the same lung function decrements would be arbitrary. Nevertheless, even if reliance on the results of this study was rational, it would not diminish the conclusion that a substantial subset of children would experience lung function decrements of at least 10% when exposed to concentrations of 60 ppb for periods of 6.6 hours, as occurred in healthy adults.

[167] Proposed Rule at 75,264 ("In addition, in their letter advising the Administrator on the reconsideration of the 2008 final decision, CASAC stated that '[a] 10% decrement in FEV1 can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease.'").

[168] ATS 2000.

[169] *Id.*

[170] *See* ATS 2000 at 671 ("this committee recommended that reversible loss of lung function in combination with the presence of symptoms should be considered adverse.").

3. Individuals with Asthma Will Experience Impacts Properly Characterized as Adverse Based on Exposures to 60 ppb

The same analysis as above applies to individuals with asthma. As EPA acknowledges, "[i]n this review, CASAC concurred that '[a]n FEV_1 decrement of ≥10 percent is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease'."[171] Indeed, in the last ozone NAAQS review, EPA itself agreed that lung function decrements of 10 percent in FEV_1 standing alone were adverse for people with asthma.[172] Moreover, asthmatics have been found to have double the decrements in lung function of healthy people when exposed to the same levels of ozone.[173] Given that a statistically significant percentage of healthy adults were found to experience lung function FEV_1 decrements of >10 percent when exposed to levels of 60 ppb for 6.6 hours, asthmatics and other individuals with lung disease would experience FEV_1 decrements far in excess of 10 percent when similarly exposed to ozone concentrations of 60 ppb for 6.6 hours. In light of CASAC's advice that such lung function decrements are an "adverse health outcome" for these sensitive groups, it is clear that these groups would experience impacts properly characterized as adverse based on exposures to 60 ppb.

4. Lung inflammation in Healthy Adults at 60 ppb is Properly Characterized as Adverse

In its proposal, EPA observes that "[e]vidence new to this review indicates that 6.6-hour exposures to 60 ppb O3 during moderate exertion can result in pulmonary inflammation in healthy adults (based on study mean)."[174] EPA explains that:

> As discussed in the ISA, the initiation of inflammation can be considered as evidence that injury has occurred. Inflammation induced by a single O3 exposure can resolve entirely but, as noted in the ISA (U.S. EPA, 2013a, p. 6-76), 'continued acute inflammation can evolve into a chronic inflammatory state,' which would be adverse.[175]

"Unlike O3-induced decrements in lung function, which are attenuated following repeated exposures over several days . . . some markers of O3-induced inflammation and tissue damage remain elevated during repeated exposures, indicating ongoing damage to the respiratory system."[176] Because this continued acute inflammation can evolve into a chronic inflammatory state, which EPA acknowledges to be adverse, the evidence of pulmonary inflammation when healthy adults are exposed to ozone at concentrations of 60 ppb for 6.6 hours must be considered adverse.

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[171] Proposed Rule at 75,264, *citing* CASAC Letter 2014a.
[172] 73 Fed. Reg. at 16,454/3-55/1 (Mar. 27, 2008).
[173] Proposed Rule at75,265 ("For instance, Horstman et al. (1995) observed that mild-to-moderate asthmatics, on average, experienced double the O3-induced FEV1 decrement of healthy subjects (19% versus 10%, respectively, p = 0.04).").
[174] *Id*. 75,264.
[175] *Id*. 75,264; *see also* Policy Assessment at 3-19 ("In addition, one recent controlled human exposure study has reported O3-induced PMN influx following exposures of healthy adults to O3 concentrations of 60 ppb (Kim et al., 2011), the lowest concentration at which inflammatory responses have been evaluated in human studies.").
[176] Policy Assessment at 3-19, *citing* U.S. EPA, 2013, section 6.2.1.1 and 6.2.3.1, p. 6-81.

5. Population Level Effects Linked to Ozone Exposures at Levels of 60 ppb are Properly Characterized as Adverse.

In its guidance on adverse impacts, ATS makes clear that "[a]t the population level, any detectable increment in symptom frequency should be considered as constituting an adverse health effect."[177] Noting that "[a] wide range of clinical outcome measures has been considered in relation to air pollution, including population-level effects, such as increases in numbers of emergency room visits for asthma or hospitalizations for pneumonia, and individual level effects, such as increased need for bronchodilator therapy," ATS explained that: "The present committee shared the view of the previous group: detectable effects of air pollution on clinical measures should be considered adverse."[178]

A robust epidemiological literature links exposure to ozone at levels of 60 ppb with numerous clinical measures. As discussed in section V.B below, many studies have demonstrated an independent association of short-term exposures of ozone to premature mortality, and respiratory and cardiac effects, often at mean or median concentrations of 60 ppb and below. In addition, epidemiological studies support the ISA's conclusion that there is a likely causal relationship between long-term exposures to ozone and respiratory effects. Moreover, the ISA finds that "long term ozone exposure is associated with adverse effects ranging from episodic respiratory illness to permanent respiratory injury to progressive respiratory decline."[179] The ISA document details numerous studies showing that long-term exposures to ozone are associated with new onset asthma, increased asthma symptoms, increased risk of asthma hospital admissions, deficits in lung function growth rate in children, and increased risk of premature death. These population level "detectable effects of air pollution on clinical measures" occurring at levels of 60 ppb are properly considered adverse.

Further, as discussed more fully below, the statistically significant group mean decrease in FEV1 observed in controlled human exposure studies as low as 60 ppb is an adverse effect at the population level. In the last revision of the sulfur dioxide ("SO_2") NAAQS, EPA correctly found that "diminished reserve lung function in a population that is attributable to air pollution is considered an adverse effect under ATS guidance."[180] Decrements of that type occur in controlled human exposure studies at ozone levels down to 60 ppb.

V. COMMENTS ON THE PROPOSAL

A. The Current Standard of 75 ppb Does Not Protect the Public Health with an Adequate Margin of Safety As Required by the Clean Air Act

EPA correctly acknowledges adverse health effects caused by ozone levels of 75 ppb and the need to revise the primary standard in order to protect public health. Based on current scientific evidence, risk assessment modeling, CASAC advice, and public comment, EPA

[177] ATS 2000 at 671 (emphasis added).
[178] *Id.* (emphasis added).
[179] ISA Section 7.2.8
[180] 75 Fed. Reg. 35,220, 35,226/2 (June 22, 2010).

concludes that the current primary standard of 75 ppb "is not requisite to protect public health with an adequate margin of safety, and that it should be revised to provide increased public health protection."[181]

The fact that the current primary standard of 75 ppb is inadequate to protect public health and meet the requirements of the Clean Air Act is not new, however, and has long been recognized by EPA and its scientific advisors. According to EPA, "significant risks to public health are likely to occur at a standard level of 0.075 ppm," which is "not sufficient to meet the statutory requirement that the standard be set at a level requisite to protect public health with an adequate margin of safety."[182]

As discussed below, CASAC has also repeatedly and unanimously advised EPA that the current primary standard of 75 ppb is not protective of human health and recommended a more protective standard including after the standard was set, during the reconsideration, and in this current review.[183]

The scientific record before EPA today is inarguably stronger than in prior reviews, including those concluding in 2008 and 2011. EPA has determined that revising the primary standard is warranted to protect against adverse health effects that include decreased lung function and respiratory symptoms, serious morbidity, and premature mortality.[184] Since the last scientific review, evidence of health impacts caused by exposure to ozone pollution has strengthened significantly. The record shows extensive evidence that the present 75 ppb standard allows adverse health effects for millions of Americans each year, including early death, hospitalization, and asthma attacks. As described in the previous sections, controlled human exposure studies demonstrate adverse health impacts at levels below the current standard. In healthy adults and at exposures of only 6.6 hours, lung function decrements and respiratory symptoms are seen that clearly meet the ATS criteria for an adverse response.

Further, EPA has demonstrated the critical public health impacts experienced by concentrations at the current standard. According to the HREA's modeling, when meeting the current primary standard, 3-8 percent of children in the 15 urban study areas examined - or 900,000 children, including 90,000 children with asthma - would experience repeated exposures of concern to ozone concentrations higher than 60 ppb.[185] In years with especially poor air quality, over 14 percent of children would be expected to experience repeat exposures to ambient ozone concentrations above 60 ppb. Under the current standard, the HREA's numbers for single exposure incidents are even higher: 10-17 percent of children in the 15 urban areas would

[181] Proposed Rule at 75,236.
[182] Letter from U.S. EPA Administrator Lisa P. Jackson to the Honorable James Inhofe, May13, 2011. (See Exhibit 1).
[183] Letter from CASAC Chair Dr. Rogene Henderson to U.S. EPA Administrator Stephen L. Johnson on CASAC Recommendations Concerning the Final Rule for the National Ambient Air Quality Standards for Ozone (EPA-CASAC-08-009), Apr. 7, 2008 (See Exhibit 2); Letter from CASAC Chair Dr. Jonathan M. Samet to U.S. EPA Administrator Lisa P. Jackson on CASAC Response to Charge Questions on the Reconsideration of the 2008 Ozone National Ambient Air Quality Standards, (EPA-CASAC-11-004), Mar. 30, 2011 (See Exhibit 3); CASAC Letter 2014a.
[184] Policy Assessment at 3-1, 135-136.
[185] *Id.* at 3-100; HREA chapter 5.

experience (one or more) exposures to concentrations higher than 60 ppb - corresponding to almost 2.5 million children of whom 250,000 are asthmatics - and up to 26 percent of children in bad years.[186] The HREA predicts that if the current standard were met, 14 to 19 percent of children would experience lung function decrements, corresponding to approximately 3 million children (in the case study areas alone), of whom about 300,000 have asthma.[187] Nationally, far more children experience exposures of concern and lung function decrements. Thousands are estimated to die each year based upon exposure to ozone when complying with a primary standard of 75 ppb.[188]

Overwhelming evidence documents that the current standard fails to protect public health. EPA is correct, although too conservative, in concluding that the "the available evidence and exposure and risk information clearly calls into question the adequacy of public health protection provided by the current primary standard."[189]

1. Broad Scientific Consensus Supports a More Protective Standard Than 75 ppb

a. Since 2008 CASAC Has Consistently Supported Lowering the Standard Below 75 ppb

The Clean Air Act assigns responsibility for reviewing the air quality criteria and making recommendations on revisions of the NAAQS to the Clean Air Scientific Advisory Committee (CASAC). CASAC is an independent review committee comprised of leading scientific experts from universities, state government, and industry. Members of the committee have expertise in diverse scientific disciplines ranging from atmospheric science, toxicology, dosimetry, biostatistics, pulmonary medicine, epidemiology, ecology, risk assessment, monitoring, and other fields relevant to the review of the NAAQS. There are seven members of the statutory CASAC and additional consulting members are impaneled for each review to ensure that the committee has appropriate expertise in all pertinent disciplines. CASAC has an extensive record of providing independent peer review of EPA documents related to the ozone NAAQS.

Three separate CASAC committees, under three different Chairs, have unanimously confirmed that the current ozone standard is not protective of public health and that the standard should be set in the range of 60 to 70 ppb. Indeed, as noted below, CASAC has concluded that the standard should be set at the lower end of this range.

The first such committee was convened for the review that culminated in the 2008 revisions to the standard. From 2005 to 2008, CASAC reviewed two drafts of the Staff Paper, two drafts of the Criteria Document, two drafts of the risk assessment and two drafts of the exposure assessment. The Committee wrote to EPA Administrator Stephen L. Johnson three times, on October 24, 2006, March 26, 2007 and April 7, 2008, unanimously recommending selection of an 8-hour average ozone NAAQS within the range of 60 to 70 ppb and (Henderson,

[186] *Id*.
[187] Policy Assessment at 3-111; HREA chapter 6.
[188] *Id*.
[189] Policy Assessment at ES-3, 3-1.

taking the unusual step of commenting on the final rule (published Mar. 12, 2008) to register its concern that 75 ppb was not protective of public health.[190]

The second committee was constituted during the administrative reconsideration process following the 2008 review. In response to EPA's reconsideration of the 2008 ozone NAAQS and the proposal published on January 19, 2010, CASAC reaffirmed its support for the selection of an 8-hour average ozone NAAQS within the 60–70 ppb range.[191]

During the reconsideration process, EPA requested specific advice from CASAC on a number of important scientific questions under consideration. The Committee responded to these charges in an extremely detailed letter.[192] The letter again reaffirmed the Committee's scientific judgment that:

> the evidence from controlled human and epidemiological studies strongly supports the selection of a new primary ozone standard within the 60–70 ppb range for an 8-hour averaging time. As enumerated in the 2006 Criteria Document and other companion assessments, the evidence provides firm and sufficiently certain support for this recommended range for the standard.[193]

The second CASAC also reached a number of other specific findings and provided supporting evidence that is relevant to the current review:[194]

- Healthy individuals have been shown to have clinically relevant responses, even at 60 ppb.
- Since the majority of clinical studies involve young, healthy adult populations, less is known about health effects in such potentially ozone sensitive populations as the elderly, children and those with cardiopulmonary disease. For these susceptible groups, decrements in lung function may be greater than in healthy volunteers and are likely to have a greater clinical significance.
- Children and adults with asthma are at increased risk of acute exacerbations on or shortly after days when elevated ozone concentrations occur, even when exposures do not exceed the NAAQS concentration of 75 ppb.
- Large segments of the population fall into what EPA terms a "sensitive population group," i.e., those at increased risk because they are more intrinsically susceptible

[190] Letter from CASAC Chair Dr. Rogene Henderson to U.S. EPA Administrator Stephen L. Johnson on CASAC's Peer Review of the Agency's Final Ozone Staff Paper (EPA-CASAC-07-001), Oct. 24, 2006; Letter from CASAC Chair Dr. Rogene Henderson to U.S. EPA Administrator Stephen L. Johnson on CASAC's Review of the Agency's 2nd Draft Ozone Staff Paper (EPA-CASAC-07-002), Mar. 26, 2007; Letter from CASAC Chair Dr. Rogene Henderson to U.S. EPA Administrator Stephen L. Johnson on CASAC Recommendations Concerning the Final Rule for the National Ambient Air Quality Standards for Ozone (EPA-CASAC-08-009), Apr. 7, 2008.
[191] Letter from CASAC Chair Dr. Jonathan M. Samet to U.S. EPA Administrator Lisa P. Jackson on Review of EPA's Proposed Ozone National Ambient Air Quality Standard (*Federal Register*, Vol. 75, Nov. 11, Jan. 19, 2010) , (EPA-CASAC-10-007), Feb. 9, 2010.
[192] Letter from CASAC Chair Dr. Jonathan M. Samet to U.S. EPA Administrator Lisa P. Jackson on CASAC Response to Charge Questions on the Reconsideration of the 2008 Ozone National Ambient Air Quality Standards, (EPA-CASAC-11-004), Mar. 30, 2011.
[193] *Id*.
[194] *Id*.

(children, the elderly, and individuals with chronic lung disease) and those who are more vulnerable due to increased exposure because they work outside or live in areas that are more polluted than the mean levels in their communities.

These comments were based solely on the science record available for the 2008 review, for which the scientific record closed in 2006. The letter provides the Committee's consensus scientific advice regarding such important issues – such as the adversity of effects, the responses of sensitive populations, and other matters that will be discussed in more detail below.[195]

b. CASAC Concluded That a 70 ppb Standard Provides "Little" Margin of Safety and Specifically Cited a Level of 60 ppb as Meeting the Requirements to Protect Public Health.

The current CASAC committee has agreed with the previous two panels about the under-protectiveness of the current 75 ppb standard, and now underlined the limitations at the top end of a 60 to 70 ppb range. This panel included certain members who had served on the two previous panels, but also new members who had not previously served. All told, 44 individuals served on one or more CASAC ozone review panels. It is remarkable that 44 scientists from universities across the country and a range of expertise all reached a unanimous conclusion that the current standard fails to protect public health and that the 8-hour NAAQS must be revised downward to protect public health.

As discussed above in Section II.G., the Administrator is bound by the Clean Air Act to give due deference to the advice of CASAC. In light of this legal requirement, it is particularly indefensible that the proposal departs from the CASAC consensus recommendations by eliminating the more health-protective 60 ppb option specifically recommended by CASAC, especially in light of CASAC's finding that 60 ppb is a scientifically justifiable lower bound based on findings of adverse effects at that level. [196]

In its most recent letters to the EPA Administrator concerning the current review, the CASAC panel made clear that there is "sound" and "clear" scientific support to strengthen the standard to the lower end of a range between 60 and 70 ppb. Summarized below and discussed in more detail following, CASAC reached these conclusions about the range then under consideration:

- The current standard is inadequate to protect public health,
- At 70 ppb, there is substantial scientific evidence of adverse effects, including decrease in lung function, increase in respiratory symptoms, and increase in airway inflammation,

[195] Indeed, CASAC's conclusions that the current standard of 75 ppb is insufficient is consistent with EPA's own staff scientists' Policy Assessment "…available health evidence and exposure/risk information calls into question the adequacy of the public health protection provided by the current standard," Policy Assessment at 4-73, and that EPA should be considering a "range of 70 ppb to 60 ppb." *Id*. 4-75. In its review of the second draft Policy Assessment, CASAC took issue with EPA's staff recommendations, in advising that the upper end of the range be below 70 ppb.

[196] CASAC Letter 2014a at 7.

- The standard should be set below 70 ppb to meet the statutory requirement in the Clean Air Act to protect public health with an adequate margin of safety, and

CASAC has made expressly made clear that it views the higher end of the range that EPA proposes as insufficient to prevent adverse effects:

> At 70 ppb, there is substantial scientific evidence of adverse effects . . . including decrease in lung function, increase in respiratory symptoms, and increase in airway inflammation. Although a level of 70 ppb is more protective of public health than the current standard, it may not meet the statutory requirement to protect public health with an adequate margin of safety.
>
> Thus, our policy advice is to set the level of the standard lower than 70 ppb within a range down to 60 ppb, taking into account your judgment regarding the desired margin of safety to protect public health, and taking into account that lower levels will provide incrementally greater margins of safety.[197]

CASAC's advice in 2014 is considerably stronger and more explicit than CASAC's advice during the 2008 review and the reconsideration in 2011. Both previous reviews were based on a record of evidence that ended in 2006. However, the 2014 review is based on the overwhelming scientific and medical evidence of ozone's adverse health impacts--even at 70 ppb--that has amassed since 2006. And it is overwhelming. With more than 1,000 new studies included in the current review, the newer evidence documented the weaknesses of any potential standard above 60 ppb.

For example, in its letter to EPA, CASAC specifically found that the choice of 60 ppb as the lower end of the recommended range was justified scientifically "based on findings of adverse effects, including clinically significant lung function decrements and airway inflammation, after exposures to 60 ppb ozone in healthy adults with moderate exertion."[198] CASAC further emphasizes that these findings likely understate adverse impacts in sensitive populations. For example, children with asthma are likely to experience more significant adverse effects at 60 ppb than demonstrated in healthy adults: "[The] clinical studies do not address sensitive subgroups, such as children with asthma, and that there is a scientific basis to anticipate that the adverse effects for such subgroups are likely to be more significant at 60 ppb than for healthy adults."[199]

In writing to EPA, CASAC could not be clearer that exposures to 70 ppb ozone are of significant concern for children, asthmatics, the elderly and other susceptible populations.

> [A] level of 70 ppb offers only modest incremental reductions in short-term mortality risk compared to the current standard. Taken as a whole, these findings indicate that ozone exposures of 70 ppb pose reduced risk compared to the current standard but, nonetheless, are of significant concern, especially for children, asthmatics, the elderly and other at risk

[197] *Id.*
[198] *Id.*
[199] *Id.*

populations.[200]

The studies in the record, including the studies cited at length by CASAC to EPA, inform our understanding of the health impacts of ozone at low concentrations. As discussed in greater detail below, the ISA reaches stronger conclusions about the causality for a broad range of health endpoints, there is new evidence of adverse effects at low concentrations and there is new information on the extent of populations at risk since the last review. All of these factors informed CASAC's decision to find that adverse effects occur at 70 ppb, to recommend a range below 70 ppb, and to conclude that 60 ppb should be the lower end of the range considered based on findings of adverse effects at that level.

Given CASAC's clear recommendations, it is arbitrary and unlawful that EPA's Draft Rule proposes a range that essentially dismisses much of the evidence since 2006, as well as CASAC's assessment of the evidence. EPA's inclusion of 70 ppb in its proposed range when CASAC specifically found substantial scientific certainty of adverse effects at that level and that standard should be set below 70 ppb, runs counter to the evidence in the record and to EPA's own independent scientific advisors. And it is arbitrary and unlawful that EPA's proposed range fails to include the more health-protective half of the range recommended by CASAC, including the level of 60 ppb at which CASAC determined that adverse effects had been found. Indeed, where, as here, the evidence has strengthened since 2006, it is arbitrary for EPA to propose a less protective range than the 60-70 ppb range it proposed in the 2010 reconsideration rule that was based solely on that older evidence. Simply put, EPA's actions cannot be justified on the record before it, particularly why it has veered from years of unanimous recommendations by CASAC to include 60 ppb in the range under consideration.

c. The Children's Health Protection Advisory Committee Recommends a Standard of 60 ppb

EPA's own Children's Health Protection Advisory Committee (CHPAC) has been clear that the level of the NAAQS requisite to protect children's health is 60 ppb. The Committee is charged with advising the EPA regarding regulatory actions, research and communications related to children's health. The Committee is comprised of 24 experts on children's health from academia, state government, and the research community. CHPAC has repeatedly recommended that EPA revise the ozone NAAQS to 60 ppb. Most recently in 2014, the Committee stated:

> Children suffer a disproportionate burden of ozone-related health impacts due to critical developmental periods of lung growth in childhood and adolescence that can result in permanent disability. In addition, children have increased susceptibility due to increased ventilatory rates and increased outdoor physical activity compared with adults. The 6.8 million children suffering from asthma in the US are some of the most vulnerable to ozone-related respiratory impacts.[201]

[200] *Id*.

[201] Letter from CHPAC Chair Dr.Sheela Sathyanarayana to CASAC Chair Dr. Christopher Frey, May 19, 2014. (See Exhibit 4).

Because of the children's developmental stage and increased exposure to ozone, and in light of expanding evidence of adverse health effects in children, CHPAC recommended an 8-hour average ozone standard of 60 ppb:

> In 2007, the US EPA Children's Health Protection Advisory Committee (CHPAC) submitted two letters to Administrator Johnson that highlighted scientific findings regarding ozone-related children's health effects and urged [EPA] to support an ozone standard of 60 ppb in order to adequately protect children's health with a sufficient margin of safety. I am writing now to strongly re-affirm the recommendation of 60 ppb based on the expanding scientific evidence base documenting adverse childhood health impacts in relation to ambient ozone exposure. The higher end of the range, 60 ppb – 70 ppb, put forth by the Clean Air Scientific Advisory Committee (CASAC) in 2007 will not be sufficient to protect children's health.[202]

d. Medical and Public Health Organizations Support a Standard of 60 ppb

There is broad consensus in the medical community that the NAAQS must be revised to a level of 60 ppb to protect public health from harmful ambient ozone pollution.
On August 3, 2011, fourteen leading medical and public health organizations cosigned a letter to the President[203] stating the following:

> The ozone health standard must protect those who are most vulnerable from the dangerous health impacts of ozone, including infants, children, older adults, and those with chronic diseases. To safeguard the health of the American people, help to save lives, and reduce health care spending, we support the most protective standard under consideration: 60 parts per billion (ppb) averaged over eight hours.

Signers of the letter included American Academy of Pediatrics, American Association of Cardiovascular and Pulmonary Rehabilitation, American College of Preventive Medicine, American Heart Association, American Lung Association, American Public Health Association, American Thoracic Society, Asthma and Allergy Foundation of America, National Association for Medical Direction of Respiratory Care, National Association of County and City Health Officials, National Environmental Health Association, National Home Oxygen Patients Association, Physicians for Social Responsibility, and Trust for America's Health.

The American Thoracic Society (ATS), the leading medical association dedicated to advancing the clinical and scientific understanding of pulmonary diseases, speaks with authority on the adverse impacts of air pollution on health. ATS offered qualified applause to EPA's proposal, stating:

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[202] *Id*.
[203] Letter from medical and public health organizations to President Obama, Aug. 3, 2011, *available at* http://c.ymcdn.com/sites/www.acpm.org/resource/resmgr/policy-files/2011_ltr_presidentozone.pdf. (See Exhibit 5).
[203] ATS, *Press Release*: *ATS Applauds EPA's Proposed National Ambient Air Quality Standard for Ozone*, Nov. 26, 2014, *available at*http://www.thoracic.org/newsroom/press-releases/journal/articles/ats-applauds-epas-proposed-national-ambient-air-quality-standard-for-ozone.php.

> The ATS has long supported an 8-hour ozone standard of 0.060 parts per million (ppm) to best protect public health.
>
> Recent evidence links ozone pollution and adverse health effects including studies showing dose-response relationships between ozone exposure and hospital admissions for asthma in children and hospital admissions for asthma and COPD in adults, lung function deficits in healthy adults exposed to ozone at levels between 0.060 and 0.070 ppm, and an increased mortality risk associated with ozone exposure, primarily affecting the elderly and patients with chronic diseases. [204]

The American Thoracic Society discussed its position further in an editorial in the *American Journal of Respiratory and Critical Care Medicine*.[205]

> In 2007, 2010, and now again in 2014, the American Thoracic Society (ATS) has recommended that the U.S. Environmental Protection Agency (EPA) adopt an 8-hour ozone national ambient air quality standard of 60 ppb in order to adequately protect public health(1, 2). While the recommended standard endorsed by ATS has not changed during this time, the scientific evidence supporting this recommendation has significantly strengthened . . . .
>
> Highlights of this new body of evidence include a study of emergency department visits among children aged 0 to 4 in Atlanta, which found that each 30 ppb increase in the 3-day average of ozone was associated with an 8 percent higher risk of pneumonia and a 4 percent higher risk for upper respiratory infection(5) [Darrow et al 2014]. Several studies have demonstrated dose-response relationships between ozone exposure and childhood asthma admissions at exposure levels in the 60 to 80 ppb range (6–9) [Strickland et al 2014, Strickland et al 2010, Gleason et al 2014, Silverman et al 2010]. Similar associations have been found for adult admissions for asthma (9–11) [Silverman and Ito 2010, Glad et al 2012, Meg et al 2010] and COPD(12, 13) [Ko and Hui 2012, Media-Ramon et al 20076]. A population-based cohort study of generally healthy adults found that FEV1 was 56 mL lower after days when ambient ozone ranged from 59 to 75 ppb compared to days with levels under 59 ppb(14) [Rice et al 2013]. Controlled human exposure studies have re-affirmed lung function decrements in healthy adults after exposure to 60 to 70 ppb of ozone(15,16) [Schelegle et al 2009, Kim et al 2011]. Perhaps of greatest concern, there is now stronger evidence of increased mortality in association with ozone (17–19)[Peng et al 2013, Romieu et al 2012,Zanobetti and Schwartz 2008], particularly among the elderly and those with chronic disease(20, 21)[Medina-Ramon and Schwartz 2008, Zanobetti and Schwartz 2011]".

Likewise, the American Academy of Pediatrics has also endorsed a standard of 60 ppb:

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[204] ATS, *Press Release*: *ATS Applauds EPA's Proposed National Ambient Air Quality Standard for Ozone*, Nov. 26, 2014, *available at*http://www.thoracic.org/newsroom/press-releases/journal/articles/ats-applauds-epas-proposed-national-ambient-air-quality-standard-for-ozone.php.

[205] M.B. Rice, T.L. Guidotti, & K.R. Cromar (2014). on behalf of the ATS Environmental Health Policy Committee. Scientific Evidence Supports Stronger Limits on Ozone, *Am. J. Crit. Care Med*..

> Scientific evidence strongly supports a level of 60 parts per billion . . . . Ozone pollution in the air disproportionately impacts children . . . whose unique health and developmental needs make them more susceptible to pollutants. [206]

And the American Medical Association, the nation's largest medical society has also gone on record in support of a 60 ppb ozone standard in letters during the comment periods in both the previous review and the reconsideration. [207]

e. Many Other Countries, and the World Health Organization, Have Concluded That a Standard of 75 ppb Is Far Too Weak and that the 65 – 70 ppb Range is Insufficient to Protect Public Health.

i. World Health Organization

In October 2006, the World Health Organization (WHO) revised their international air quality guidelines for ozone.[208] The prior guideline for 8-hour average ozone concentrations of 120 µg/m3 (0.061 ppm) was reduced to 100 µg/m3 (0.051 ppm). The previous guideline and the new guideline are both substantially lower than both the current and proposed U.S. air quality standard. WHO provided a twofold basis for the revised guidelines. First, new epidemiological studies showed convincing evidence of associations between daily mortality and ozone levels. Similar associations have been observed in both North America and Europe. These time-series studies have shown effects without clear evidence of a threshold. Second, evidence from both chamber and field studies also indicated that there is considerable individual variation in response to ozone. According to WHO, the previously recommended guideline value, "which was fixed at 120 µg/m3 8-hour mean [61 ppb], has been reduced to 100 µg/m3 [51 ppb] based on recent conclusive associations between daily mortality and ozone levels occurring at ozone concentrations below 120 µg/m3."[209]

Likewise, Environment Canada and Health Canada adopted new ambient air quality standards for ozone in 2013 that are significantly more stringent than the range EPA is proposing in this process. The Canadian Ambient Air Quality Standard for ozone was lowered from the 8-hour standard of 65 ppb down to 63 ppb, to take effect in 2015, and to 62 ppb to take effect in 2020.[210]

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[206] James M. Perrin, MD, FAAP, President of the American Academy of Pediatrics, AAP Statement on New Ozone Standards Proposal, Nov. 26, 2014.
[207] Letter from American Thoracic Society, American Medical Association, et al. to U.S. EPA Administrator Lisa P. Jackson, Mar. 22, 2010.
[208] World Health Organization, WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global update 2005. Summary of risk assessment, *available at* http://www.who.int/phe/air/aqg2006execsum.pdf
[209] WHO, Ambient (outdoor) air quality http://www.who.int/mediacentre/factsheets/fs313/en/index.html
[210] Environment Canada, Backgrounder: Canadian Ambient Air Quality Standards, *available at* http://www.ec.gc.ca/default.asp?lang=En&n=56D4043B-1&news=A4B2C28A-2DFB-4BF4-8777-ADF29B4360BD (accessed Mar. 16, 2015).

Once a leader in environmental protection, the United States now lags behind other developed and developing nations in the protectiveness of air quality standards for ozone. As shown in Table 3 that follows, numerous developed and developing countries have promulgated ozone standards that are more stringent than the current U.S. standard, and that are significantly more stringent than even the 65-70 ppb standard that EPA is proposing.

Table 3: Comparison of Ozone Standards Worldwide (ppb)[211]

Country	1 hour	8 hour	Exceedances Allowed per year
WHO		51	
European Union -2010		61	25 (per 3 years)
Australia	100	80 (4-hr)	1
Cambodia	102		
Canada		65	3
(Jakarta)	102		
Ireland		61	
Japan	60		
Malaysia	102	61	
Mexico	110		1
Mongolia		61	
New Zealand	76		0
People's Republic of China (PRC) residential zone	61		
PRC commercial zone	82		
PRC industrial zone	102		
Republic of Korea	102	61	
Sri Lanka	102		
Switzerland		61	1
Thailand	102	71	
Viet Nam	92	61	
United Kingdom		51	10
United States		75	3

B. EPA's Proposal of 65-70 Fails to Protect the Public: EPA Must Revise the Primary NAAQS to a Level No Higher than 60 ppb

1. The Body of Science Evidence Demonstrates a Compelling Likelihood of Adverse Effects to Sensitive Populations at 60 ppb

As discussed at length above, there is overwhelming consensus among EPA's advisors, independent medical and public health organizations, and regulatory bodies in other countries that the current standard of 75 ppb is insufficiently protective of the public health, that the high

[211] Compiled from online sources, including: Clean Air Initiative, Clean Air Initiative for Cities Around the World, *available at* http://www.cleanairnet.org/caiasia/1412/articles-71889_Ozone_standards.pdf; UK-AIR, Air Information Resource; *available at* www.airquality.co.uk/archive/standards.php; Ireland EPA, Air Quality Standards, *available at* http://www.epa.ie/air/quality/standards/#.VQcN_2MhArg; Aarhus University Department of Environmental Science, Air Quality Standards: (limit values, target values, etc.) *available at* http://www2.dmu.dk/AtmosphericEnvironment/Expost/database/docs/AQ_limit_values.pdf.

end of EPA's proposed range under consideration—70 ppb—is insufficiently protective of public health, and that a lower level of 60 ppb is necessary to protect public health with an adequate margin of safety. As the following review of the primary scientific and medical research demonstrates, this consensus that 60 ppb is needed is well supported by the evidence in the record before EPA.

a. As the ISA Reflects, the Scientific Record is Significantly Stronger in this Review than Prior Reviews and Warrants a Standard of 60 ppb

In this review, the scientific evidence laid out in the ISA is more robust and compelling across the board than what was available during the review completed in 2008 and the reconsideration. Using an objective, transparent, and vetted set of criteria, the ISA characterizes the strength of evidence for various health endpoints. Compared to the 2008 review, the causal findings are strengthened from "suggestive" to "likely causal" for cardiovascular effects and total mortality from short-term exposures, and for respiratory effects from long-term exposures. Furthermore, the evidence for central nervous system effects from short-term exposure and for cardiovascular effects, reproductive and developmental effects, central nervous system effects, and total mortality from long-term exposure strengthened to "suggestive of a causal relationship." This stronger evidence necessitates greater public health protection.

Based upon the substantial new information available, the 2013 ISA reached much stronger conclusions about the health effects of ozone than had been reached in the prior review of the science nine years earlier. The criteria for evaluating studies and reaching causal determinations is carefully laid out in the ISA, and has been thoroughly vetted by CASAC. Conclusions are reached based on multiple lines of evidence and multiple studies, demonstrating coherence, consistency, and plausibility.

Specifically, the 2013 ISA reaches or identifies:

- A conclusive determination that ozone causes adverse respiratory effects;
- Several additional controlled human exposure studies demonstrating respiratory deficits and inflammation in healthy young adults at 60 ppb;
- Stronger findings that the adverse effect of ozone on cardiovascular health are likely causal;
- New information suggesting reproductive effects, such as increased risk of low birth weight babies;
- New information about suggestive central nervous system and neurological effects;
- New community health studies strengthening the link between ozone exposure and mortality, including at concentrations below the current standards; and
- New information about the impact of longer-term exposures on respiratory health endpoints such as pulmonary inflammation and injury, and new onset asthma.

Like the 2006 *Air Quality Criteria Document*, the 2013 ISA found there was a causal relationship between short-term exposure to ozone and respiratory effects. For almost every other health outcome and exposure duration evaluated, the ISA reached stronger causal

determinations in 2013 than in 2008. For three critical health outcomes, scientific evidence in 2013 was so strong enough to indicate a "likely causal relationship:"

- Cardiovascular effects from short-term exposures;
- Total mortality from short-term exposures; and
- Respiratory effects from long-term exposures.

ISA Table 1-1, below, compares the causal findings from the 2013 ISA with those of the 2006 *Criteria Document* and highlights those health outcomes for which the causal determination has been strengthened since the last review.[212]

Table 4. ISA Table 1-1 on summary of ozone causal determinations

Table 1-1 Summary of O_3 causal determinations by exposure duration and health outcome.

Health Outcome[a]	Conclusions from 2006 O_3 AQCD	Conclusions from this ISA
Short-term Exposure to O_3		
Respiratory effects	The overall evidence supports a causal relationship between acute ambient O_3 exposures and increased respiratory morbidity outcomes.	Causal Relationship
Cardiovascular effects	The limited evidence is highly suggestive that O_3 directly and/or indirectly contributes to cardiovascular-related morbidity, but much remains to be done to more fully substantiate the association.	Likely to be a Causal Relationship
Central nervous system effects	Toxicological studies report that acute exposures to O_3 are associated with alterations in neurotransmitters, motor activity, short and long term memory, sleep patterns, and histological signs of neurodegeneration.	Suggestive of a Causal Relationship
Total Mortality	The evidence is highly suggestive that O_3 directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality.	Likely to be a Causal Relationship
Long-term Exposure to O_3		
Respiratory effects	The current evidence is suggestive but inconclusive for respiratory health effects from long-term O_3 exposure.	Likely to be a Causal Relationship
Cardiovascular effects	No conclusions in the 2006 O_3 AQCD.	Suggestive of a Causal Relationship
Reproductive and developmental effects	Limited evidence for a relationship between air pollution and birth-related health outcomes, including mortality, premature births, low birth weights, and birth defects, with little evidence being found for O_3 effects.	Suggestive of a Causal Relationship
Central nervous system effects	Evidence regarding chronic exposure and neurobehavioral effects was not available.	Suggestive of a Causal Relationship
Cancer	Little evidence for a relationship between chronic O_3 exposure and increased risk of lung cancer.	Inadequate to Infer a Causal Relationship
Total Mortality	There is little evidence to suggest a causal relationship between chronic O_3 exposure and increased risk for mortality in humans.	Suggestive of a Causal Relationship

[a]Health effects (e.g., respiratory effects, cardiovascular effects) include a spectrum of outcomes, from measureable subclinical effects (e.g., blood pressure), to more obvious effects (e.g., medication use, hospital admissions), and cause-specific mortality. Total mortality includes all-cause (non-accidental) mortality, as well as cause-specific mortality (e.g., deaths due to heart attacks).

[212] ISA at 2-23.

Several other types of health effects are newly classified as suggestive of a causal relationship, by the 2013 ISA:

- Central nervous system effects from short-term exposures;
- Cardiovascular effects from long-term exposures;
- Neurological effects from long-term exposure; and
- Total mortality from long-term exposure.

These stronger causal determinations are a reflection of the strength of evidence given scientific advancements since the last review, and refute EPA claims that uncertainty justifies rejection of standards lower than 65 ppb or supports selection of levels at or near 70 ppb.

The level of the standard must be based on consideration of these stronger causal findings. The new evidence indicates that a standard of 60 ppb is needed to protect public health. The evidence base for ozone is stronger than for any other air pollutant. There are strong lines of evidence from all three major scientific disciplines: toxicology, epidemiology, and controlled human exposure studies. Under the Clean Air Act, EPA is obliged to set air quality standards that protect public health from proven, as well as anticipated health effects. Revisions to the standards must reflect the increased strength of the evidence, and the breadth of adverse health effects now attributable to ozone air pollution.

b. Controlled Studies Demonstrate Lung Function Decrements and Airway Inflammation at 60 ppb in Healthy Adults

As noted above, there are twice as many controlled human exposure studies available in this review. These studies show lung function decrements at 60 ppb in healthy young adults, including a new study indicating inflammation at 60 ppb. Again, inflammation is clearly an adverse effect, especially in asthmatics, and a ten percent decline in lung function, which was observed, is adverse for people with respiratory conditions whose breathing is already impaired. A primary standard below 60 ppb is needed to protect children, the elderly, and people with asthma.

Strong evidence of the adverse effects of ozone pollution comes from the controlled human exposure studies dating back to the 1980's. Studies where human volunteers are exposed to known concentrations of ozone in an experimental chamber are sometimes considered the gold standard in ozone research. Chamber studies have a number of advantages,[213] including known subject characteristics, precise measurement of pollutant concentration, elimination of confounding influences of temperature and other pollutants, precise measurement of effects, ability to assess acute and reversible effects, randomized and double blind designs, repeatability, temporal order of exposure and effects, and ability to demonstrate dose-response relationships.

At the same time, there are also substantial limitations with controlled human exposure studies, including:

[213] J. Q. Koenig (1993). Health Effects of Ambient Air Pollution: How Safe is the Air We Breathe? Norwell, MA: Kluwer Academic Publishers (2000); W.F. McDonnell, Utility of controlled human exposure studies for assessing the health effects of complex mixtures and indoor air pollutants, *Environ. Health Perspect.*, 101 Suppl 4: 199-203.

- Subjects are generally healthy, young adults-- not severely ill people or children;
- Only short-term exposures are feasible;
- Measurements must be minimally invasive;
- The studies cannot directly examine a pollutant's most severe effects;
- Inability to assess rare effects;
- Small number of subjects;
- Inability to study effects of repeated exposure;
- Statistical limitations of small sample size that make it difficult to detect small effects;
- Difficulty in studying complex pollutant mixtures;
- Homogeneous test subjects (generally healthy college students); and
- High costs to outfit laboratories and perform testing.

Of these limitations, several are of particular importance here. First, individuals tested in chamber studies are generally young, healthy, nonsmokers—that is, not children, not severely ill people, and not sensitive subpopulations. As discussed at length above, people with asthma, particularly children, have been found to be more sensitive and to experience larger decrements in lung function in response to ozone exposures than would healthy adults.[214] Mudway et al. 2004 noted that since chamber studies use only healthy subjects, individuals with lung disease or other risk factors will experience responses at even lower levels.[215]

Second, exposures in these studies were for 6.6 hours, not 8 hours. Ozone harm clearly increases with cumulative dose. A standard applying a longer exposure time than the study period, as EPA has previously used and proposed to use again for the ozone NAAQS, demands a lower level than that shown to induce adverse respiratory effects. In other words, if the study protocol is eliciting adverse effects at 80 ppb or 60 ppb after 6.6 hour exposures, a standard set for an 8-hour period must be somewhat lower than the level at which effects are observed because of the longer averaging time and greater accumulated dose of ozone.

Third, the full range of human responses cannot be detected in studies with a small number of subjects. By using a small and select group, usually of young, healthy adults, the studies fail are generally unable to access the full range of human responses and individual sensitivity.

Fourth, chamber studies do not fully capture the potential adverse effects of real world outdoor ozone exposures that invariably occur in combination with other pollutants and environmental conditions that can exacerbate ozone's harms.

It is important to keep these strengths and limitations in mind when discussing the controlled human exposure studies of ozone. It is because of the studies' limitations that it is also critical to consider evidence from other types of studies, including toxicological studies and epidemiological studies, in the discussion of health science. Consideration of all these types of

[214] Mortimer et al. 2002, *supra* note 85.
[215] I.S. Mudway & F.J. Kelly (2004). An Investigation of Inhaled Ozone Dose and the Magnitude of Airway Inflammation in Healthy Adults, *Am. J. Respir. Crit. Care Med.*, 169: 1089-1095.

studies and evidence together, as in the ISA and by CASAC, provides the most complete picture of ozone-related health effects and physiological mechanisms.

i. Controlled Human Exposure Studies Demonstrate Lung Function Decrements and Airway Inflammation at 60 ppb in Healthy Adults, as well as Pulmonary Inflammation, Injury, and Oxidative Stress

a. Lung Function Decrements and Airway Inflammation

Even in the 2008 review, there were several controlled human exposure studies that found evidence of harm from 6.6 hour exposures to 60 ppb ozone. Since the last review, several more important studies have been published reporting adverse effects at 70 ppb down to 60 ppb. In addition, two new models were been developed based on data from the controlled human exposure studies that make quantitative predictions of lung function response to ozone exposure and predict the degree of interindividual response to that exposure. This evidence demonstrates conclusively that the existing standard of 75 ppb (8 hour average) fails to protect public health.

Four separate studies discussed in the ISA evaluate exposures to 60 ppb ozone: Kim et al. 2011[216]; Schelegle et al. 2009[217]; Adams 2002[218]; and Adams 2006[219]. These studies found that ten percent of healthy young adults experience clinically significant declines in lung function when exposed for 6.6 hours to 60 ppb ozone, while engaged in moderate physical activity. Prolonged exposure to an average ozone concentration of 60 ppb results in group mean FEV_1 decrements ranging from 1.8 percent to 3.6 percent.[220] Based on data from these four studies, the weighted average group mean decrement was 2.7 percent. Some responsive subjects consistently experienced larger than average responses. This is important, as it provides insight into individual variable sensitivity, as discussed above.

The Adams 2002 study reported that the results of an unreleased 1998 study revealed that "some sensitive subjects experience notable effects at 0.06 ppm." This finding is based on the observation that 20 percent of the subjects exposed to 0.06 ppm ozone had a greater than 10 percent decrement in FEV_1. Even though the group mean response was not statistically different from the filtered air response, in a study with a small number of subjects the response of individual subjects is more important than the group mean response. This is particularly true for ozone exposure, where research has long recognized the variability in individual responses.

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[216] C.S. Kim et al. (2011). Lung function and inflammatory responses in healthy young adults exposed to 0.06 ppm ozone for 6.6 hours, *Am. J. Respir. Crit. Care Med.*, 183(9): 1215-21.
[217] E.S. Schelegle, C.A. Morales, W.F. Walby, S. Marion , & R.P. Allen (2009). 6.6-hour inhalation of ozone concentrations from 60 to 87 parts per billion in healthy humans, *Am. J. Respir. Crit. Care Med.*, 180(3): 265-72.
[218] W.C. Adams (2002). Comparison of chamber and face-mask 6.6 hour exposures to ozone on pulmonary function and symptoms responses, *Inhalation. Toxicol.*, 14: 745-764.
[219] W.C. Adams (2006). Comparison of chamber 6.6-h exposures to 0.04-0.08 PPM ozone via square-wave and triangular profiles on pulmonary responses, *Inhal. Toxicol*, 18(2): 127-36.
[220] Adams 2006 *id.*; Adams 2002, *supra* note 217; Schelegle et al. 2009, *supra* note 216; Kim et al. 2011, *supra* note 215.

A 2006 study by Adams showed respiratory symptoms and pain on deep inspiration, though did not find statistically significant lung function decrements at 6.6 hour exposures to 60 ppb. However, though not reported by the original investigators, a reanalysis by Brown et al. 2008[221] of the Adams 2006 data reported that FEV_1 decrements were highly statistically significant. As illustrated below in part A of Figure 3, the Brown et al. reanalysis found small, statistically significant declines in lung function using several common statistical tests, even after removal of three potential outliers.

The Brown et al. reanalysis found that the statistical techniques used in the Adams 2006 study were overly conservative for the evaluation of pre- to post-exposure changes in FEV_1, when comparing filtered air and ozone exposure. The reanalysis employed a standard approach used by other researchers. The pre- to post-exposure analysis showed that exposure to 60 ppb causes a small but statistically significant decrease in group mean FEV_1 responses compared to filtered air, as illustrated in following figure. Brown et al. did not reanalyze Adams 2006's respiratory symptoms data, but noted that Adams 2006 is "suggestive of an effect of 0.06 ppm on respiratory symptoms as well as FEV_1."[222]

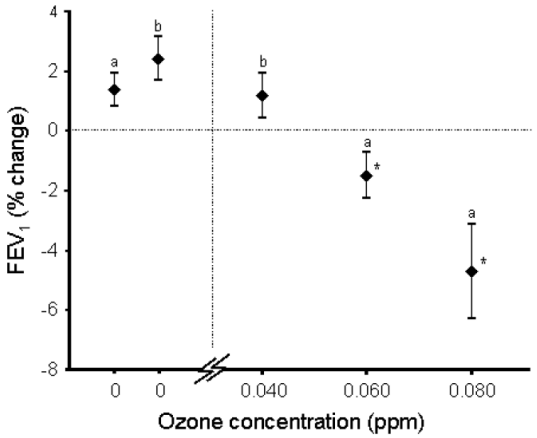

Figure 2. Effects of ozone on FEV_1 in healthy young adults exposed for 6.6 h during quasi continuous exercise to a constant (square-wave) O_3 concentration. Data are from a) Adams (2006) and b) Adams (2002). *Significantly different from responses to air exposure ($p \leq 0.001$, two-tail paired *t* test).

While the average response is relatively small in these studies, it is important because this is an average response in a sample size limited to healthy, young adults and not sensitive populations. The data show considerable variability in lung function responses between similarly exposed subjects, with some individuals experiencing distinctly larger effects even when the group mean responses are small.

[221] J.S. Brown, T.F. Bateson, & W.F. McDonnell (2008). Effects of exposure to 0.06 ppm ozone on FEV_1 in humans: A secondary analysis of existing data, *Env, Health Perspec.*, 116: 1023-1026.
[222] *Id*. at 1026.

Furthermore, when the Adams (2002, 2006) study data are corrected for the effect of exercise in clean air, 7 percent of subjects experience FEV1 decrements greater than 10 percent for ozone exposures of 0.04 ppm. Thirteen percent experience such decrements at 0.06 ppm,[223] and 23 percent at 0.08 ppm, as shown by EPA.[224] Larger decrements in FEV_1 would be expected in more susceptible populations. Adams 2006 reported total symptom scores and pain on deep inspiration following 60 ppb exposures and group mean FEV_1 responses during the 60 ppb exposures diverged from filtered-air and 40 ppb ozone exposures. The evaluation of pre- to post-exposure effects on both total subjective symptoms and pain on deep inspiration are indicative of significant respiratory symptom effects at 60 ppb ozone.

A more recent study, Schelegle et al. 2009 investigated the effect of 6.6-hour inhalation of ozone concentrations from 60 to 87 ppb in 31 healthy, young adults. Using a different statistical methodology than both Adams 2006 and Brown et al. 2008, this study reported statistically significant group mean FEV_1 decrements of 6.1 percent at 70 ppb, and a statistically significant increase in respiratory symptoms (compared to those observed in filtered air) in healthy individuals. These findings dispel any remaining arguments that the current standard of 75 ppb is protective of public health. The study also found decrements in lung function at 60 ppb, of approximately the same magnitude as reported in the Adams studies. Sixteen percent of the subjects tested had lung function decrements greater than ten percent at 60 ppb.[225]

In an editorial commenting on the Schelegle et al. 2009 study, Brown noted:

> *"There are at least three important findings from this study that have public health implications. First, statistically significant changes in FEV_1 and symptoms occurred in healthy individuals at 70 ppb. Second, the magnitude of the mean FEV_1 decrement (3.5% corrected for filtered air) at 60 ppb was about the same as reported by Adams. These findings further support a smooth dose-response curve without evidence of a threshold for exposures between 40 and 120 ppb O_3. Third, consistent with numerous studies, there is considerable intersubject variability in response to O_3. The distribution of response to O_3 becomes skewed with increasing concentration, with a few individuals exhibiting large FEV_1 decrements. Schelegle and colleagues found 16% of individuals to have greater than 10% FEV_1 decrements at 60 ppb, and this proportion increased to 19, 29, and 42% at 70, 80, and 87 ppb, respectively.*[226]

Most recently, Kim et al. 2011 investigated the effects of a 6.6-hour exposure to 60 ppb ozone during moderate exercise in young healthy adults. The study demonstrated statistically significant lung function deficits and inflammation compared to filtered air

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[223] In reporting results at 0.06 ppm, Figure 21 below only includes information from Adams (2006). Adams (2002) at 747, 761, reported that 6 of 30 (or 20%) of subjects experienced FEV_1 decrements $\geq$ 10% at 0.06 ppm. Thus, 8 of 60 subjects (or 13%) experienced such decrements.

[224] U.S. EPA (2007). Review of National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper (EPA-452/R-07-07), 3-7.

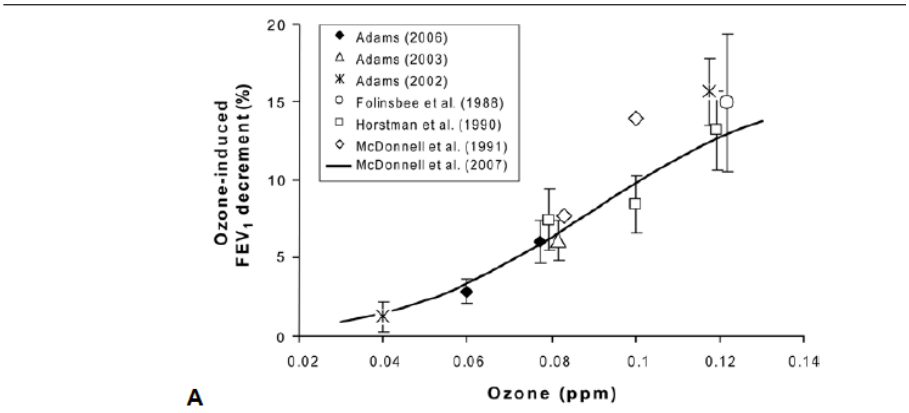
[225] Schelegle et al. 2009, *supra* note 216.

[226] .S. Brown (2009). Acute Effects of Exposure to Ozone in Humans: How Low Can Levels Be and Still Produce Effects?, *Am. J. Resp. Crit. Care Med.*, 180: 200-201.

exposures. Again, inflammation is an adverse health effect as discussed above. Unlike the earlier Adams 2006 and Schelegle et al. 2009 studies, which "were designed to compare multiple concentrations at multiple time points," this study "was specifically designed to limit the need for multiple comparisons" and thus gets more directly at the question of what effect 6.6-hour exposure to 60 ppb ozone has on young healthy adults.[227] In addition, this study provides important new evidence of airway inflammation, a mechanism by which ozone may cause other serious respiratory effects including asthma exacerbations. Persistent inflammation and injury, when observed in primate studies of chronic and intermittent exposure to ozone, is associated with remodeling of the airways.[228] Though the earlier Adams and Schelegle et al. studies did not examine inflammation responses to ozone exposure, CASAC states that decrements of 10 percent FEV_1 "are usually associated with inflammatory changes, such as more neutrophils in the bronchoalveolar lavage fluid."[229] Thus, inflammation likely resulted also from the exposures in those studies.

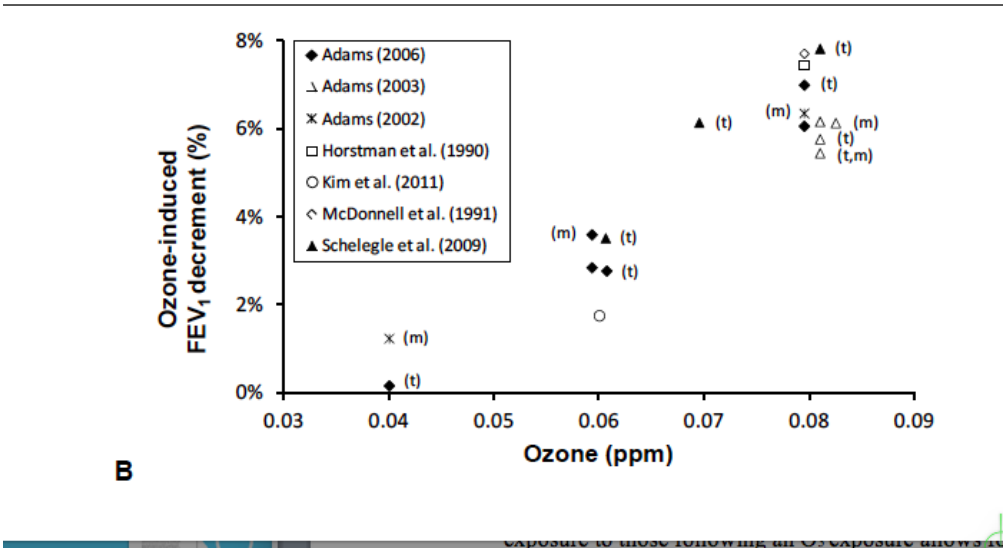
Given the results of these studies, the ISA's conclusion that lung function is reduced at 60 ppb--that "mean FEV_1 is clearly decreased by 6.6-hour exposures to 60 ppb O3 and higher concentrations in [healthy, young adult] subjects performing moderate exercise"—is inescapable.[230] Figure 6-1 of the ISA, below, illustrates that the group mean FEV_1 responses at 60 ppb fall on a smooth dose-response curve – without evidence of a threshold -- for exposures between 40 and 120 ppb ozone.

Figure 3. ISA Figure 6-1- Cross-study comparison of mean ozone-induced FEV_1 decrements following 6.6 hours of ozone exposure.



Source: Brown et al. (2008).

[227] Kim et al. 2011, *supra* note 215, at 1218.
[228] Guarnieri & Balmes 2014, *supra* note 75.
[229] Letter from CASAC Chair Dr. H. Christopher Frey to U.S. EPA Administrator Gina McCarthy re: CASAC Review of the EPA's *Health Risk and Exposure Assessment for Ozone (Second External Review Draft, February 2014)* (EPA-CASAC-14-005), 2, July 1, 2014 [hereinafter CASAC Letter 2014b] (response to charge questions, discussing HREA Fig.3-3).
[230] ISA at 6-9.



Based on Adams 2002, Adams 2006, Schelegle et al 2009, and Kim et al 2011, the weighted average proportion of individuals with greater than ten percent FEV_1 decrements is 10 percent following 6.6 hours of exposure to 60 ppb ozone and 25 percent following exposure to 80 ppb ozone.[231] Ten percent of responders is an underestimate because some subjects experienced greater than ten percent decrements in lung function. Further, some responses are not corrected for filtered air exposures, during which lung function typically improves, which would therefore increase the size of the change, pre-and post-exposure.[232]

A finding of 10 percent FEV_1 decrements and pulmonary inflammation at 60 ppb necessitates a NAAQS standard no higher than 60 ppb for several reasons. First, it cannot be emphasized enough that these are tests of healthy adults, not sensitive populations like children or individuals with asthma. Second, as the studies themselves evince, there is a significant amount of variation in individual responses, meaning that in the general population, a large subset of healthy adults will have similar significant, enhanced responses.[233] Third, and for these reasons, numerous authorities have concluded that such impacts constitute or are surrogates for adverse health effects.

For example, the American Thoracic Society views a 10 percent decrement in FEV_1 as an abnormal response and a reasonable criterion for assessing exercise-induced

[231] Final Policy Assessment at 3-14; ISA at 6-18.
[232] ISA sec. 6.2.1.1.
[233] As discussed above, some people experience enhanced responses to ozone exposure. This is significant because extrapolating results of clinical chamber studies from a small number of subjects, where the intersubject variability is less than for the general population, to the larger population will likely results in larger numbers of more sensitive people. Indeed, Schelegle et al. 2009 and Kim et al. 2011 added to the previously available evidence for interindividual variability in the responses of healthy adults following exposures to ozone. As the proposal points out, following prolonged exposures to 80 ppb ozone while at moderate exertion, the proportion of healthy adults experiencing FEV_1 decrements greater than 10 percent was 17 percent by Adams2006, 26 percent by McDonnell 1996, and 29 percent by Schelegle et al. 2009. Following exposures to 60 ppb ozone, that proportion was 20 percent by Adams 2002, 3 percent by Adams 2006, 16 percent by Schelegle et al. 2009, and 5 percent by Kim et al. 2011.

bronchoconstriction.[234] In previous NAAQS reviews, the EPA judged that for people with lung disease, moderate decrements in FEV_1 of greater than 10 percent but less than 20 percent lasting up to 24 hours would likely interfere with normal activity for many individuals, and would likely result in more frequent use of medication.[235] As discussed in Section IV.C above, more frequent use of medication is a change in clinical status that ATS considers to be adverse at the individual level. In the last review, CASAC advised that FEV decrements of 10 percent should be considered adverse in people with lung disease, especially children with asthma.[236] CASAC has stated that "[a] 10% decrement in FEV_1 can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (i.e., decreased baseline FEV_1) such that a $\geq$ 10 percent decrement could lead to moderate to severe respiratory symptoms."[237] In this review, CASAC has again said that an "FEV_1 decrement of $\geq$10% is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease."[238]

b. Pulmonary Inflammation, Injury, and Oxidative Stress

In addition to lung function and respiratory impacts, controlled human exposure studies also demonstrate that ozone exposures can result in increased respiratory tract inflammation and epithelial permeability. Earlier controlled human exposure studies performed at concentrations of 80 ppb and above frequently reported the presence of inflammatory markers such as polymorphonuclear neutrophils (PMNs) in the lungs. These neutrophils are the most abundant type of white blood cells in mammals. PMNs are recruited to the site of injury following trauma and are the hallmark of acute inflammation.

The findings of the earlier human exposure studies were reinforced by a meta-analysis of 21 human chamber studies where airway responses were assessed using bronchoscopy based lavage. Linear relationships were observed between ozone dose, airway inflammation, and protein leak into the airways over the early- and late-acute response time periods. Researchers found that exposure to 8-hour ozone concentrations of 0.08 ppm at moderate ventilation rates would be sufficient to trigger acute airway inflammation. The researchers noted that since chamber studies use only healthy subjects, individuals with lung disease or other risk factors will experience responses at even lower levels.[239] In addition, most controlled human exposure studies have reported that people with asthma experience larger ozone-induced inflammatory responses than do people without asthma.[240]

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[234] ATS 2000.
[235] 75 Fed. Reg. 2973 (Jan. 19, 2010).
[236] Letter from CASAC Chair Dr. Rogene Henderson to U.S. EPA Administrator Stephen L. Johnson on CASAC's Peer Review of the Agency's Final Ozone Staff Paper (EPA-CASAC-07-001), Oct. 24, 2006; Transcript of CASAC meeting, Aug. 24, 2006, at 149.
[237] Letter from CASAC Chair Dr. Jonathan M. Samet to U.S. EPA Administrator Lisa P. Jackson on CASAC Response to Charge Questions on the Reconsideration of the 2008 Ozone National Ambient Air Quality Standards, (EPA-CASAC-11-004), Mar. 30, 2011.
[238] CASAC Letter 2014a at 3.
[239] Mudway & Kelly 2004, *supra* note 214.
[240] Policy Assessment at 3-19.

In the current review, inflammation has been demonstrated following 6.6 hour exposures to 60 ppb.[241] Inflammation is a host response to injury, and the presence of inflammation an indication that injury has occurred.

Oxidative stress plays a key role in initiating and sustaining ozone-induced inflammation. The ISA explains that secondary oxidation products formed as a result of reactions between ozone and components of the epithelial lining fluid can increase the expression of cytokines and other molecules that enhance airway epithelium permeability.[242] Ozone exposures can initiate an acute inflammatory response throughout the respiratory tract that has been reported to persist for at least 18-24 hours after exposure.[243] The ISA notes that inflammation induced by ozone can have several potential outcomes:[244]

- It can resolve entirely;
- Continued acute inflammation can evolve into a chronic inflammatory state and can alter the structure and function of other pulmonary tissue, leading to diseases such as asthma;
- Inflammation can alter the body's host defense response to inhaled microorganisms, particularly in potentially at-risk populations or lifestages such as the very young and old; and
- Inflammation can alter the lung's response to other agents such as allergens or toxins.

Lung injury and the resulting inflammation provide a mechanism by which ozone may cause serious health effects such as asthma exacerbations. CASAC concurs that "[s]uch changes may be linked to the pathogenesis of chronic lung disease."[245]

Taken together, the chamber studies provide powerful evidence of the need to lower the 8-hour ozone standard to 60 ppb or below. Clearly, EPA's proposed standard of 65 to 70 ppb cannot be considered protective of public health in light of experimental evidence demonstrating adverse respiratory effects in healthy individuals exposed to 60 ppb, and the legal requirement to protect sensitive populations with an adequate margin of safety.

ii. New Models of Controlled Human Exposure Studies Support Data Showing Adverse Health Impacts at 60 ppb and Below

McDonnell et al. 2012[246] and Schelegle et al. 2012[247] developed models using data from a number of controlled human exposure studies on ozone exposure concentrations, ventilation rates, duration of exposures, and lung function responses. These models can make quantitative predictions of the potential lung function responses to ozone exposure, and the degree of

[241] Kim et al. 2011, *supra* note 215.
[242] ISA sec. 5.3.3 and 5.3.4.
[243] *Id*., sec. 6.2.3.
[244] *Id*.
[245] CASAC Letter 2014b at 2.
[246] W.F. McDonnell, P.W. Stewart, M.V. Smith, C.S. Kim, & E.S.Schelegle (2012). Prediction of lung function response for populations exposed to a wide range of ozone conditions, *Inhal. Toxicol.* 24(10): 619-33.
[247] E.S. Schelegle, W.C. Adams, W.F. Walby, & M.S. Marion (2012). Modelling of individual subject ozone exposure response kinetics, *Inhal. Toxicol.*, 24(7): 401-15.

interindividual variability.

These studies analyzed large datasets from the controlled human exposure studies to fit compartmental models to estimate the dose of onset in lung function response, or a response threshold based upon the inhaled ozone dose. The McDonnell et al. 2012 model was fit to a dataset consisting of the FEV_1 responses of 741 young, healthy adults (18-35 years of age) from 23 different controlled exposure studies. Concentrations across individual studies ranged from 40 ppb to 400 ppb, activity level ranged from rest to heavy exercise, and duration of exposure was from 2 to 7.6 hours. The Schelegle et al. 2012 model was fit to the FEV_1 responses of 220 healthy young adults from 21 controlled human exposure studies. The resulting responses to ozone in these studies were adjusted for responses observed following exposure to filtered air.

The models estimate the frequency distribution of individual responses for various exposure scenarios and can provide summary measures of the distribution such as the mean or median response and estimates of the proportions of individuals with FEV_1 decrements greater than 10, 15, and 20 percent. The dose is a function of exposure time as well as the level of exposure, as the models take into account the duration of exposure and level of oxidant stress in response to ozone exposure increasing over time as a function of dose rate, as well as decreasing over time by clearance or metabolization.

The results of the McDonnell and Schelegle models are consistent with real-life observed results from individual controlled human exposure studies of ozone-induced FEV_1 decrements.[248]

In applying their model, McDonnell et al. 2012 estimated that 9 percent of healthy exercising adults would experience FEV_1 decrements greater than 10 percent following 6.6 hour exposure to 60 ppb ozone, and that 22 percent would experience such decrements following exposure to 80 ppb ozone.[249]

Schelegle et al. (2012) estimated that, for 6.6 hours ozone exposure with moderate, exercise, the average dose of onset for FEV_1 decrement would be reached following 4 to 5 hours of exposure to 60 ppb, and following 3 to 4 hours of exposure to 80 ppb. However, 14 percent of individuals in the Schelegle model began responding after one to two hours exposure to 50-80 ppb ozone with moderate exercise. This estimate is consistent with the threshold FEV_1 responses reported by McDonnell et al. 2012.[250]

CASAC judged these models as an advance over the exposure-response modeling approach used in the last review:[251]

> the comparison of the MSS [McDonnell-Stewart-Smith] model results to those obtained with the exposure-response (E-R) model is of tremendous importance. Typically, the MSS model gives results about a factor of three higher than the E-R model for school-aged children, which is expected because the MSS model

[248] *See* Policy Assessment at 3-15.
[249] ISA at 6-18, Fig. 6-3.
[250] *Id*. at 6-16.
[251] CASAC 2014a at 7.

includes responses for a wider range of exposure protocols.

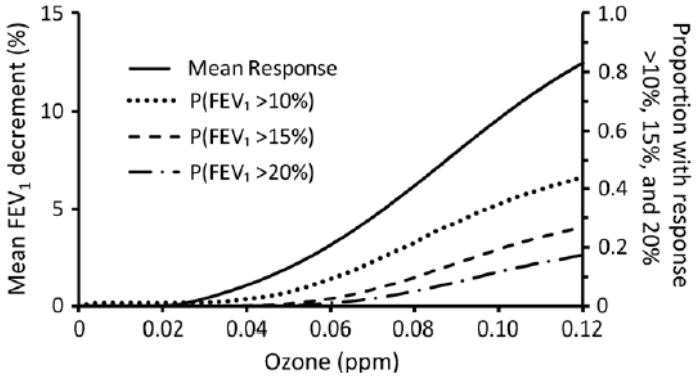
In addition to the consensus letter and responses to the charge questions, each individual member of the CASAC panel filed additional individual comments that were appended to the letter. As an appendix to these comments, we include relevant excerpts from these individual comments because they provide detailed insight into CASAC's rationale.

Consistent with the data from published studies, this model predicts that 9 percent of healthy people would suffer FEV1 decrements greater than 10 percent and 1 percent of people would experience FEV_1 decrements greater than 20 percent following 6.6 hour exposures to 60 ppb ozone, clearly indicating the need for a standard of 60 ppb, or below.

The exposure studies (and above-cited model based thereon) clearly demonstrate O3-induced adverse effects at 60 ppb ozone. Those studies are both corroborated by, and provide biological plausibility to, the epidemiological studies and other evidence showing adverse effects at and about 60 ppb. Accordingly EPA must set the primary standard at (or below) that level. *Lead Indus. Ass'n*, 647 F.2d at 1153 (primary standard must "be set at a level at which there is 'an absence of adverse effect' on [] sensitive individuals").

The models are consistent with the results of controlled human exposure studies finding adverse health impacts at 60 ppb. While the body of human exposure studies demonstrating lung function decrements and pulmonary inflammation at 60 ppb stand on their own and warrant a primary standard set at 60 ppb, these models provide significant additional support and verify the exposure study findings.

Figure 4. ISA Figure 6-3-Proportion of individuals predicted to have FEV_1 decrements following 6.6-hour exposures



Note: Predictions based threshold model of McDonnell et al. (2012) for healthy 23.8 year old adults exposed to a constant concentration of O_3 for 6.6 hrs. During each hour of the exposures, subjects were presumed engaged in moderate quasi continuous exercise (20 L/min/BSA) for 50 minutes and rest for 10 minutes. Following the third hour, subjects had an additional 35 minute rest period for lunch.

Source: Adapted from McDonnell et al. (2012).

Figure 6-3 Proportion of individuals predicted to have greater than 10%, 15%, and 20% O_3-induced FEV_1 decrements a following 6.6-hour exposure to O_3 with moderate exercise.

iii. New Research Confirms Additional Adverse Health Effects in Healthy Adults Exposed to Levels of 60 ppb for 6.6 Hours

Since the completion of the ISA, new information has been published reporting further analyses from the Kim et al controlled human exposure study at 60 ppb. This follow-up information provides important additional evidence of adverse health effects following 6.6 hour exposures to 60 ppb ozone. The new report provides additional measurements from the Kim et al controlled human exposure study including genotyping for genes reported to impact risk for responsiveness to ozone (GSTM1, NQO1, TNF), sputum cell assessment of markers of innate immune activation and function and inflammatory cells and cytokines.

Researchers reported that:

> individuals with an elevated PMN response to low level O3 are 13 times more likely of having the GSTM1null genotype than non-responders. Furthermore, responders have increased immuno-inflammatory responses to O3 compared to non-responders, and have elevated markers of inflammation following CA, suggesting the presence of a primed inflammatory airway in non-O3 exposed conditions. PMN responsiveness was also confirmed to be independent of the spirometric (FEV1) response to low level O3 in healthy people.

As the researchers explained, "Since GSTM1 is a risk factor for asthma exacerbation and ozone, these data support the hypothesis that genetic modifiers of oxidative stress modulate the health effects of O3 in individuals with allergic airways disease."[252]

c. Epidemiological Studies Confirm Meaningful Associations Between Mortality, Morbidity and Ozone Exposure at Levels Of 70 ppb and Below

i. Findings from Short-Term Epidemiological Studies Compel a Standard of 60 ppb

Epidemiological studies evaluated in the ISA provide valuable information on morbidity and mortality associated with low-level exposures to ozone, and on exposure-response relationships.

Many studies have demonstrated an independent association of short-term exposures of ozone to premature mortality, and respiratory and cardiac effects, often at mean or median concentrations of 60 ppb and below.

[252] N.E. Alexis et al. (2012). The Glutathione-S-Transferase null genotype and increased neutrophil response to low level ozone (0.06 ppm), *J. Allergy Clin. Immunol.*, 131(2): 610-612,

The strengths and limitations of epidemiological studies complement those of the controlled human exposure studies in many ways:

- While the subjects in the chamber study are healthy young adults, epidemiological and panel studies can include sensitive populations in the natural environment.

- Chamber studies can isolate the effect of a single pollutant, while real world exposures in epidemiological studies entail exposures to mixtures.

- Controlled human exposure studies can focus on acute effects, while epidemiological studies can explore chronic effects and serious health endpoints such as hospitalization and mortality.

As CASAC Chair Dr. Rogene Henderson aptly stated:

> *"The epidemiology data showing increased use of medication, school absences, and hospital admissions is one way to evaluate the response of sensitive populations to ozone. The controlled human exposures gives you a ceiling level which is higher than the level that would be protective of sensitive populations."*

ii. The Current Standard is Not Protective of Public Health

EPA's Policy Assessment highlights panel studies and epidemiological studies that were performed in areas that met the current air quality standard, or for multi-city studies, where the majority of locations would have met the current standard.[253]

This approach provides compelling evidence that the current standard is not protective of public health.

Table 3-2 from the Policy Assessment identifies five panel studies including a summer camp study of children, studies of outdoor workers, and studies of exercising adults that reported positive associations with lung function decrements and ozone concentrations of 75 ppb or below.

[253] Policy Assessment at 3-60–63.

Table 3-2. Panel studies of lung function decrements with analyses restricted to O_3 concentrations below 75 ppb.

Study	Population	O_3 Concentrations	Statistically Significant Association with Lung Function Decrements
Spektor et al. (1988a)	Children at summer camp	Restricted to 1-hour concentrations below 60 ppb	Yes
Chan and Wu (2005)	Mail carriers	Maximum 8-hour average was 65 ppb	Yes
Korrick et al. (1998)	Adult hikers	2- to 12-hour average from 40 to 74 ppb during hikes	Yes
Brauer et al. (1996)	Farm workers	Restricted to 1-hour maximum below 40 ppb	Yes
		Restricted to 1-hour maximum below 30 ppb	No
Brunekreef et al. (1994)	Exercising adults	Restricted to 10-minute to 2.4-hour averages below 61 ppb	No
		Restricted to 10-minute to 2.4-hour averages below 51 ppb	No
		Restricted to 10-minute to 2.4-hour averages below 41 ppb	No

These studies provide compelling evidence that the current standard fails to protect public health. Several of them are discussed in more detail below.

The study by Chan and Wu reported acute lung function decline in mail carriers exposed to ozone concentrations below the current ambient air quality standard.[254] The 8-hour average concentration of ozone in this study was 36 + 12 ppb (mean + SD), and the maximum 8-hour concentration was 65.1 ppb. For a 10 ppb increase in the 8-hour average ozone concentration, the night peak expiratory flow rate was decreased by 0.54 percent for a 0-day lag, 0.69 percent for a 1-day lag, and 0.52 percent for a 2-day lag.

[254] C. Chan & T. Wu (2005). Effects of ambient ozone exposure on mail carriers' peak expiratory flow rates, *Environ Health Perspect*, 113: 735-738.

Table 2

Summarized statistics for air pollutants and meteorologic data during the study period (14 November through 31 December 2001).

Variable	No.	Mean ± SD [a]	Minimum	Maximum
8-hr average during exposure periods				
O_3 (ppb)	44	35.6 ± 12.1	7.6	65.1
PM_{10} ($\mu g/m^3$)	43	74.7 ± 37.9	19.1	213.8
NO_2 (ppb)	43	30.0 ± 10.1	17.3	65.9
Temperature (°C)	45	19.1 ± 3.4	12.2	24.2
Relative humidity (%)	45	71.5 ± 6.6	59.0	88.0
Maximum during exposure periods				
O_3 (ppb)	44	52.6 ± 18.8	5.6	95.5
PM_{10} ($\mu g/m^3$)	43	106.8 ± 44.8	11.4	249.0
NO_2 (ppb)	43	52.9 ± 21.8	14.0	91.6

[a]Mail carriers' exposure periods are about 8 hr between 0900 and 1700 hr every working day.

The discussion in this paper pointed to earlier studies of adverse effects at concentrations below the current standard:

> *"Because none of our study subject's daily O3 exposure exceeded the hourly standard of 120 ppb, our study supports previous findings from studies in the United States and Canada of a dose-response relationship between lung function change and O3 exposure at relatively low daytime ambient concentrations for healthy adults. Exercising healthy adults in New York City (USA) who were exposed to < 80 ppb O3 were reported to have a 0.55-L/min decrease in their PEFR per 1 ppb O3 (Spektor et al. 1988); healthy women exposed to 8-hr O3 at 54 ppb in Connecticut and Virginia (USA) were reported to have a 0.083-L/min/ppb decrease in their PEFR per 1 ppb O3 (Naeher et al. 1999); farm workers in Fraser Valley (Canada) who were exposed to a 1-hr daily maximum O3 of 40 ppb were reported to have 3.3-mL and 4.7-mL decreases in their FEV1.0 and FVC, respectively, per 1 ppb O3 (Brauer et al. 1996). A similar dose-response relationship between O3 and PEFR reduction was also reported in some European studies. Male cyclists in the Netherlands who were exposed to < 60 ppb O3 were reported to have 0.57-L/min decreases in PEFR per 1 ppb O3 (Brunekreef et al. 1994); healthy workers and athletes in Germany who were exposed to < 80 ppb O3 were also reported to have decrements in their FEV1 (Hoppe et al. 1995)."*

Studies that excluded higher concentration days from the analysis that still find effects can provide very powerful evidence of effects at low concentrations.

Brauer et al. (1996) is an important such study of the effect ozone exposure on lung function of outdoor farm workers undertaken in the Fraser Valley of British Columbia.[255] The

[255] M. Brauer, J. Blair, & S. Vedal (1996). Effect of ambient ozone exposure on lung function in farm workers, *Am. J. of Resp. and Crit. Care Med.*, 154(4): 981-987.

mean daily maximum one-hour ozone concentration was 40 ppb (range: 13 to 84 ppb). Importantly, concentrations of acid aerosols and fine particulates, potential confounders of ozone effects, were very low. The study found that these exposures to ambient ozone were associated with decreased lung function over the day, which persisted to the following day.

Even after excluding all days when the ozone was greater than 40 ppb, investigators still observed reduced lung function, demonstrating adverse effects at very low concentrations. Outdoor workers are the population most likely to have prolonged exposure to ambient ozone under conditions of exercise. The express value of this study is that it is one of the few to focus on outdoor workers, a population especially susceptible to ozone exposures and health effects.

Brunekreef et al. (1994) is another such study that examined effects of ozone on a cohort of healthy young men who exercise outdoors—in this case, a group of amateur bicyclists in Netherlands.[256] Researchers collected lung function measurements before and after training sessions or competitive races during the summer of 1991. Ozone concentrations were low on most occasions, with an average of just 43 ppb. Eight-hour ozone concentrations exceeded 50 ppb only once during this study period, and concentrations of other pollutants were low. These low ozone concentrations were significantly associated with a decline in lung function over a race or training period. There was also an increase in respiratory symptoms, especially shortness of breath, chest tightness and wheeze in relation to ozone exposure. The effects persisted, even after removing all observations with hourly ozone greater than 60 ppb. This study provides vital evidence of the need for a 60 ppb standard or below.

In a study of hikers at Mount Washington in New Hampshire, Korrick et al. (1998) evaluated the effects of acute ozone, $PM_{2.5}$, and strong aerosol acidity on the pulmonary function of exercising adults.[257] The mean 8-hour ozone concentration in this study was 40 ppb, and the maximum was 74 ppb. Lung function was measured before and after hiking, with the greatest responsiveness to ozone observed in those with asthma or wheezing, or in those who hiked longer. Hikers with a history of physician-diagnosed asthma or severe wheeze had a fourfold greater responsiveness to ozone than other hikers. Furthermore, the results found that a substantially increased fraction of the exercising population had significant declines in lung function (>10% declines in FEF 25-75%) on days with higher ozone.

Kinney et al (1996) used bronchoalveolar lavage to assess biomarkers of lung inflammation in recreational joggers exposed to relatively low doses of ozone in the New York City metropolitan area.[258] Maximal hourly ozone concentrations on the day preceding the bronchoalveolar lavage ranged from 35 to 91 ppb, with a mean of 63 ppb. The average of daily maxima in the 7 and 28 days preceding the lavage were 56 ppb and 62 ppb, respectively. This study found that some of the individuals tested experience inflammation of the airways at concentrations of 60 ppb and below.

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[256] B. Brunekreef, et al. (1994). Respiratory effects of low-level photochemical air pollution in amateur cyclists, *Am. J. of Resp. and Crit. Care Med.*, 150(4): 962-966.

[257] S.A. Korrick et al.(1998). Effects of Ozone and Other Pollutants on the Pulmonary Function of Adult Hikers. *Env. Health Perspec*., 106: 93-99.

[258] P.L. Kinney et al. (1996), Biomarkers of lung inflammation in recreational joggers exposed to ozone *Am. J. of Resp. and Crit. Care Med.*, 154: 1430–1435.

iii. A Standard of 60 ppb is Needed to Protect Public Health

The Policy Assessment provides further analysis of additional studies that confirm evidence of harm at concentrations well below the current standard.

Many areas of Canada have cleaner air than parts of the U.S., making Canadian studies especially useful for evaluating health effects of air pollution at relatively low concentrations. The Policy Assessment identifies five multi-city and one single city study that would likely have met various alternative standards under consideration.

Shown below, Table 4-1from the Policy Assessment arrays information from these studies which reported that ozone is associated with an increased risk of hospital or emergency department visits for respiratory causes, or with premature death. The table shows that most of the study cities would have met a standard of 65 ppb during the entire study period, indicating that a standard of 65 would not be protective of public health. Even at 60 ppb, a number of study cities would have met the standard, suggesting that adverse effects persist at 60 ppb and that a lower standard is justified.

Table 4-1 Numbers of epidemiologic study locations likely to have met potential alternative standards with levels of 70, 65, and 60 ppb

			Number of study cities meeting potential alternative standards during entire study period		
Study	**Result**	**Cities**	**70 ppb**	**65 ppb**	**60 ppb**
Cakmak et al. (2006)	Positive and statistically significant association with respiratory hospital admissions	10 Canadian cities	7	6	2
Dales et al. (2006)	Positive and statistically significant association with respiratory hospital admissions	11 Canadian cities	5	4	0
Katsouyanni et al. (2009)	Positive and statistically significant associations with respiratory hospital admissions	12 Canadian cities	9	9	5
Katsouyanni et al. (2009)	Positive and statistically significant associations with total and cardiovascular mortality	12 Canadian cities	7	5	1
Mar and Koenig (2009)	Positive and statistically significant associations with asthma emergency department visits	Single city: Seattle	0	0	0
Stieb et al. (2009)	Positive and statistically significant association with respiratory emergency department visits	7 Canadian cities	5	4	3

These studies demonstrate serious adverse effects at low level exposures to ozone in the real world and they compel adoption of a final 8-hour average standard of 60 ppb.

Mar and Koenig[259] performed a time-series analysis using 1-hour and 8-hour ozone concentrations. They reported consistent positive associations across all lag times, with asthma emergency department visits for children in Seattle, a location that would have met the current standard over the entire study period. This analysis indicates that the current standard would allow concentrations of ozone shown to be associated with increased risk of respiratory emergency department visits for children.

Single city studies also document a correlation between high ozone days and hospital admissions for asthma. Silverman and Ito[260], for example, demonstrate a 19 percent increase in intensive care unit asthma admissions in New York hospitals on high ozone days. School-aged children ages 6-18 with asthma consistently had the highest risk. The Silverman and Ito study of asthma hospital admissions of children in New York City reported positive associations with ozone, even when over 99 percent of the days would have had 8-hour maximum ozone levels below the level of the current standard.

As illustrated in the figure below, the concentration-response relationship for ozone exposure and pediatric asthma emergency room visits in this study found no evidence of a threshold. Ozone concentrations as low as 30 ppb were associated with elevated rates of hospital admissions for asthma in children.

[259] T.F. Mar & J.Q. Koenig (2009). Relationship between visits to emergency departments for asthma and ozone exposure in greater Seattle, Washington, *Annals of Allergy, Asthma, and Immunol.*, 103(6): 474-479.
[260] R.A. Silverman & K. Ito (2010). Age-related association of fine particles and ozone with severe acute asthma in New York City, *J. Allergy Clin. Immunol.*, 125(2), 367-373.

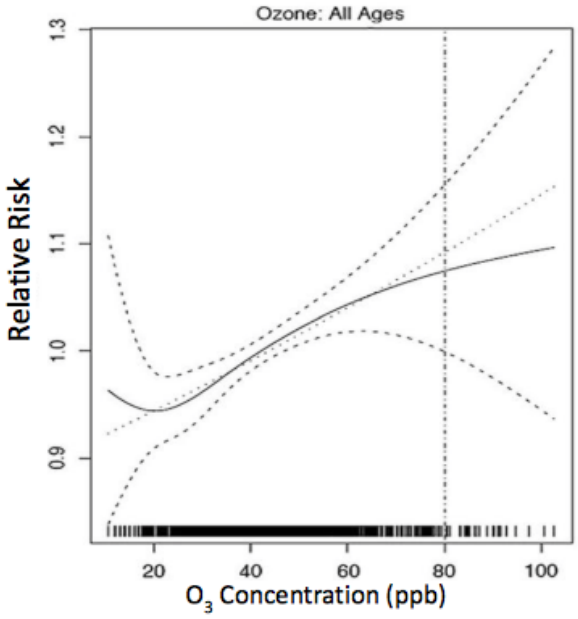


Figure 3-4. Concentration-response function for asthma hospital admissions over the distribution of area-wide averaged O_3 concentrations (adapted from Silverman and Ito, 2010).[50]

Strickland et al.[261] used population-weighting to combine daily pollutant concentrations across monitors in metropolitan Atlanta. The authors observed a 6.4 percent (95% CI: 3.2, 9.6%) increase in pediatric emergency room visits in hospitals throughout the metropolitan area, with a 30 ppb increase in 8-hour maximum ozone concentrations in the at all-year analysis. Stronger associations were observed during the warm season. Ozone risk estimates were not substantially changed when controlling for other pollutants. The study found elevated associations with ozone at 8-hour maximum concentrations as low as 30 ppb, with no evidence of a threshold.

[261] M.J. Strickland et al. (2010). Short-term associations between ambient air pollutants and pediatric asthma emergency department visits, *Am. J. of Resp. and Crit. Care Med.*, 182(3): 307-316.

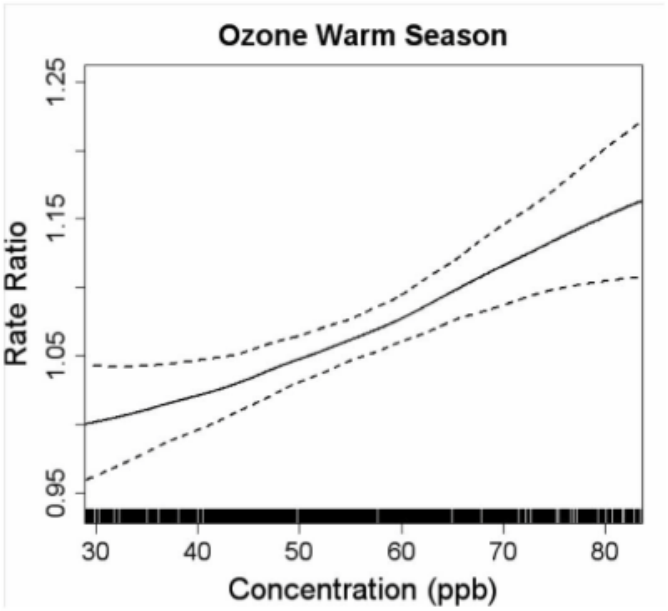


Figure 3-5. Concentration-response function for pediatric asthma emergency department visits over the distribution of averaged, population-weighted 8-hour O_3 concentrations (reprinted from Strickland et al., 2010).[53]

Loess C-R estimates and twice-standard error estimates from generalized additive models for associations between 8-hour max 3-day average O3 concentrations and ED visits for pediatric asthma. Originally published in Strickland et al., 2010.

In the analyses of the studies by Silverman and Ito, the Policy Assessment discusses a range of "averaged" concentrations of 26 to 45 ppb where the studies found a high degree of confidence in the statistical association with emergency department visits for respiratory events.[262] On more than 99 percent of the days when area-wide "averaged" ozone concentrations ranged from 26 and 45 ppb, the highest daily maximum 8-hour ozone concentrations were below 75 ppb.[263]

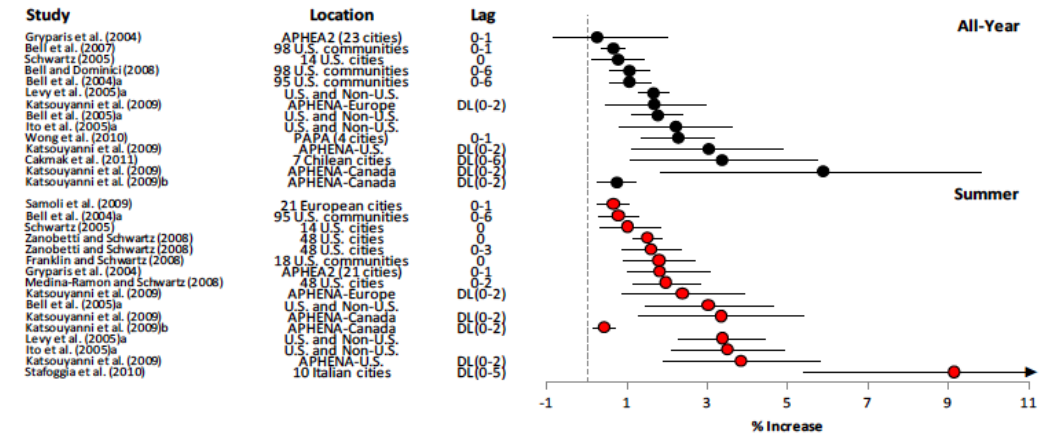
The Policy Assessment states that an examination of the concentration-response relationship for ozone exposure and pediatric asthma emergency department visits found no evidence of a threshold with elevated associations with ozone at concentrations as low as 30 ppb.[264]

These studies, which examine the impact of real world exposures among one of the most at risk populations—children with asthma—provide strong support for a standard of 60 ppb.

[262] Policy Assessment at 3-66-69.
[263] ISA at 3-67.
[264] Policy Assessment at 3.4.1.2, discussing Silverman and Ito 2010, *supra* note 259, and Strickland et al. 2010, *supra* note 260.

iv. Increased Evidence of Ozone-Caused Mortality in this Review

Substantial new information in this review supports earlier evidence that short-term exposures to ozone can increase the risk of premature death. Numerous epidemiological studies now have shown that short-term elevations in ozone pollution, particularly during the summer months, contribute to an increased risk of premature death, as illustrated in the following figure from the ISA.



Note: Effect estimates are for a 40 ppb increase in 1-h max, 30 ppb increase in 8-h max, and 20 ppb increase in 24-h avg O_3 concentrations. An "a" represent multicity studies and meta-analyses from the 2006 O_3 AQCD. Bell et al. (2005), Ito et al. (2005), and Levy et al. (2005) used a range of lag days in the meta-analysis: Lag 0, 1, 2, or average 0-1 or 1-2; single-day lags from 0 to 3; and lag 0 and 1-2; respectively. A "b" represents risk estimates from APHENA-Canada standardized to an approximate IQR of 5.1 ppb for a 1-h max increase in O_3 concentrations (see explanation in Section 6.2.7.2).

Figure 6-27 Summary of mortality risk estimates for short-term O_3 exposure and all-cause (nonaccidental) mortality from all-year and summer season analyses.

A significant body of strong, consistent evidence links short-term exposures to ozone to premature deaths. The substantiation rests in a growing number of epidemiological studies supplemented by emerging animal research providing evidence of biological plausibility.

The ISA reports that a dozen newer multi-city studies and single-city studies, and several meta-analyses of these studies, have provided increased evidence for associations between short-term ozone exposure and total mortality, even after adjustment for the influence of season and particulate matter.

In summarizing the evidence, the Policy Assessment points to multi-city studies from the U.S. (Zanobetti and Schwartz 2008),[265], Europe (Samoli et al. 2009),[266] Italy (Stafoggia et al.

[265] A. Zanobetti & J. Schwartz (2008). Mortality displacement in the association of ozone with mortality: an analysis of 48 cities in the United States, *Am. J. Respir. Crit. Care Med.*, 177 (2): 184-9.
[266] E. Samoli et al. (2009). The temporal pattern of mortality responses to ambient ozone in the APHEA project, *J. Epidemiol. Community Health*; 63(12): 960-6.

2010),[267] and Asia (Wong et al. 2010),[268] as well as a multi-continent study (Katsouyanni et al. 2009)[269], that report associations between short-term ozone exposures and mortality from respiratory causes.[270] The studies of respiratory mortality during the summer ozone season were consistently positive, and most were statistically significant.

The biological plausibility of the ozone-mortality link is supported by the experimental evidence of respiratory effects. The evidence cited in the ISA substantiates that increased risk of mortality is evident at levels well below the proposed standard. The study designs have taken a variety of approaches including single- and multi-city time series and case-crossover analyses. They have explored potential confounding by temperature, and other pollutants. The discussion below touches briefly on the results of those studies and emerging evidence of the possible biological mechanisms at work. The mounting evidence provides powerful support for selecting a standard no higher than 60 ppb.

Two critical multi-city studies published in 2004 show clear evidence of the ozone-related mortality risk in the U.S. and Europe. Bell et al. published a large 14-year study of residents of 95 U.S. cities, in which short-term increases in ozone were found to increase total non-accidental mortality and deaths from cardiovascular and respiratory causes.[271] A major 23-city European study by Gryparis et al.[272] reported a positive association between one- and eight-hour concentrations of ozone air pollution and daily mortality, especially respiratory mortality, during the warm season.

People may die from ozone exposure even when concentrations are well below the current standards. Bell and colleagues followed up on their 2004 multi-city study to estimate the exposure-response curve for ozone and risk of mortality and to evaluate whether a threshold exists below which there is no effect.[273] They applied several statistical models to data on air pollution, weather, and mortality for 98 U.S. urban communities for the period 1987-2000. The results provide strong and consistent evidence that daily changes in ozone pollution are linked to premature death. The ozone and mortality results do not appear to be confounded by temperature or PM_{10}.

Significantly, as indicated in the figure below, the relationship between mortality and ozone was evident even on days when pollution levels were well below 60 ppb.

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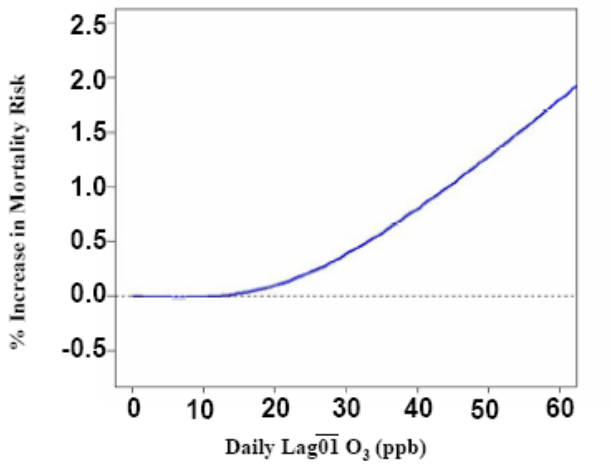
[267] M. Stafoggia (2010). Susceptibility factors to ozone-related mortality: a population-based case-crossover analysis, *Am. J. Respir. Crit. Care Med.*, 182(3): 376-84.
[268] Wong et al. (2010). Part 5. Public health and air pollution in Asia (PAPA): a combined analysis of four studies of air pollution and mortality. Res. Rep, Health Eff. Inst., (154): 377-418.
[269] K. Katsouyanni (2010). Air pollution and health: a European and North American approach (APHENA). Res. Rep, Health Eff. Inst., (142): 5-90.
[270] Policy Assessment at 3-35 – 36.
[271] M.L. Bell et al. (2004). Ozone and short-term mortality in 95 US urban communities, 1987-2000, *JAMA*, 292: 2372-2378.
[272] A. Gryparis et al. (2004). Acute effects of ozone on mortality from the "air pollution and health: a European approach" project, *Am. J. Respir. Crit. Care Med.*, 170 (10): 1080-7.
[273] M.L. Bell et al. (2006). The Exposure-Response Curve for Ozone and Risk of Mortality and Adequacy of Current Ozone Regulations, *Environ. Health Perspect.*, 114(4): 32-536.



Exposure Response Curve for ozone and mortality using the spline approach: percentage increase in daily nonaccidental mortality at various ozone concentrations. Originally published in Bell, et al. 2006, taken from Bell, ML "Recent Evidence on the Relationship between Ozone and Mortality," Presentation to the Estimating Mortality Risk Reduction Benefits from Decreasing Tropospheric Ozone Exposure Panel, National Research Council on March 29, 2007.

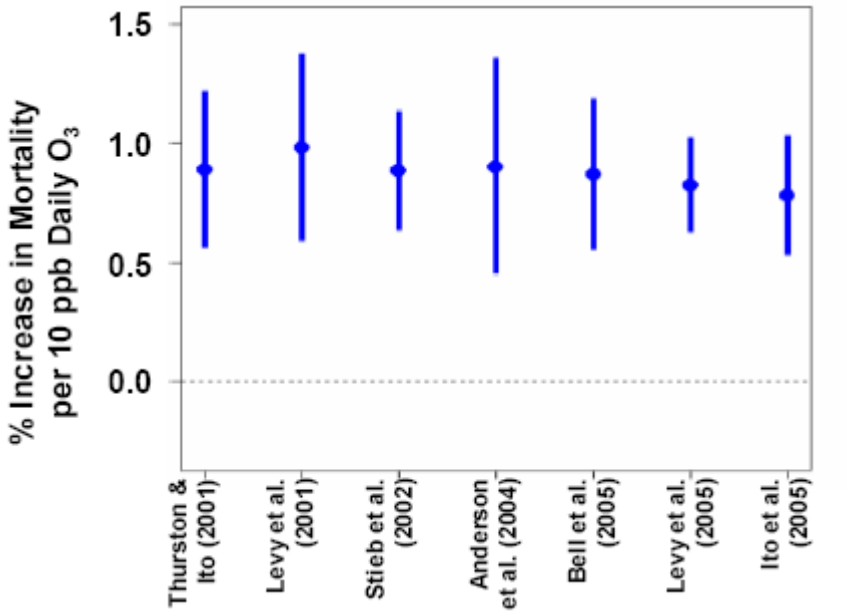
Zanobetti and Schwartz (2011) conducted a large multicity study of 48 U.S. cities and reported a positive association between ozone and all-cause mortality during the summer months.[274] The researchers found that ozone was also associated with deaths from cardiovascular disease, strokes, and respiratory causes. Mean 8-hour ozone concentrations in the study ranged by city from 15.1 to 62.8 ppb. The 75th percentile 8-hour ozone concentrations ranged from 19.8 ppb in Honolulu to 75.9 ppb in Los Angeles.

Franklin and Schwartz (2008) studied 18 U.S. communities and reported an association between summertime ozone levels and non-accidental mortality.[275] This association was robust to the inclusion of $PM_{2.5}$ in the analysis, strengthening confidence in the ozone-mortality link. The researchers concluded that the association of ozone with daily deaths in the summer does not represent short-term mortality displacement and is an issue of public health concern. The study found that the impact of ozone on mortality was reduced when sulfate exposures were also taken into account. Average daily ozone concentrations in the study ranged by community from 21.4 in Seattle to 48.7 ppb in Fresno.

Meta-analyses offer compelling evidence that these ozone-mortality findings are consistent. Four meta-analyses completed between 2001 and 2004 reported evidence that ozone

[274] A. Zanobetti & J. Schwartz (2008). Mortality displacement in the association of ozone with mortality: an analysis of 48 cities in the United States, *Am. J. Respir. Crit. Care Med.*, 177 (2): 184-9.
[275] M. Franklin & J. Schwartz (2008). The impact of secondary particles on the association between ambient ozone and mortality, *Environ. Health Perspect.*,116: 453-458.

contributes to early death.[276] Three independent analyses in 2005 used statistical techniques to synthesize the results of different studies of ozone and mortality. Separate research groups from Johns Hopkins University, Harvard University, and New York University conducted independent meta-analyses, using their own study selection criteria and methods. All three meta-analyses reported a remarkably consistent link between daily ozone levels and total mortality.[277,278,279] The results of these meta-analyses are summarized in the figure below, which illustrates the consistency in the findings.



Results of the Meta-Analyses studies. From Bell, ML. "Recent Evidence on the Relationship between Ozone and Mortality," Presentation to the Estimating Mortality Risk Reduction Benefits from Decreasing Tropospheric Ozone Exposure Panel, National Research Council on March 29, 2007.

[276] J.I. Levy (2001). Assessing the Public Health Benefits of Reduced Ozone Concentrations. *Environ. Health Perspect.*, 109: 1215-1226; G.D. Thurston & K. Ito K.(2001). Epidemiological Studies of Ozone Exposures and Acute Mortality, *J. Exposure Analysis and Environ. Epidemiology*, 11: 286-294; H.R. Anderson et al. (2004). Meta-Analysis of Time-Series Studies and Panel Studies of Particulate Matter (PM) and Ozone (O_3). Report of a WHO Task Group. Copenhagen: World Health Organization; D.M. Stieb, S. Judek, & R.T. Burnett (2002). Meta-analysis of time-series studies of air pollution and mortality: Effects of gases and particles and the influence of cause of death, age and season. *J .Air & Waste Manage Assoc.*, 52: 470-84.

[277] M.L. Bell, F. Dominici, & J.M. Samet (2005). A Meta-Analysis of Time-Series Studies of Ozone and Mortality with Comparison to the National Morbidity, Mortality, and Air Pollution Study. *Epidemiology*, 16: 436-445.

[278] J.I. Levy, S.M. Chermerynski, & J.A. Sarnat (2005). Ozone Exposure and Mortality: An Empiric Bayes Metaregression Analysis, *Epidemiology*, 16: 458-468.

[279] K. Ito, S.F. De Leon, & M. Lippmann (2005). Associations Between Ozone and Daily Mortality: Analysis and Meta-Analysis. *Epidemiology*, 16: 446-429.

Analyses clearly indicate that the death effect of ozone is distinct from the effect of temperature and particle pollution. Bell et al. (2007)[280] analyzed the effect of PM on the association between short-term exposure to ozone and mortality, using data from 98 U.S. communities. By estimating the correlation between daily PM and ozone concentrations, and including PM as a covariate in various models, Bell et al. concluded that neither PM_{10} nor $PM_{2.5}$ is a likely confounder of the observed relationship between ozone and mortality.

A case-crossover study of 14 U.S. cities was designed to control for the effect of temperature on daily deaths.[281] The study concluded that the association between ozone and mortality risk reported in the multi-city studies is unlikely to be due to confounding by temperature.

v. Biological Mechanisms

Evidence is emerging on biological mechanisms. A review article offers possible mechanisms for altered morbidity and mortality associated with ozone air pollution, related to a complex interaction with the innate immune system.[282]

As shown in the figure below, inhalation of ozone impairs antibacterial defense in many types of cells in the lung. Ozone can disrupt the epithelial barrier and mucociliary clearance and can induce production of proinflammatory factors. Ozone is directly cytotoxic to macrophages. Ozone can modify macrophage phagocytosis of microbial pathogens, intracellular killing, and levels of secreted factors.

[280] M.L. Bell, J.Y. Kim, & F. Dominici (2007). Potential confounding of particulate matter on the short-term association between ozone and mortality in multisite time-series studies. *Environ. Health Perspect*., 115(11): 1591-5.
[281] J. Schwartz (2005). How sensitive is the association between ozone and daily deaths to control for temperature? *Am. J. Resp. Crit. Care Med.*, 171: 627- 631.
[282] J.W. Hollingsworth, S.R. Kleeberger, & W.M. Foster (2007). Ozone and Pulmonary Innate Immunity. *Proc. Am. Thorac. Soc.*, 4: 240-246.

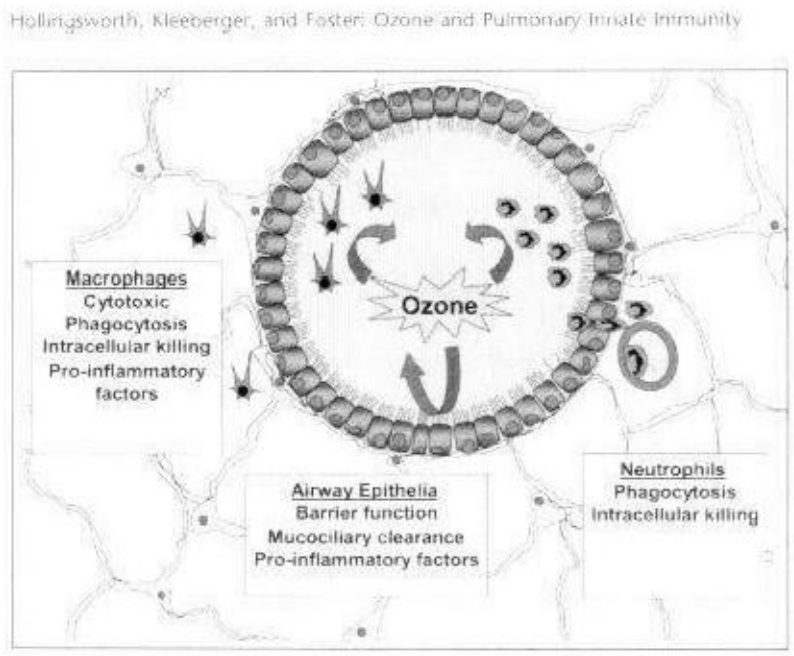


Illustration of possible mechanisms for ozone interaction with cells in the lungs. From Hollingsworth et al 2007.

An animal study takes this research further. Hollingsworth and colleagues found that ozone shuts down the responses of the immune system in the lungs of mice, making them more responsive, and therefore more vulnerable to infections and diseases.[283] Ozone primes the immune system to hyper-respond and destroys some of the protective immune cells, leaving the lungs vulnerable to later bacterial infections.

vi. Confidence in the Associations Shown Does Not Decrease at 60 ppb

EPA specifically posted this question to the CASAC:

"To what extent does your confidence that the effects observed in epidemiological studies are attributable specifically to O3 lessen or otherwise change, if at all, at the lower levels in the proposed range [60-70 ppb] as compared to the higher levels?"

CASAC's unanimous response to this question belies EPA's assertion that that there is increased uncertainty of effects at lower levels such as 60 ppb:[284]

"While epidemiological studies are inherently more uncertain as exposures and risk estimates decrease (due to the greater potential for biases to dominate small effect estimates), specific evidence in the literature does not suggest that our confidence on the

[283] J.W. Hollingsworth et al. (2007). Ambient Ozone Primes Pulmonary Innate Immunity in Mice. *J. Immunology*, 179: 4367-4375

[284] Letter from CASAC Chair Dr. Jonathan M. Samet to U.S. EPA Administrator Lisa P. Jackson on CASAC Response to Charge Questions on the Reconsideration of the 2008 Ozone National Ambient Air Quality Standards, (EPA-CASAC-11-004), 10, Mar. 30, 2011

specific attribution of the estimated effects of ozone on health outcomes differs over the proposed range of 60-70 ppb. In framing our answer to this question, we note that the range covered is quite narrow and we would not anticipate major differences in the characteristics of the pollution mixture across this range."

Regarding the epidemiological studies, the Committee went on to state its concern for effects ranging from increased risk of morality to lung function declines:

"Several distinct classes of epidemiological studies are relevant in this range and some examples are given below. For instance, mortality effects for ozone have been found in time-series studies in communities where mean ambient concentrations are well below the proposed range (e.g., Vedal et al 2003). Exercise-induced decrements in lung function, known to be causally related to ozone in controlled exposure studies, have been observed in field studies of healthy volunteers. For instance, in a cross-sectional study, Korrick et al. (1998) found that hikers on Mount Washington experienced significant decreases in FEV1 after prolonged exercise on days when ozone averaged 40 ppb (range 21 to 74 ppb). The magnitude of these decrements increased as mean ozone levels increased and it was nearly fourfold higher for persons with asthma than for persons without asthma. Panel studies of campers are yet another class of field studies that have shown effects on children's lung function are associated with ambient ozone. For example, in a panel of healthy children, Spektor et al. (1988) showed significant reductions in FEV1 associated with one-hour average ambient ozone, even when restricted to days with ozone below 60 ppb. Similarly, in panels of children with moderate to severe asthma attending summer camp, Thurston et al. (1997) reported not only respiratory function changes, but also more clinically significant responses, including increases in physician prescribed rescue medication and respiratory symptoms. In yet another class of epidemiological studies, health care utilization for asthma has been shown to decrease when ozone concentrations decreased. For example, Friedman et al (2001) found that during the Summer Olympic Games in Atlanta in 1996 there was significantly decreased use of pediatric care for asthma that correlated best with a reduction in peak ozone concentrations. In this study, the relative risk of asthma events increased stepwise at cumulative ozone concentrations 60 to 89 ppb and 90 ppb or more compared with ozone concentrations of less than 60 ppb. The reduction of the adverse effects on asthma in this study was dependent on reduction of ozone exposures to levels below 60 ppb."

"Our confidence that the effects from epidemiological studies are attributable to ozone is also bolstered by the recognition that the endpoints of concern do not change at the lower levels of the proposed range. While it may be difficult to disentangle the effect of a single pollutant in epidemiological studies, the evidence regarding ozone-related health effects from epidemiological studies is consistent with the evidence from controlled exposure studies that involve ozone alone. Indeed, evidence from observational studies of individuals exercising outdoors indicates ozone may have even stronger lung function effects than those estimated in controlled exposure studies, suggesting the possibility that a mixture of photochemical oxidants may be more toxic than ozone alone. Finally, whether or not the effects attributed to ozone in epidemiological studies are specific to

ozone vs. the entire photochemical oxidant pollutant mixture, it is likely that reductions in population exposures to ozone will result in fewer adverse health effects. Our confidence in this statement does not change at the lower levels of the proposed range."[285]

vii. Effects Persist Even After Excluding High Concentration Days above a Certain Level

Studies that find positive effects after excluding days above a certain concentration provide compelling evidence of associations evident at low concentrations and are especially pertinent to the setting of air quality standards. Some important examples include:

- Brunekreef et al. 1994[286]: Even after removing all observations with hourly ozone concentrations greater than 60 ppb, researchers found a decline in lung function and an increase in respiratory symptoms in this group of amateur cyclists.

- Brauer et al. 1996[287]: Even after excluding all days when the ozone was greater than 40 ppb, investigators still observed reduced lung function in a cohort of outdoor workers.

- Mortimer et al. 2002[288]: After excluding days when 8-hour average ozone was greater than 80 ppb, the associations with morning lung function decrements remained statistically significant.

- Bell et al. 2004[289]: Estimates of premature mortality attributable to ozone changed little when days with 24-hour average concentrations greater than 60 ppb were excluded.

- Bell et al. 2006[290]: There was little difference in the mortality effect estimate when days with 24-hour ozone concentrations above 20 ppb were excluded.

- Spektor et al. (1988)[291]: Statistically significant reductions in FEV_1 were associated with one-hour average ambient ozone, even when restricted to days with ozone below 60 ppb.

viii. EPA Must Base Standards on Concentrations Below the Mean in Epidemiological Studies

[285] *Id*. at 10-11.
[286] B. Brunekreef, et al. (1994). Respiratory effects of low-level photochemical air pollution in amateur cyclists, *Am. J. of Resp. and Crit. Care Med.*, 150(4): 962-966.
[287] M. Brauer, J. Blair, & S. Vedal (1996). Effect of ambient ozone exposure on lung function in farm workers, *Am. J. of Resp. and Crit. Care Med.*, 154(4): 981-987.
[288] K.M. Mortimer, L.M. Neas, D.W. Dockery, S. Redline, & I.B. Tager (2002). The effect of air pollution on inner-city children with asthma. *Eur. Respir. J.*, 19(4): 699–705.
[289] M.L. Bell et al. (2004). Ozone and short-term mortality in 95 US urban communities, 1987-2000, *JAMA*, 292: 2372-2378.
[290] M.L. Bell et al. (2006). The Exposure-Response Curve for Ozone and Risk of Mortality and Adequacy of Current Ozone Regulations, *Environ. Health Perspect.*, 114(4): 32-536.
[291] D.M. Spektor, et al. (1998). Effects of ambient ozone on respiratory function in active, normal children, *Am. Rev. of Resp. Disease*, 137(2): 313-320.

EPA must interpret the epidemiological studies to set a standard that is protective of public health with an adequate margin of safety.

Many short-term epidemiological studies that find positive associations with adverse respiratory health outcomes report air quality concentrations as a mean or median metric of varying time periods: 1-hour; 8-hour; or 24-hour. The mean concentrations reported as a summary statistic in many studies and summarized in numerous tables in Chapter 6 of the ISA, do not tell the whole story. That is because adverse effects are occurring along a continuum of ozone concentrations.

CASAC made this point strongly in its November 6, 2012 letter[292]:

> *"The epidemiologic studies should be used to draw inferences regarding the shape and magnitude of the concentration-response functions between ozone exposures and various health outcomes across the full range of the ozone exposure distribution. An important consideration will be the level of confidence in the concentration-response functions for concentrations in the range of interest, especially 60 to 70 ppb and perhaps below 60 ppb. With this approach, the EPA can utilize information from various studies regardless of whether the way in which ozone was assessed directly matches the form or averaging period used in the standard. The purpose is to infer the general causal relationship (i.e., shape of and magnitude of the concentration-response function) between exposure levels and risk of various outcomes. As noted above, even studies performed in locations that did not meet the current standard may provide useful information on the relationship between ozone and health across the entire distribution of ozone."*

CASAC member Dr. Ana Diez Roux[293] elaborated on this point in her individual comments on the draft Policy Assessment:

> *In several places the chapter notes that selected epidemiologic studies that were conducted in cities that would not have met the current standard provide no insight into the appropriateness of the degree of public health protection provided by the current standard (this statement is made several times in reference to both short term and long term exposure studies). This seems an overstatement. The informativeness of these studies depends on the actual distribution and range of ozone concentrations investigated rather than on whether the standard was or was not met. To the extent that these studies allow estimation of the dose-response gradient extending into the ozone exposure distribution that would be expected even if the current standard were met, they do indeed provide important evidence that can be used to determine the health benefit that could be*

[292] Dr. Chris Frey, Chair, Clean Air Scientific Advisory Committee and Dr. Jonathan M. Samet, Immediate Past Chair, Clean Air Scientific Advisory Committee, Letter to the Honorable Lisa P. Jackson, Administrator, U.S. Environmental Protection Agency, re CASAC Review of the EPA's *Policy Assessment for the Review of the National Ambient Air Quality Standards (First External Review Draft—August 2012*). EPA-CASAC-13-003, November 26, 2012.

[293] Frey and Samet letter, November 26, 2012 – Appendix A – Comments of Dr. Ana Diez Roux.

expected if the standard were lowered even further.

CASAC panel member Ed Avol agreed[294]:

> *"I agree that there is data from recent epidemiological investigations on which to consider exposure distributions across the entire O3 concentration range, and it would be informative to do so, and I do think there is interesting and important information to consider at ranges below the existing standard."*

In considering the results reported for epidemiological studies, EPA has to look at the distribution of air quality values. Adverse effects are occurring at concentrations both above and below the mean. The bulk of the effects occur within one standard deviation of the mean. One standard deviation below the mean in the most relevant statistic to consider for standard setting purposes, because it reflects the lower end of the distribution of air quality values where adverse effects are occurring.

Most epidemiological studies report mean concentrations and the distribution of air quality values around the mean. EPA must use this information to set a standard that provides an adequate margin of safety. The highest concentration day in a study is not the appropriate metric upon which to base the level of the standard.

While the current form of the ozone standard is based on the average of the fourth highest daily max over three years, the level should be set based on the full distribution of the air quality data reported in the epidemiological studies.

In addition to the numerous studies discussed above, a number of other epidemiological and field studies have reported effects of acute exposures to ozone at concentrations less than 60 ppb. These studies report positive, statistically significant effects of acute ozone exposure on lung function, respiratory symptoms, and cardiovascular outcomes, daily emergency department visits, daily hospital admissions, and mortality.

These comments cite statistics drawn from the studies themselves of mean and sometimes maximum ozone concentrations. This information is can be very useful to inform the standard-setting process. Depending on the study design, a variety of statistics may be reported, for example 1-hour maximum, 8-hour average, 24-hour average, or various percentile concentrations.

ix. Many Additional Epidemiological Studies Show Need for 60 ppb Standard

Additional epidemiological studies demonstrate that a standard at least as stringent as 60 ppb is needed to protect against a range of serious adverse health impacts. This review considers substantial evidence showing the effects of ozone on hospital admissions and emergency department visits.

[294] Frey and Samet Letter, Nov. 26, 2012 - Appendix A – Comments of Ed Avol.

A very large case-crossover study of Medicare recipients in 36 U.S. cities evaluated the effect of ozone and PM_{10} on respiratory hospital admissions in the elderly over a 13-year period.[295] *Medina-Ramón, et al* found that the risk of daily hospital admissions for chronic obstructive pulmonary disease (COPD) and pneumonia increased with short-term increases in ozone concentrations during the warm season, but not during the cold season. Importantly, 8-hour mean warm season ozone concentrations in this study ranged from 15 ppb in Honolulu to 63 ppb in Los Angeles. As indicated in the table below, concentrations in most cities were in the 40-55 ppb range. This study provides powerful evidence for a standard 60 ppb or below.

TABLE 1. Environmental variables and respiratory hospital admissions in 36 US cities during 1986–1999

City, state	Mean (SD*) ozone level (ppb)		Mean (SD) PM_{10}* level ($\mu g/m^3$)	Mean (SD) apparent temperature (°C)	Total population aged ≥65 years (no.)	COPD* admissions (no.)	Pneumonia admissions (no.)
	Warm season	Cold season					
Albuquerque, New Mexico	50.5 (9.3)	34.5 (10.2)	27.9 (16.5)	12.2 (8.9)	50,379	3,115	9,035
Atlanta, Georgia	55.9 (21.4)		33.0 (16.4)	17.1 (10.2)	155,955	15,503	36,488
Baltimore, Maryland	52.3 (20.2)	26.8 (13.0)	32.4 (17.1)	13.0 (11.1)	197,438	19,950	40,858
Birmingham, Alabama	49.7 (17.0)		36.1 (21.0)	17.4 (10.5)	119,809	13,134	33,011
Boston, Massachusetts	42.3 (17.8)	28.3 (11.3)	25.4 (11.7)	10.0 (10.3)	342,322	34,700	88,936
Boulder, Colorado	51.3 (14.2)		24.2 (15.5)	8.5 (9.7)	17,048	1,678	3,427
Canton, Ohio	52.6 (17.8)		26.1 (12.6)	9.3 (11.2)	53,216	7,534	12,965
Chicago, Illinois	40.0 (16.1)	22.7 (9.8)	33.6 (17.4)	9.5 (11.9)	631,826	49,581	142,576
Cincinnati, Ohio	50.0 (17.8)		32.2 (15.6)	11.9 (11.5)	115,000	10,797	33,323
Cleveland, Ohio	44.6 (17.6)		37.1 (19.1)	9.8 (11.3)	220,659	29,947	50,262
Colorado Springs, Colorado	45.5 (11.3)	30.4 (11.6)	23.3 (13.4)	7.8 (9.0)	31,674	2,497	5,729
Columbus, Ohio	49.8 (18.1)		30.5 (14.6)	11.1 (11.5)	92,485	12,571	21,900
Denver, Colorado	44.0 (14.0)	22.1 (12.7)	33.2 (18.8)	8.5 (9.7)	64,152	4,219	11,820
Detroit, Michigan	41.7 (17.2)		33.7 (19.7)	9.3 (11.5)	263,997	5,751	12,393
Honolulu, Hawaii	15.0 (8.4)		15.9 (6.2)	27.5 (2.9)	91,485	28,404	57,682
Houston, Texas	44.9 (22.1)	32.9 (17.1)	30.3 (16.0)	22.2 (10.1)	196,474	3,798	14,463
Jersey City, New Jersey	50.3 (23.4)		32.2 (17.0)	12.4 (11.1)	70,014	18,863	41,754
Los Angeles, California	63.0 (23.4)	31.4 (20.2)	44.0 (19.3)	16.5 (4.3)	855,666	9,211	12,645
Minneapolis, Minnesota			27.3 (14.6)	7.4 (12.5)	175,854	63,316	174,241
Nashville, Tennessee	44.9 (16.8)	23.9 (13.5)	32.2 (14.9)	15.5 (11.3)	59,235	9,805	26,923
New Haven, Connecticut	45.4 (19.5)		26.0 (16.1)	9.6 (10.8)	117,863	5,962	14,719
New York City, New York	41.0 (19.5)	19.7 (10.0)	28.9 (13.9)	12.5 (10.8)	952,731	8,082	22,954
Palm Beach, Florida	28.6 (12.7)	33.7 (12.0)	20.0 (8.1)	27.1 (6.3)	210,389	70,181	187,043
Philadelphia, Pennsylvania	47.8 (21.0)	23.0 (13.0)	32.1 (15.8)	12.9 (11.1)	241,206	10,626	22,170
Pittsburgh, Pennsylvania	48.4 (19.9)		30.3 (20.0)	10.3 (10.9)	232,505	26,604	47,126
Provo, Utah	54.6 (10.9)		35.1 (26.7)	9.6 (10.4)	18,429	33,408	52,148
Sacramento, California	55.6 (15.7)	32.7 (14.2)	31.1 (19.7)	14.4 (7.0)	109,674	718	4,081
Salt Lake City, Utah	54.0 (12.5)		35.7 (23.9)	9.6 (10.4)	61,079	8,680	21,840
San Diego, California	47.6 (12.1)	40.4 (15.2)	33.3 (13.1)	17.0 (4.4)	272,348	2,090	9,348
San Francisco, California	22.8 (8.1)	19.3 (10.2)	27.7 (16.8)	12.6 (3.8)	105,263	17,632	43,446
Seattle, Washington	35.0 (14.2)		28.8 (18.6)	9.5 (6.3)	167,328	4,711	18,139
Steubenville, Ohio	46.1 (17.3)		34.7 (19.9)	10.3 (10.9)	23,878	9,334	23,732
St. Louis, Missouri	48.4 (17.1)		27.7 (12.7)	13.7 (12.3)	214,492	4,039	9,412
Spokane, Washington	44.6 (10.4)		32.2 (28.3)	6.5 (9.0)	47,877	5,633	8,976
Washington, DC	48.4 (20.2)	20.1 (12.3)	27.7 (13.4)	14.2 (11.2)	77,672	17,665	54,386
Youngstown, Ohio	47.1 (20.3)		31.2 (15.6)	8.9 (11.0)	61,122	8,267	14,862

* SD, standard deviation; PM_{10}, particulate matter with an aerodynamic diameter of ≤10 μm; COPD, chronic obstructive pulmonary disease.

[295] M. Medina-Ramón, A. Zanobetti, & J. Schwartz (2006). The Effect of Ozone and PM_{10} on Hospital Admissions for Pneumonia and Chronic Obstructive Pulmonary Disease: A National Multicity Study, *Am. J. of Epid*, 163: 579-588.

Medina-Ramón, et al.2006

A large study in Atlanta by Tolbert et al.[296] found a positive association between short-term ozone exposure (mean 8-hour ozone concentration 53.0 ppb) and respiratory disease-related emergency visits during non-winter months. This association remained robust in multipollutant models.

Similarly, Wilson et al (2005) found that ozone increases in New England were correlated with emergency room visits for asthma in Portland, Maine, but not in Manchester, New Hampshire, a smaller city with fewer visits to analyze. The maximum 8-hour mean ozone concentration in Portland was 43.1 ppb (13.5 SD).[297]

Yang et al. (2007) reported positive associations between ozone and hospital admissions for COPD in Taipei, Taiwan in single- and two-pollutant models. Mean ozone concentrations were 20.52 ppb, and maximum ozone concentrations were 62.79 ppb in this study.[298]

In a study published last year, researchers in Stockholm, Sweden total used ten years of data from the Swedish cardiac arrest register with a time-stratified case-crossover design to analyze exposure to air pollution and the risk of out-of-hospital cardiac arrest.[299] Exposure to ozone, $PM_{2.5}$, PM_{10}, NO_2, and NO*x* was defined as the mean urban background level during 0–2, 0–24, and 0–72 hours before the event and control time points. After adjusting for temperature and relative humidity, Raza et al found that ozone was associated with an increased risk of out-of-hospital cardiac arrest for all time windows analyzed. The mean 8-hour maximum ozone concentration in urban areas during the warm season was just 34 ppb.

The study suggests that short-term elevations of ozone urban background levels are associated with an increased risk of out-of-hospital cardiac arrest with no indication of a threshold, in a region with 23 ppb 24-hour mean ozone levels.

[296] Tolbert et al. (2007). Multipollutant modeling issues in a study of ambient air quality and emergency department visits in Atlanta, *J. Expo. Sci. Environ. Epidemiol.*, 17 Suppl. 2: S29-35.
[297] A.M. Wilson (2005). Air Pollution, Weather, and Respiratory Emergency Room Visits in Two Northern New England Cities: an Ecological Time-Series Study, *Environ, Res.*, 97: 312 -321.
[298] C.Y. Yang & C.J. Chen (2007). Air pollution and hospital admissions for chronic obstructive pulmonary disease in a subtropical city: Taipei, Taiwan, *J. Toxicol. Environ. Health*, 70: 1214-1219.
[299] A. Raza, et al. (2014). Short-term effects of air pollution on out-of-hospital cardiac arrest in Stockholm, *Eur. Heart J.*, 35 (13), 861-8.

The exposure–response suggests a sustained risk of OHCA on exposure to O_3 concentrations below the WHO guideline of 100 µg/m^3 (*Figure 5*).

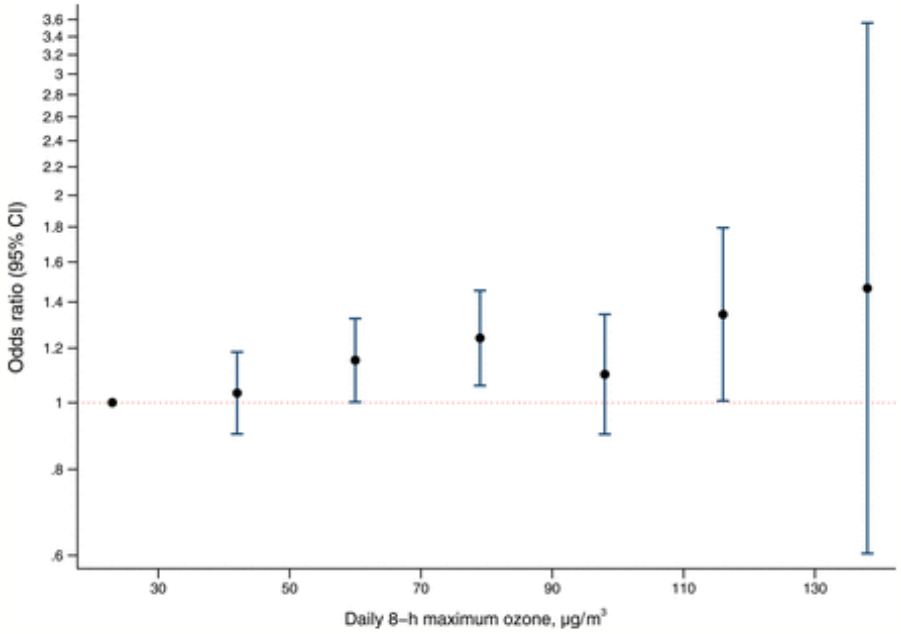


Figure 5

Exposure–response relationship for O_3 and out-of-hospital cardiac arrest. Preceding 8-h max O_3 and OHCA by 20 $\mu g/m^3$ categories with the mid-point of lowest category (23 $\mu g/m^3$) as a reference. The first and last categories were categorized as min to 30 and 130 to max (143), respectively. The odd ratio is plotted on the log-scale.

From Raza, et al. 2014.

Additional studies document evidence of harm at levels well below both the existing standard and the proposed range. Not surprisingly, most provide additional evidence of the risks faced by vulnerable populations at low levels of exposure. A number of these studies are discussed in more detail below.

x. Studies of Infants, Children, and the Elderly

Epidemiological studies of effects of low concentrations of ozone on infants, children, and adults over age 65 indicate not only that the current standards do not protect these sensitive populations and need to be lowered, but document harm to these populations at levels well below the EPA proposal.

Triche et al (2006) examined respiratory effects of ozone in 700 infants living in nonsmoking households in southwestern Virginia.[300] The authors concluded:

[300] E.W. Triche et al. (2006). Low-level ozone exposure and respiratory symptoms in infants, *Environ Health Perspect.*, 114 (6): 911-6.

"At levels of ozone exposure near or below the current U.S. EPA standards, infants are at increased risk of respiratory symptoms, particularly infants whose mothers have physician-diagnosed asthma."

Although the "current" 8-hour standard in 2006 was the equivalent of 84 ppb (0.08 parts per million), the conclusion still holds true today. In this study there were no days when the one-hour standard was exceeded, and only two days when the 8-hour ozone standard was exceeded. As shown in the following table, the mean 8-hour maximum ozone concentration was 54.5 ppb, with a standard deviation $\pm$ 13.0.

Table 2. Distribution of pollutants over study period (n = 166 days), summers of 1995 and 1996.

Pollutant	Mean ± SD	Median	Range	25th–75th percentile	IQR
24-hr average O_3 (ppb)	35.2 ± 8.4	35.7	13.5–56.6	28.8–40.6	11.8
8-hr maximum O_3 (ppb)	54.5 ± 13.0	55.3	23.5–87.6	45.1–64.1	19.0
1-hr peak O_3 (ppb)	60.8 ± 13.4	60.5	26.0–95.0	52.0–70.0	18.0
$PM_{2.5}$ (μg/m^3)	23.2 ± 10.3	22.3	3.5–59.6	15.7–29.4	13.7
Coarse (μg/m^3)	6.2 ± 3.2	5.9	0.0–19.8	4.2–7.8	3.6

Dales et al. studied 15 years of data on newborns 0-28 days of age in 11 large Canadian cities to determine the influence of gaseous air pollutants on neonatal respiratory disease.[301] Daily hospitalizations for respiratory causes were correlated with daily concentrations of ambient air pollutants. Results were adjusted for day of the week, temperature, barometric pressure, and relative humidity. As illustrated in the following table, ozone concentrations were extremely low in this study, ranging from a 24-hour mean level of 13.3 ppb in Vancouver to 23.1 ppb in Saint John, with a population weighted average of 17 ppb. Effects evident at these low concentrations strongly suggest the need for a final standard of 60 ppb or below.

Table 2. Population size, 24-hr mean air pollution levels (5th, 95th percentiles), and weather variables for 11 Canadian cities, 1 January 1986 to 31 December 2000.

City	O_3 (ppb)	NO_2 (ppb)	SO_2 (ppb)	CO (ppb)	Mean temperature (°C)	24-hr change in barometric pressure	% Relative humidity
Calgary	17.8 (4.7, 32.3)	25.6 (13.3, 41.0)	3.6 (1.0, 8.0)	0.9 (0.4, 2.0)	4.5 (–15.5, 18.4)	0.0 (–1.1, 1.13)	61.2 (37, 86)
Edmonton	17.0 (4.0, 33.1)	24.6 (11.5, 43)	2.7 (0, 6.0)	1.1 (0.4, 2.4)	3.0 (–19.5, 18.1)	0.0 (–1.2, 1.2)	68.6 (47, 88)
Halifax	20.8 (9, 35)	15.1 (3, 28)	10.1 (2, 23)	0.8 (0.3, 1.7)	6.4 (–10.4, 10.3)	0.0 (–1.7, 1.6)	77.5 (54, 96)
Hamilton	19.0 (3.3, 41.8)	20.8 (11, 34)	8.2 (1.7, 17.5)	0.9 (0.2, 1.6)	7.9 (–9.4, 22.8)	0.0 (–1.3, 1.3)	73.5 (50, 95)
London	22.3 (6, 46)	20.0 (8, 35)	3.7 (0, 11)	0.4 (0, 1.2)	7.9 (–9.7, 22.9)	0.0 (–1.25, 1.27)	75.7 (55, 93)
Ottawa	16.4 (4.5, 31.0)	21.2 (7, 38)	3.9 (0, 10)	0.9 (0.2, 1.9)	6.3 (–15, 23)	0.0 (–1.5, 1.5)	69.4 (46, 91)
Saint John	23.1 (10.7, 38.5)	9.2 (2, 21)	8.3 (0.5, 23.5)	0.7 (0.1, 1.7)	5.1 (–12.6, 18.6)	0.0 (–1.6, 1.5)	75.4 (52, 95)
Toronto	18.3 (5, 36.7)	25.1 (14, 39)	4.5 (0.2, 11.3)	1.2 (0.6, 1.9)	8.1 (–9.6, 23.4)	0.0 (–1.4, 1.3)	71.9 (52, 90)
Vancouver	13.3 (3.2, 24.9)	19.0 (11.4, 30.2)	4.6 (1.2, 9.8)	0.9 (6.4, 1.9)	10.5 (1.5, 19.1)	0.0 (–1.1, 1.2)	79.3 (64, 94)
Windsor	18.7 (3, 42)	24.9 (11, 41)	7.6 (1.7, 15.7)	0.8 (0, 1.5)	9.8 (–7.3, 25.1)	0.0 (–1.2, 1.3)	70.8 (51, 91)
Winnipeg	18.5 (6, 34)	15.2 (6, 28)	1.2 (0, 3.5)	0.6 (0.3, 1.0)	3.1 (–22.6, 22.1)	0.0 (–1.4, 1.4)	71.9 (49, 91)
Population weighted average	17.0	21.8	4.3	1.0	7.2	48.1	72.3

A study of the impact of ozone on daily respiratory admissions on children less than three years old and another sensitive population, the elderly, in Vancouver, British Columbia revealed associations between ozone and respiratory hospital admissions, which persisted after adjustment

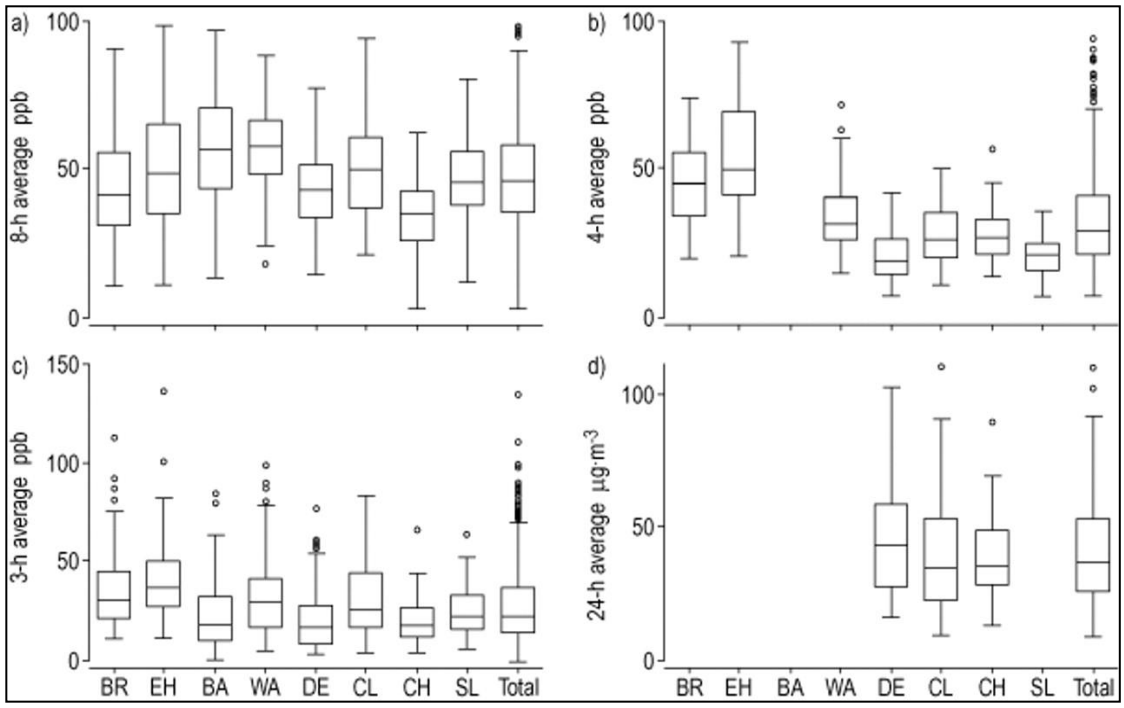
[301] R.E. Dales, S. Cakmak, & M.S. Doiron (2006). Gaseous Air Pollutants and Hospitalization for Respiratory Disease in the Neonatal Period, *Environ. Health Perspect*., 114: 1751-1754.

for copollutants and socioeconomic status.[302] The 24-hour average ozone concentrations in this study were very low at 13.4 ppb.

Important evidence of the increased sensitivity of children with asthma also comes from two studies by Mortimer and colleagues. They examined the effect of daily ambient air pollution in a cohort of 864 asthmatic children in 8 urban areas of the U.S. in a longitudinal study. The cities studied were Baltimore, Chicago, Cleveland, Detroit, Bronx/East Harlem, St. Louis, and Washington DC. Eight-hour average ozone concentrations from 10 a.m. to 6 p.m. were 48 ppb. Median concentrations across cities ranged from 34 to 58 ppb.[303] Researchers found that summertime air pollution at levels below the current air quality standards was significantly related to symptoms and decreased pulmonary function in children with asthma. Ozone was most influential on peak expiratory flow rate. Adverse respiratory effects were observed in all cities. This compelling provides strong support for an 8-hour ozone standard of 60 ppb or below.

A follow-up study of the same cohort found that asthmatic children born prematurely or with low birth weight have the greatest response to ozone.[304] Scientists sought to ascertain which subgroups in a cohort of 846 inner-city asthmatic children aged 4-9 years old were most susceptible to the effects of summertime ozone. Children were recruited from emergency departments and primary care clinics the eight U.S. cities. Mean 8-hour ozone concentrations from 10 a.m. to 6 p.m. across these cities was 48 ppb, as shown in the figure below. The study reported that "children of low birth weight or of premature birth are at greater risk for respiratory problems, and appear to be substantially more susceptible to the effects of summer air pollution than children of normal birth weight or full-term gestation."

[302] Q. Yang et al. (2003). Association between ozone and respiratory admissions among children and the elderly in Vancouver, Canada, *Inhal, Toxicol,* 15(13): 1297-308.
[303] K.M. Mortimer, L.M. Neas, D.W. Dockery, S. Redline, & I.B. Tager (2002). The effect of air pollution on inner-city children with asthma. *Eur. Respir. J.*, 19(4): 699–705.
[304] K.M. Mortimer, I.B. Tager, D.W. Dockery, L.M. Neas, & S. Redline (2000). The effect of ozone on inner-city children with asthma: identification of susceptible subgroups, *Am. J. Respir. Crit. Care Med.*, 162(5): 1838-1845.



Mortimer et al., 2000.

Additional evidence of the increased sensitivity of asthmatic children is provided by the study by Gent et al. Yale University researchers studied a group of 271 asthmatic children under age 12, living in Connecticut and Springfield, Massachusetts in a prospective study of asthma severity.[305] The children's mothers tracked their asthma symptoms such as wheeze, persistent cough, chest tightness, and shortness of breath, and their medication use, on a daily basis. The study found that children with severe asthma were at significantly increased risk due to ozone, even after controlling for co-exposure to fine particles, and at pollution levels well below the current EPA air quality standards for ozone. According to the study, "An ozone level of 63.3 ppb or higher (same-day 8 hour average) was associated with a 30% increase in chest tightness. Previous day levels of 52.1 ppb or above were associated with chest tightness, persistent cough and shortness of breath." This study also provides evidence of the sensitivity of asthmatic children on maintenance medication to ozone, and of the need to lower the standard due to effects at low concentrations. As indicated in the following table, mean 8-hour ozone concentrations in this study were 51.3 ppb, with a standard deviation of 15.5.

[305] J.F. Gent et al. (2003). Association of Low-Level Ozone and Fine Particles with Respiratory Symptoms in Children with Asthma. *JAMA*, 290(1)*:* 1859-1867.

Table 1. Ozone, Particulate Matter of 2.5 µm or Less ($PM_{2.5}$), and Temperature in Southern New England, April 1 to September 30, 2001

			Percentile				
	Mean (SD)	Range	20th	40th	50th	60th	80th
Ozone, ppb							
1-Hour average	58.6 (19.0)	27.1-125.5	43.2	51.6	55.5	58.9	72.7
8-Hour average	51.3 (15.5)	21.4-99.6	39.1	45.9	50.0	52.1	63.3
$PM_{2.5}$, 24-hour total, µg/m³	13.1 (7.9)	3.7-44.2	6.9	9.0	10.3	12.1	19.0
Temperature, 24-hour maximum, °C	23.5 (6.0)	4.89-36.2	17.6	23.7	25.0	26.1	28.4

Gent et al., 2003.

Tolbert et al. examined pediatric emergency room visits for asthma in relation to air quality.[306] As shown in the following table, mean 8-hour ozone concentrations in this study were 59.3 ppb, with a standard deviation of 19.1. Ozone was found to be associated with asthma emergency room visits, with a relative risk of 1.026 per 20 ppb ozone. Associations were robust to analytical method and model specifications. The authors conclude that both ozone and PM_{10} are independently associated with asthma exacerbation, and that the data "suggest continuing health risks at pollution levels that commonly occur in many US cities." This study provides strong evidence of the need to set the 8-hour average standard at 60 ppb.

TABLE 1. Means values, ranges, and Spearman's rank correlation coefficients for air quality variables in a study of pediatric asthma emergency room visits, Atlanta, Georgia, June through August, 1993–1995

	Mean	Range	Spearman's rank correlation coefficient				
			8-hour ozone	24-hour PM_{10}†	1-hour NO_x†	24-hour pollen	24-hour mold
8-hour ozone (ppb)	59.3 (19.1)‡	18.2–113	1.0				
1-hour ozone (ppb)	68.8 (21.1)	22.8–132	0.99*				
24-hour PM_{10} (µg/m³)	38.9 (15.5)	9–105	0.75*	1.0			
1-hour NO_x (ppb)	81.7 (53.8)	5.35–306	0.51*	0.44*	1.0		
24-hour pollen (grains/m³)	3.8 (4.5)	0–29.8	0.29*	0.18*	0.25*	1.0	
24 hour mold (grains/m³)	474 (342)	91–2,710	–0.15*	–0.17*	0.11	0.43*	1.0
Minimum temperature (°F)	71.4 (3.4)	57–78	0.28*	0.43*	0.12*	–0.09	–0.29*
Wind speed (m/s)	8.28 (2.37)	4.1–19.3	–0.45*	–0.39*	–0.48*	–0.05	0.07

* $p < 0.05$.
† PM_{10}, particulate matter ≤ 10 µm in aerodynamic diameter; NO_x, total oxides of nitrogen.
‡ Numbers in parentheses, standard deviation.

Tolbert et al., 2000.

Babin et al in a 2007 study reported associations between pediatric emergency department visits and outdoor ozone concentrations are strongest for school-age children 5-12 years old. In this group, a 1 ppb increase in ozone concentration indicated a mean 3.2 percent increase in daily emergency department visits, and a mean 8.3 percent increase in daily

[306] P.E. Tolbert (2000). Air Quality and Pediatric Emergency Room Visits for Asthma in Atlanta, Georgia, *Am. J. Epidemiol.*, 151: 798-810.

emergency admissions for asthma exacerbations. The 8-hour daily maximum ozone concentrations reached Code Red levels on only five days during the study period.[307]

xi. Two Important New Studies Published Since the Completion of the ISA Provide Additional Support for a 60 ppb Standard

Two studies published after the completion of the ISA provide strong new evidence of adverse effects at concentrations of 60 ppb or below.

A study in Sublette County, Wyoming, an area of concentrated oil and gas drilling, explored the association between daily ozone concentrations and visits to primary care clinics for respiratory problems.[308] Sublette County had been reporting high ozone levels in the winter.

Pride and colleagues reported that for every 10-ppb increase in the 8-hour maximum average ground-level ozone, there was a three percent increase in clinic visits the following day. The effect was strongest during the winter months. Furthermore, the mean 8-hour maximum ozone concentrations ranged from 41 ppb to 53 ppb among the monitoring stations included. The results were consistent when days with ozone concentrations greater than 75 ppb were excluded. This provides powerful evidence that clinic visits for respiratory causes increase even on days when the ozone is lower than the current standard of 75 ppb.

[307] Babin et al. (2007). Pediatric patient asthma-related emergency department visits and admissions in Washington, DC from 2001-2004, and associations with air quality, socio-economic status and age group. *Environ. Health*, 21: 6-9.

[308] K.R. Pride (2014). Association of short-term exposure to ground-level ozone and respiratory outpatient clinic visits in a rural location - Sublette County, Wyoming, 2008-2011. *Environ Res*., 137C: 1-7.

Table 1: Descriptive Analyses of All 13 Ground-Level Ozone Monitoring Stations, Sublette County, Wyoming, January 1, 2008–December 31, 2011

Monitor	Observation Days	Mean ppb	SD	Median ppb	Minimum ppb	Maximum ppb
Boulder	1429	49	10	49	22	123
Daniel	1363	47	8	47	19	84
Big Piney	190	51	6	52	38	72
Wyoming Range	273	50	7	49	34	83
Jonah	89	49	15	45	15	102
Pinedale CastNET	122	53	6	53	42	70
Juel Springs	726	49	8	49	28	94
Pinedale	879	46	8	46	14	89
FARS	424	46	9	46	25	65
SADR	422	47	8	48	18	70
MARB	440	44	8	45	16	75
Lab1	427	41	8	41	20	65
BARG	440	49	7	49	30	75

Table from Pride et al., 2014.

The Framingham Heart Study is a large cohort study of adult men and women in the Boston, Massachusetts area.[309] For this study, Rice and colleagues used the Air Quality Index values to examine the effect of low levels air pollution, within the current NAAQS, on lung function within this well-studied cohort. The study measured lung function with previous days' exposure to ozone (among other pollutants).

Exposure to pervious days' ozone concentrations in the "moderate" range was associated with lung function declines of 55.7 ml lower FEV_1, compared to days when the air quality was in the "good" range. According to the authors, these findings suggest that "the general population, not just 'unusually sensitive' people, may experience respiratory effects from ambient pollution at levels that are considered to be safe." This important finding underlines the broad public health risks of ozone at the current standard level.

The table shows that mean 8-hour ozone concentrations in this study were 28.7 ppb, with a range of 2 to 59.6 ppb. In other words, lung function declines occurred despite ozone concentrations having never exceeded 60 ppb over the course of the study.

[309] M.B. Rice, et al. (2013). Short-term exposure to air pollution and lung function in the Framingham Heart Study, *Am. J. Respir. Crit. Care Med.*, 188(11), 1351-7.

TABLE 2. POLLUTANTS AND METEOROLOGY DISTRIBUTIONS

						Spearman Correlation Coefficients			
	N Obs	Mean	SD	Range	IQR	$PM_{2.5}$	NO_2	O_3	Temp
$PM_{2.5}$, $\mu g/m^3$	5,272	10.4	5.5	1.1 to 34.6	6.4 to 12.8	1			
NO_2, ppb	5,358	21.3	7.2	6.0 to 57.4	15.9 to 26.0	0.63	1		
O_3*, ppb	2,475	28.7	9.9	2.0 to 59.6	21.2 to 35.3	0.33	0.0051	1	
Temp, °C	5,344	10.3	9.1	−18.3 to 30.8	3.6 to 17.7	0.17	−0.17	0.18	1
RH, %	5,344	67.6	16.4	20.7 to 100	55.4 to 81.0	0.20	−0.0048	−0.32	0.16

Definition of abbreviations: IQR = interquartile range; NO_2 = nitrogen dioxide; O_3 = ozone; obs = observations; $PM_{2.5}$ = particulate matter < 2.5 μm in diameter; RH = relative humidity; temp = temperature.

*Ozone data were analyzed during the Environmental Protection Agency ozone monitoring season for Massachusetts (April–September).

Table from Rice et al (2014) showing air pollutants and relevant meteorological conditions.

Under the current Air Quality Index, air quality is rated in the "good range," when 8-hour average concentrations are less than 59 ppb. The "moderate range" is from 60 to 75 ppb, below the current NAAQS.

xii. EPA Analysis Shows that Numerous Studies Have 98th Percentile 8-hour Daily Maximum Concentrations Below 70, 65, and 60 ppb

In the last review, our organizations filed comments[310] identifying twenty North American studies which reported positive, statistically significant results for various health endpoints, for which EPA derived 98th percentile 8-hour daily maximum concentrations of about 85 ppb or lower.[311] The studies in Table 5 below, are drawn from Appendix 3B of the 2007 Staff Paper.[312] Even where the areas studied would have met the 98th percentile form of alternative standard levels below 70 ppb, adverse health effects were observed at those lower levels.

EPA argues that the 98th percentile statistic may be relevant to standard-setting because it approximates the 4th highest daily maximum concentrations averaged over 3 years. As such, the studies indexed in the Table 5 provide additional evidence for a standard of 60 ppb.

It is noteworthy that five studies report positive, statistically significant relationships between 8-hour ozone concentrations and various adverse effects at concentrations below 60 ppb, seven additional studies (for a total of 12) report effects below 70 ppb.

[310] Comments of American Lung Association, Environmental Defense, and Sierra Club on U.S. EPA's Proposed Revisions to the National Ambient Standards for Ozone, 72 Fed. Reg. 37,818 (Oct. 9, 2007).
[311] L. McCluney, M. Rizzo, & R. Ross (2006). Development of descriptive statistics for 8-hr daily maximum ozone data from epidemiologic studies. U.S. EPA Memorandum to Ozone NAAQS Review Docket (OAR-2005-0172).
[312] U.S. EPA (2007). Review of National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper (EPA-452/R-07-07).

Study Endpoints	98th percentile 8-hr daily max (ppb)
Respiratory Symptoms	
Mortimer et al., 2002	64.3
Delfino et al., 2003	34.8
Ross et al., 2002	68.8
Lung Function Changes	
Mortimer et al., 2002	64.3
Naeher et al., 1999	74
Brauer et al., 1996	55
Emergency Department Visits: Respiratory Diseases	
Delfino et al., 1997	57.5
Wilson et al., 2005 (Portland)	85
Friedman et al., 2001	85.8
Emergency Department Visits: Cardiovascular Outcomes	
Rich et al., 2005	74
Hospital Admissions: Cardiovascular Diseases	
Koken et al., 2003	64.5
Hospital Admissions: Respiratory Diseases	
Delfino et al., 1994	69
Burnett et al., 1994	79
Burnett et al., 1997	62
Yang et al., 2003	42.7
Moolgavkar et al., 1997	83.2
Burnett et al., 2001	77.7
Burnett et al., 1999	68.4
Schwartz et al., 1994	82.8

Ozone Epidemiological Studies Showing Effects at Low Concentrations: EPA Derived 98th Percentile Statistics Near or Below the Current Standard
Source: American Lung Association, 2007, Derived from Staff Paper Appendix 3B. Ozone Epidemiological Study Results: Summary of effect estimates and air quality data reported in studies, distribution statistics for 8-hr daily maximum ozone concentrations for the study period and location, and information about monitoring data used in the study.

In short, taking into consideration all the available evidence, from the short-term epidemiological studies, commenters find overwhelming evidence of the need for a 8-hour average standard of at least 60 ppb.

xiii. Findings from Long-Term Epidemiological Studies Compel a Standard of 60 ppb

As discussed above, controlled human exposure studies, while critical to understanding the adverse health effects of ozone exposure, nonetheless have a number of limitations that can work to understate the effects that real world ozone exposures can have on human health.

Epidemiological studies included in this review cycle support the ISA's new, stronger conclusion that there is a likely causal relationship between long-term exposures to ozone and respiratory effects. The ISA finds that "…long term ozone exposure is associated with adverse effects ranging from episodic respiratory illness to permanent respiratory injury to progressive respiratory decline."[313] The ISA document details numerous studies showing that long-term exposures to ozone are associated with new onset asthma, increased asthma symptoms, increased risk of asthma hospital admissions, deficits in lung function growth rate in children, and increased risk of premature death.

Many of the studies reported average ozone concentrations in the range of 60 to 70 ppb or below. Different exposure metrics are used in different studies. Some authors report the maximum 1-hour average within a 24-hour period, others report the maximum 8-hour average within a 24-hour period, and others report the 24-hour average.

xiv. Increased Premature Mortality from Long-Term Exposures

Jerrett et al. used data from the American Cancer Society Cancer Prevention Study II on 449,000 participants to examine the effect of long-term exposures to air pollution on mortality from cardiopulmonary causes in 96 metropolitan areas.[314] Investigators reported significant increase in the risk of death from respiratory causes, but not from cardiovascular causes, in association with an increase in ozone concentration, even after accounting for $PM_{2.5}$ and other factors. The average ozone concentration for each metropolitan area during the interval from 1977 to 2000 ranged from 33.3 ppb to 104.0 ppb during the summer months.

[313] ISA sec. 7.2.8
[314] M. Jerrett et al. (2009). Long-term ozone exposure and mortality, *New Eng. J. of Med.*, 360(11): 1085-1095.

Average ozone concentrations in three-quarters of the metropolitan areas were less than 62.5 ppb, and less than 57.4 ppb in half the MSAs. The map below indicates where the high and low pollution areas were located.

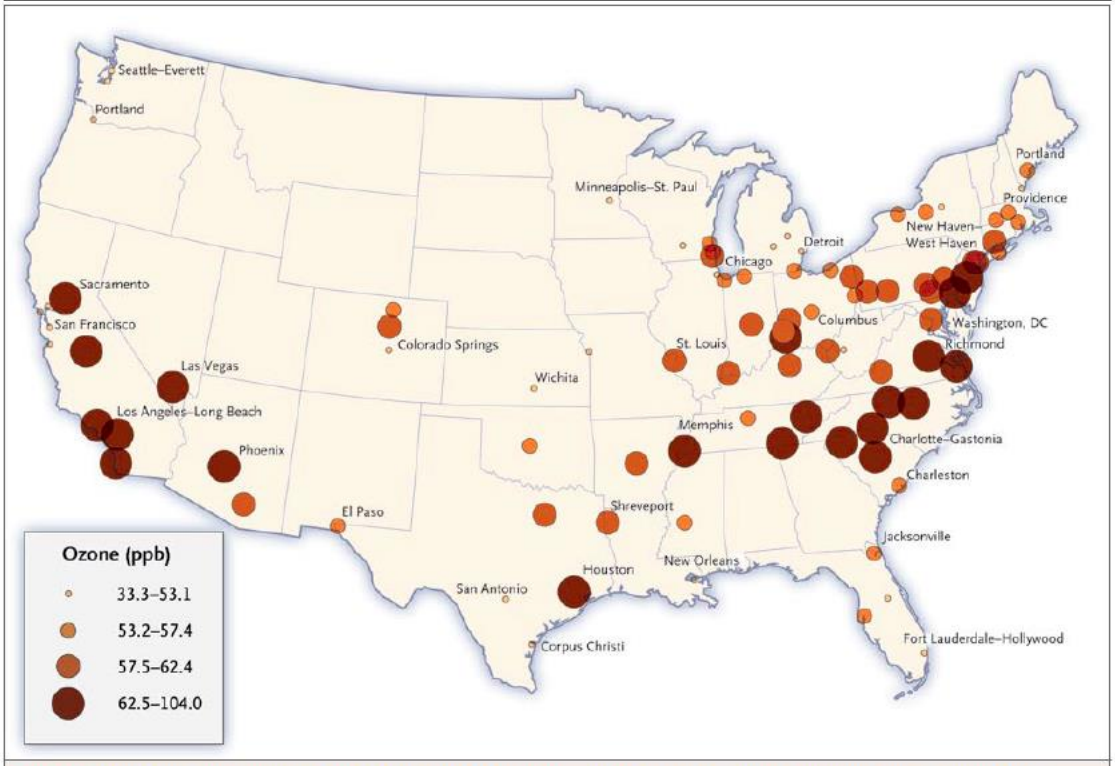


Figure 1. Ozone Concentrations in the 96 Metropolitan Statistical Areas in Which Members of the American Cancer Society Cohort Resided in 1982.

The average exposures were estimated from 1 to 57 monitoring sites within each metropolitan area from April 1 to September 30 for the years 1977 through 2000.

This table shows the number of MSAs in each of the ozone exposure categories.

Table 1. Baseline Characteristics of the Study Population in the Entire Cohort and According to Exposure to Ozone.*

Variable	Entire Cohort (N=448,850)	Concentration of Ozone			
		33.3–53.1 ppb (N=126,206)	53.2–57.4 ppb (N=95,740)	57.5–62.4 ppb (N=106,545)	62.5–104.0 ppb (N=120,359)
No. of MSAs	96	24	24	24	24
No. of MSAs with data on $PM_{2.5}$	86	21	20	23	22
Concentration of $PM_{2.5}$ ($\mu g/m^3$)		11.9±2.5	13.1±2.9	14.7±2.1	15.4±3.2
Individual risk factors					

The risk of dying from respiratory causes was more than three times as great in the metropolitan areas with the highest ozone concentrations compared to those with the lowest ozone concentrations.

A principal finding of the Jerrett study was that ozone and PM each contributed independently to the increased risk in premature death. The effect of ozone on mortality was insensitive to adjustment for individual, neighborhood, and metropolitan-area confounders or to differences in multilevel-model specifications.

The figure below, from the Jerrett study, shows that risk of premature death from ozone exposure rose sharply when daily maximum one-hour ozone concentrations were approximately 55 ppb. Since the Jerrett study examined the daily maximum one-hour concentrations, this would indicate that even a lower 8-hour average would provide increased risk of premature death.

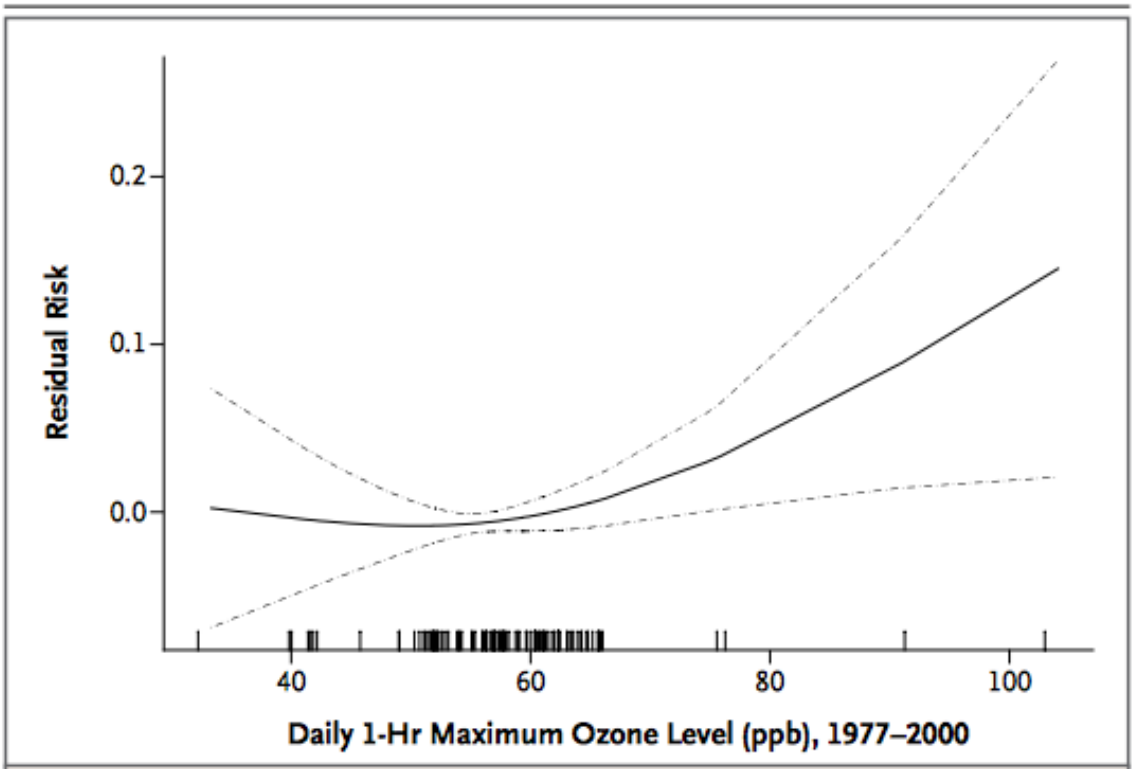


Figure 2. Exposure–Response Curve for the Relation between Exposure to Ozone and the Risk of Death from Respiratory Causes.

The curve is based on a natural spline with 2 df estimated from the residual relative risk of death within a metropolitan statistical area (MSA) according to a random-effects survival model. The dashed lines indicate the 95% confidence interval of fit, and the hash marks indicate the ozone levels of each of the 96 MSAs.

While expressing some reservations, CASAC concurred that Jerrett et al. (2009) "is an appropriate study to use at this time as the basis for the long-term mortality risk estimates given its adequacy and the lack of alternative data."

xv. Long-Term Exposure and Survival Rate in People with Pre-existing Conditions

A recent study by Zanobetti and Schwartz investigated whether long-term exposure to ozone was associated with survival in four cohorts of Medicare enrollees with specific diseases in 105 United States cities.[315] Investigators reported an increased risk of death for persons with a history of congestive heart failure, myocardial infarction, chronic obstructive pulmonary disease, and diabetes associated with each 5 ppb increase in summer average ozone concentrations. These findings are consistent with Jerrett et al that longer-term exposure to ozone is associated with reduced survival.

xvi. Long-Term Exposures and New Onset Asthma

Prospective cohort studies have reported an association between ozone exposures and asthma induction. These studies suggest that ozone may not only exacerbate asthma, but may also trigger the development of the disease.

The ASHMOG prospective cohort study of over 3,000 adults in the nonsmoking Seventh Day Adventist community sought to examine the whether long-term exposure to ozone air pollution can contribute to the prevalence of asthma. The study found that 8-hour average ambient ozone concentration averaged over a 20-year period was associated with doctor diagnoses of adult-onset asthma in nonsmoking males.[316]

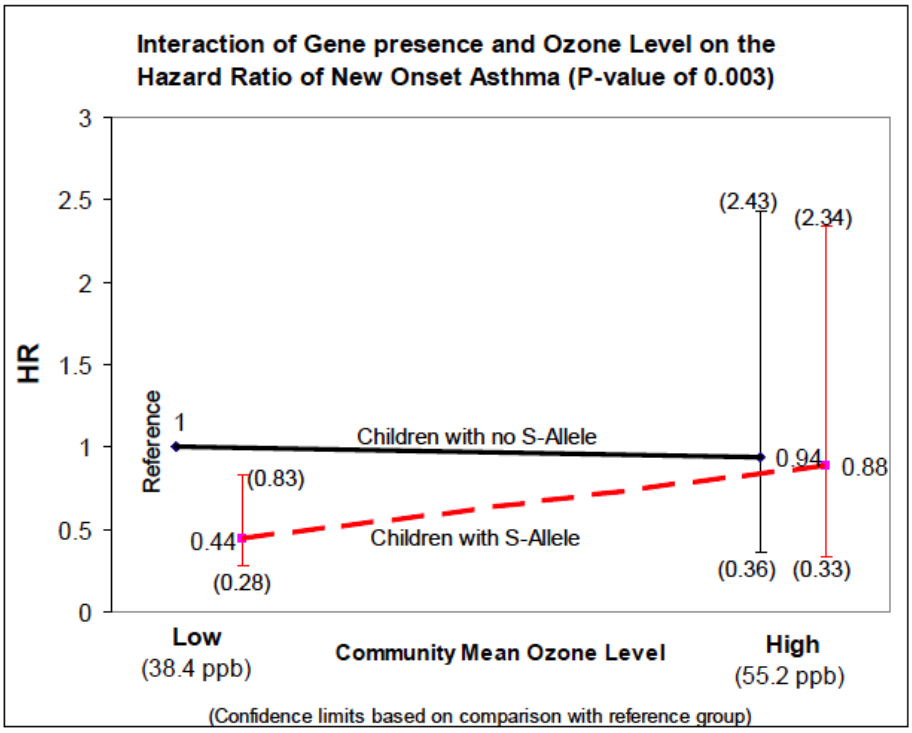
The Children's Health Study is a long-term cohort study designed to examine whether long-term exposure to air pollution was related to chronic respiratory effects in children in 12 southern California communities. McConnell et al 2010[317] reported an increased risk for new onset asthma in children living in communities with the highest ozone concentrations (59.8 ppb annual average of 8-hour daily average ozone) compared to the community with the lowest ozone concentration of 29.5 ppb. A follow-up study by Islam et al 2008[318] examined evidence of a genetic predisposition to develop asthma, and observed a dose-response relationship from the low pollution community (38.4 ppb) to the high pollution community (55.2 ppb).

Another analysis from the California Children's Health Study points strongly to ozone as a cause in the development of asthma in young people who did not previously have the disease. The study compared new asthma cases in 3,535 children who were followed over five years in 12 Southern California communities to determine the potential health damage caused by growing

[315] A. Zanobetti & J. Schwartz (2011). Ozone and survival in four cohorts with potentially predisposing diseases, *Am. J. Respir. Crit. Care Med.*, 184(7): 836-41.
[316] W.F. McDonnell, D.E. Abbey, N. Nishino, & M.D. Lebowitz (1999). Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the AHSMOG study, *Environ. Res*., 80(2), 110-121.
[317] R. McConnell et al. (2010). Childhood incident asthma and traffic-related air pollution at home and school. *Environ. Health Perspect.*, 118(7): 1021-1026 .
[318] . Islam, et al. (2008). Ozone, oxidant defense genes, and risk of asthma during adolescence, *Am. J. Respir. Crit. Care Med.*, 177 (4): 388-95.

up in polluted air. Six of the communities had higher than average ozone concentrations while six had lower than average concentrations.

As noted by Pinkerton et al., this study found that "the incidence of new diagnoses of asthma in children who exercise heavily is associated with average ozone levels of 55.8 to 69.0 ppb during the daytime (10 a.m. to 6 p.m.), levels below the current NAAQS."[319] The study found that children in the high ozone communities who played three or more sports developed asthma at a rate three times higher than those in the low ozone communities. Because participation in some sports can result in a child drawing up to 17 times the "normal" amount of air into the lungs, young athletes are more likely to develop asthma.[320]



Note: An interaction p-value of 0.003 was obtained from the hierarchical two stage Cox proportional hazard model fitting the community specific O_3 and controlling for random effect of the communities. The interaction indicates there is a greater protective effect of having a heme-oxygenase S-allele compared to having the L-allele among children living in communities with lower long-term ambient O_3 concentrations. The HRs are off-set as opposed to overlapping in the figure to allow clearer presentation of the results.

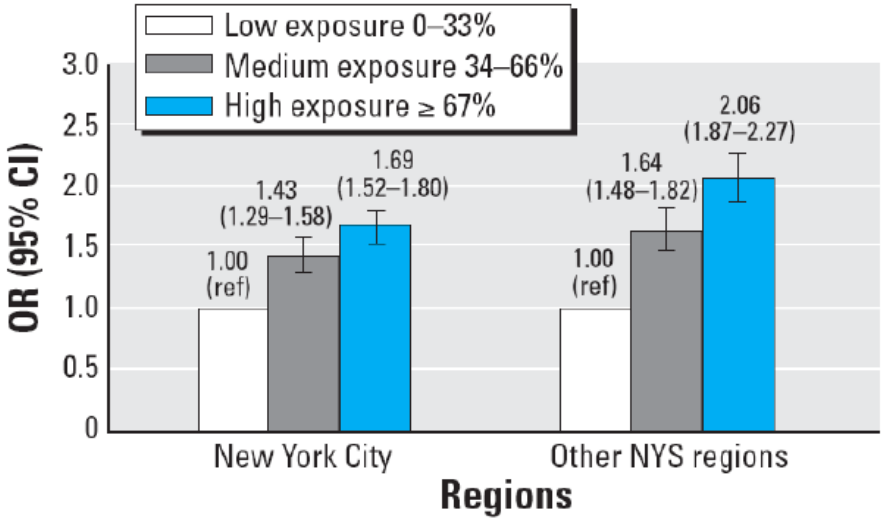
Source: Developed by EPA with data from Islam et al. (2008) (data used with permission of American Thoracic Society).

xvii. Asthma Hospital Admissions in Children

Lin et al 2008[321] examined the association between long-term ozone concentrations and first asthma hospital admission for New York children in a retrospective cohort study. The study

[319] .E. Pinkerton, J.R. Balmes, M.V. Fanucchi, & W.N. Rom (2007). Ozone, a malady for all ages. *Am. J. Respir. Crit. Care Med.,* 176: 107-8.
[320] R. McConnell et al. (2002). Asthma in exercising children exposed to ozone: A cohort study, *Lancet*, 359(9304): 386-391.
[321] S. Lin, X. Liu, L.H. Le, & S. Hwang (2008). Chronic exposure to ambient ozone and asthma hospital admissions among children, *Env. Health Perspect.*, 116(12): 1725-1730.

reported three chronic exposure indicators: annual average of 8-hour maximum ozone concentrations (41 ppb), ozone season average (50.6 ppb) and proportion of follow-up days with ozone levels greater than 70 ppb. Hospital admissions for asthma were significantly associated with increased ozone levels for each of the chronic exposure indicators in a dose-response relationship.



Note: Adjusted for child's sex, age, birth weight, and gestational age; maternal race, ethnicity, age, education, insurance, and smoking status during pregnancy; and regional poverty level and temperature. ORs by low, medium, and high exposure are shown for New York City (NYC: low [37.3 ppb], medium [37.3-38.11 ppb], high [38.11+ ppb] and other New York State regions (Other NYS regions: low [42.58 ppb], medium [42.58-45.06 ppb], high [45.06+ ppb]) for first asthma hospital admission.

Source: Lin (2010); Lin et al. (2008b)

Figure 7-3 Ozone-asthma concentration-response relationship using the mean concentration during the entire follow-up period for first asthma hospital admission.

xviii. Asthma Emergency Department Visits and Hospital Admissions

Meng et al[322] used California Health Interview Survey data to study the association between air pollution and asthma morbidity in the San Joaquin Valley of California, an area with high asthma rates. Authors reported increased asthma-related emergency department visits or hospitalization in association with ozone in all age groups. Median annual ozone concentrations (based on hourly data) were 30.3 ppb, with a 25-75 percentile range of 27.1 to 34 ppb. Potential confounding by PM was ruled out in this study.

Table 7-2 in the ISA details the annual mean concentration as well as the range of ozone concentrations for selected key new studies regarding ozone and respiratory health effects. Many of the studies in this table report low mean or median annual concentrations, and low

[322] Y,Y, Meng et al. (2010). Outdoor air pollution and uncontrolled asthma in the San Joaquin Valley, California, *J. Epidemiol. Comm. Health*, 64(2): 142-7.

maximum or 75th percentile concentrations. These data must be considered in evaluating the suitability of averaging time and level of the ozone standard.

Table 7-2 Summary of selected key new studies examining annual O_3 exposure and respiratory health effects.

Study; Health Effect; Location	Annual Mean O_3 Concentration (ppb)	O_3 Range (ppb) Percentiles
Longitudinal		
Islam et al. (2008); New-onset asthma; CHS	55.2 high vs. 38.4 low communities 10:00 a.m. to 6:00 p.m. average	See left
Islam et al. (2009); New-onset asthma; CHS	55.2 high vs. 38.4 low communities 10:00 a.m. to 6:00 p.m.	See left
Lin et al. (2008b); First asthma hospital admission; New York State - 10 regions	Range of mean O_3 concentrations over the 10 New York Regions 37.51 to 47.78 8-h max 10:00 a.m. to 6:00 p.m.	See left
Salam et al. (2009); Childhood onset asthma; CHS	O_3 greater than or less than 50 ppb	See left
Cross-sectional		
Akinbami et al. (2010); Current asthma U.S.	12 month median 39.8 8hr max	IQR 35.9 to 43.7
Hwang et al. (2005); Prevalence of asthma; Taiwan	Mean 23.14	Range 18.65 to 31.17
Jacquemin et al. (2012); Asthma control in adults; Five French cities	Median 46.9 ppb; 8-h average	25th-75th 41-52
Lee et al. (2009b); Bronchitic symptoms in asthmatic children; CHS	Above and below 50 ppb	See left
Meng et al. (2010); Asthma ED visits or hospitalizations; San Joaquin Valley, CA	Median 30.3 ppb Yearly based on hourly	25-75% range 27.1 to 34.0
Moore et al. (2008); Asthma hospital admissions; South Coast Basin	Median 87.8 ppb Quarterly 1hr daily max	Range 28.6 to 199.9
Rage et al. (2009a); Asthma severity; Five French cities	Mean 30 ppb 8-h average	25th-75th 21-36
Wenten et al. (2009); Respiratory school absence, U.S.	Median 46.9 ppb; 10a.m. – 6 p.m. average	Min-Max 27.6-65.3

xix. Long-Term Exposures and Asthma Prevalence

A study in Los Angeles and San Diego counties investigated associations between traffic and outdoor air pollution levels near residences, and poorly controlled asthma in adults. This

study reported that annual average ozone exposures were associated with poorly controlled asthma among elderly adults.[323]

xx. Low Levels of Ozone over the Long-Term Stunt Lung Function Growth in Children

A number of studies have provided evidence that long-term exposure and relatively low concentrations may have detrimental effects on full development of lung capacity in growing children.

Frischer et al. followed a group of 1,150 first and second grade children in two counties in Austria from 1994-1996, to investigate the long-term effects of ambient ozone.[324] The highest and lowest exposure to ozone differed by a factor of two. Researchers found small but consistent decrements in lung function associated with ambient ozone. They conclude: "This is the first study that suggests chronic effects of ozone on lung function growth in children. Thus, ozone would constitute a risk factor for premature respiratory morbidity during later life." This effect of ozone was confirmed in a follow-up study.[325]

Galizia et al. examined data from health status questionnaires and lung function measurements in relation to residence histories to examine the effect of long-term ozone exposures on over 500 non-smoking Yale college students. Investigators found that "living for four or more years in regions of the country with high levels of ozone and related copollutants is associated with diminished lung function and more frequent reports of respiratory symptoms."[326]

Künzli et al. developed a protocol to relate lifetime cumulative ozone exposure to small airway pulmonary function. This study included 130 nonsmoking, non-asthmatic freshmen from the University of California at Berkeley who were lifelong residents of the Los Angeles Basin or the San Francisco Bay Area, who had volunteered to participate in lung function testing. Researchers observed declines in mid- and end-expiratory flow measures of the small airways that are considered early indicators for pathologic changes that might ultimately progress to chronic obstructive lung disease. These declines were associated with estimated long-term ozone exposures.[327]

A follow-up study assessed effects of chronic exposure to air pollutants in University of California, Berkeley freshmen who were lifelong residents of the Los Angeles or San Francisco Bay areas. Students in the study had never smoked. Air pollution exposure was estimated based

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[323] Y,Y. Meng, M. Wilhelm, R.P. Rull, P. English, & B. Ritz (2007). Traffic and outdoor air pollution levels near residences and poorly controlled asthma in adults, *Ann. Allergy Asthma Immunol.*, 98, 455-463.
[324] T. Frischer et al. (1999). Lung Function Growth and Ambient Ozone: A Three-Year Population Study in School Children, *Am. J. Respir. Crit. Care Med.*, 160: 390-396.
[325] F. Horak et al. (2002). Particulate Matter and Lung Function Growth in Children: A 3-yr Follow-up Study in Austrian Schoolchildren, *Eur. Respir. J.*, 19, 838-845.
[326] A. Galizia & P.L. Kinney (1999). Long-Term Residence in Areas of High Ozone: Associations with Respiratory Health in a Nationwide Sample of Nonsmoking Young Adults, *Environ. Health Perspect*., 107(8): 675-679.
[327] N. Künzli, et al. (1997). Association between Lifetime Ambient Ozone Exposure and Pulmonary Function in College Freshmen: Results of a Pilot Study, *Environ Res*., 72(1): 8-23.

on spatial interpolation of PM_{10}, nitrogen dioxide, and ozone monitors to the students residences. Lung function measurements were gathered between February and May, when the students had not had recent exposure to increased levels of ozone. The study found that lifetime exposure to ozone in adolescents 18-20 years old is associated with reduced levels of lung function measures that reflect the function of the small airways. The associations are independent of any effects related to PM and nitrogen dioxide.[328]

The California Children's Health Study annually measured the lung function of 1,700 fourth-graders enrolled in 1996, monitored the communities' air pollution for four years until 2000, and analyzed the relationships between their lung function growth and the levels of six pollutants. Exposure to ozone was correlated with reduced growth in peak flow rate. Larger deficits in lung function growth rate were observed in children who reported spending more time outdoors. Slower lung growth over a period of several years is evidence of a chronic effect of air pollution on children's respiratory health. Children whose lungs have grown more slowly may have lower maximum lung function as adults, making them more susceptible to respiratory diseases and chronic problems as they age.[329]

A study of over 3,000 8-year old children followed for 3 years in Mexico City underlines the concern about the effects of long-term exposures. After adjusting for acute exposure and other potential confounders, deficits in (forced vital capacity) FVC and FEV_1 growth over the three year follow-up period were significantly associated with exposure to ozone and other pollutants in girls and boys. Over the course of the study period, 8-hour average ozone concentrations ranged from 60 ppb to 90 ppb. In multipollutant models, an interquartile range increase in mean ozone concentration of 11.3 ppb was associated with an annual deficit in FEV_1 of 12 ml in girls and 4 ml in boys. Early lung function deficits may increase the risk of developing chronic obstructive pulmonary disease later in life, as well increasing the risk of cardiovascular morbidity and overall mortality.[330]

Researchers compared chest x-rays from children living in heavily polluted southwest Mexico City with children living in a cleaner air region in Tlaxcala, Mexico. Ozone concentrations exceeded the U.S. NAAQS for an average of 4.7 hours per day, and $PM_{2.5}$ concentrations were above the annual standard. The x-rays of the Mexico City children showed an increased prevalence of bilateral hyperinflation and increased linear markings. CT scans of 25 Mexico City children with abnormal chest x-rays showed evidence of mild bronchial wall thickening, prominent central airways, air trapping, and pulmonary nodules in some of the children, findings suggestive of inflammatory processes. Testing showed 7.8 percent of the Mexico City children had abnormal lung function.[331]

xxi. Chronic Ozone Exposure and Lung Damage

[328] I.B. Tager, et al. (2005). Chronic Exposure to Ambient Ozone and Lung Function in Young Adults, *Epidemiology*, 16(6): 751-759.
[329] W.J. Gauderman (2000). Association between Air Pollution and Lung Function Growth in Southern California Children: Results from a Second Cohort, *Am. J. Respir. Crit. Care Med.*, 162: 1383–1390.
[330] R. Rojas-Martinex et al. (2007). Lung Function Growth in Children with Long-Term Exposure to Air Pollutants in Mexico City, *Am. J. Respir. Crit. Car Med.*, 176: 377-384.
[331] L. Calderón-Garcidueñas et al. (2006). Lung Radiology and Pulmonary Function of Children Chronically Exposed to Air Pollution, *Environ. Health Perspect.*, 114: 1432-1437.

Researchers found that the air pollution exposure produces significant chest X-ray abnormalities in the exposed children, depressed lung function, and an imbalance of blood proteins important to immune response. Twenty-two percent of the exposed children had grossly abnormal nasal mucosa, which can impair nasal defense mechanisms against inhaled gases and particles. The lung damage observed is similar to the chronic inflammatory damage observed in an earlier study of dogs in Mexico City. Researchers report that the x-ray and lung function changes they found in the exposed children could be due to pollution-associated chronic bronchiolitis, which could put the children at greater risk of developing chronic obstructive airway disease later in life. They conclude that lifelong exposure to urban air pollution causes respiratory damage in children and may predispose them to development of chronic lung disease and other problems due to suppression of the immune system.[332]

xxii. Pathological Changes to Nasal Passages

Another study by some of the same researchers reported that biopsies taken from these children exhibit a wide range of pathologic changes to the cells of the nasal passages.

> "The severe structural alteration of the nasal epithelium together with the prominent acquired ciliary defects are likely the result of chronic airway injury in which ozone, particulate matter, and aldehydes are thought to play a crucial role," concluded the researchers. "The nasal epithelium in SWMMC [Southwest Metropolitan Mexico City] children is fundamentally disordered, and their mucocilliary defense mechanisms are no longer intact. A compromised nasal epithelium has less ability to protect the lower respiratory tract and may potentially leave the distal acinar airways more vulnerable to reactive gases."[333]

xxiii. Remodeling of the Airways in Toxicology Studies of Primates

Animal toxicology studies have demonstrated that chronic exposure to ozone air pollution may result in inflammation and injury leading to interstitial remodeling that "may play an important role in the progression and development of chronic lung disease."[334]

xxiv. A Standard of 60 ppb or Below is Needed to Protect Against Long-Term Exposures

Despite the wealth of scientific information on long-term ozone exposures and their adverse impacts reviewed in the ISA, EPA fails to rationally explain how its proposed primary standard will assure prevention of such adverse impacts

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[332] L. Calderón-Garcidueñas et al. (2003). Respiratory Damage in Children Exposed to Urban Pollution, *Pediatric Pulmonology*,; 36: 148-161.
[333] L. Calderón-Garciduenas et al. (2001). Ultrastructural Nasal Pathology in Children Chronically and Sequentially Exposed to Air Pollutants, *Am. J. Resp. Cell Molec. Bio.*, 24: 132-138.
[334] ISA sec. 7.2.4.

Specifically, EPA must consider whether the current form and level of the standard assures the absence of long-term ozone-induced adverse effects. The Policy Assessment looks at the Jerrett et al. study and the HREA using a function from the Jerrett study to estimate premature respiratory deaths attributable to long-term exposures to ozone air pollution. [335]

As EPA explains:

> With regard to long-term O3 concentrations, compared to the current standard or a revised O3 standard with a level of 65 or 70 ppb, a revised standard with a level of 60 ppb would be expected to further reduce the risk of respiratory mortality associated with long-term concentrations, based on information from the study by Jerrett et al. (2009).[153] In addition, a standard with a level of 60 ppb would be expected to more effectively maintain long-term O3 concentrations below those where the study by Jerrett et al. (2009) indicates the most confidence in the reported association with respiratory mortality.
>
> Specifically, air quality analyses indicate this to be the case in all of the urban study areas evaluated at a level of 60 ppb, compared to 6 out of 12 areas for the current standard, 9 out of 12 for a standard with a level of 70 ppb, and 10 out of 12 for a standard with a level of 65 ppb (U.S. EPA, 2014c, Table 4-3). Finally, a revised standard with a level of 60 ppb would be expected to further reduce long-term O3 concentrations based on the types of metrics that have been reported in recent epidemiologic studies to be associated with respiratory morbidity (i.e., seasonal averages of daily maximum 8-hour concentrations).[336]

Thus EPA's own assessment indicates that a standard of 70 ppb would be effective in limiting long-term exposures shown to increase the risk of premature death in only 9 of 12 urban study areas, and a standard of 65 ppb would do so in only 10 out of 12 areas. Of the options analyzed, only a standard of 60 ppb would eliminate dangerous exposures in all 12 study areas.

Fundamentally, EPA's proposal argues that an 8-hour average standard of 70 or 65 ppb would reduce long-term ozone concentrations and therefore mortality risks compared to the current standard, but fails to acknowledge that millions would be left unprotected at a standard of 70 or 65.

The fact that EPA places "less weight" on epidemiological-based risk estimates does not equate to a finding that such estimates are not entitled to material weight. EPA must protect against the adverse effects shown in the epidemiological studies and associated risk estimates unless the agency finds based on reasoned analysis that above-cited factors render the results so unreliable as to make them not credible. EPA has made no such finding here, and the record would not support such a finding. The fact that the Jerrett results are from only one study does not undermine their credibility, particularly given EPA's finding that the study was well-designed. And the fact that the Jerrett study did not identify a clear threshold does not undermine the PA finding of "a relatively high degree of confidence in the linear concentration-

[335] *See* Policy Assessment at 3-75; *see* HREA at 7-6.
[336] Proposed Rule at 75,302.

response function for 'long-term' ozone concentrations at least as low as 56 ppb…"[337] EPA cites[338] the following language from the CASAC letter:

> In light of the potential nonlinearity of the C–R function for long-term exposure reflecting a threshold of the mortality response, the estimated number of premature deaths avoidable for long-term exposure reductions for several levels need to be viewed with caution.

But CASAC also found:

> In terms of quantitative risk assessment, the most relevant scientific evidence available is with respect to epidemiologic estimates of the relationship between ambient concentration and mortality. Thus, EPA's risk assessment appropriately focuses on this particular heath end point for long-term exposure. [339]

Taken together, these statements simply mean that CASAC felt the precise numerical estimates of deaths avoidable should be viewed with caution

Further, in response to EPA's claim that most of the study areas would have violated the current standard, we note that there is a linear concentration-response relationship at least down to 56 ppb, and possibly below.

The Policy Assessment also notes that:

> a revised standard with a level of 60 ppb would be expected to further reduce long-term O3 concentrations based on the types of metrics that have been reported in recent epidemiologic studies to be associated with respiratory morbidity (i.e., seasonal averages of daily maximum 8-hour concentrations).

EPA must set the 8-hour standard with a margin of safety sufficient to prevent long-term exposures to ozone that are likely to cause chronic adverse effects on lung capacity, and other respiratory health endpoints, including premature death.

The currently available information on long-term effects shows the need for an 8-hour standard of 60 ppb, at the lower end of the range recommended by CASAC and EPA staff scientists. The primary standard must "be set at a level at which there is 'an absence of adverse effect' on [] sensitive individuals" such as children, the elderly, and people with respiratory illnesses. *Lead Indus. Ass'n v. EPA*, 647 F.2d 1130, 1153 (D.C. Cir. 1980). The long-term studies cited above show that adverse effects are likely at ozone levels at and above 60 ppb: Accordingly, the primary standard must be set at a level no higher than 60 ppb.

[337] Policy Assessment at 3-76.
[338] Proposed Rule at 75,277, n. 85, citing CASAC Letter 2014b at 3.
[339] CASAC Letter 2014b at 4.

xxv. EPA's Proposal Unlawfully and Arbitrarily Discounts and Fails to Protect Against Adverse Effects Shown in Epidemiological Studies

As shown above, numerous epidemiological studies show strong associations between a variety of serious health impacts and ozone levels well below 70 ppb, including at and below 60 ppb. In deciding on the level of the standard, however, EPA either disregards or discounts the results of most of these studies. In so doing, EPA acted illegally and arbitrarily for the following reasons:

1. Except for the panel studies, EPA seems to focus almost entirely on studies published after those addressed in the 2006 CD. There is no rational basis for such an approach.

2. The focus on newer studies results in EPA arbitrarily disregarding or discounting relevant evidence. For example, EPA indicates that the only single city epidemiological study showing statistically significant associations with adverse effects in an area that would have met the current standard is the 2009 Mar and Koenig study, and no single city studies show associations in areas that would have a met a standard of 70 ppb or below. See, e.g., 79 Fed. Reg. at 75299/2-3 ("None of the single-city studies evaluated in section 4.4.1 of the PA…provide evidence for O3 health effect associations in locations meeting a standard with a level of 70 ppb or below."). EPA also asserts that there were no multicity studies for which air quality data indicated that all cities included in the analyses would likely have met alternative standard levels. Id. 75307/1-2.

These assertions ignore: a) the panel studies cited in section 4.4.1 of the PA, which do show associations in places where the subjects were exposed to ozone that never exceeded various levels below 70 ppb; and b) numerous studies compiled in the last review, and EPA's analysis thereof. As to the latter, Appendix 3B from the Staff Paper in the last review shows a number of multi-city and single city studies showing statistically significant associations in places that would have met a 98th percentile ozone standard set at various levels below 70 ppb. These include:

- Respiratory Symptoms: Mortimer et al., 2002, 8 U.S. cities (morning symptoms); Delfino et al., 2003, San Diego, CA; Ross et al., 2002, East Moline, IL (morning symptoms); Ross et al., 2002, East Moline, IL (Evening symptoms)

- Lung Function Changes: Mortimer et al., 2002, 8 U.S. cities (am PEF (%)); Ross et al., 2002, East Moline, IL PEF (L/min); Brauer et al., 1996, Fraser Valley, BC, FEV1 (mL).

- Emergency Department Visits: Delfino et al., 1997, Montreal (>64yo); Delfino et al, 1997 Montreal (>64yo);

- Hospital Admissions: Cardiovascular Diseases: Burnett et al, 1997, Toronto CV.

- Hospital Admissions: Specific Cardiovascular Diseases: Koken et al, 2003, Denver CO, myocardial infarction; Koken et al, 2003, Denver CO , Coronary Atheroschlerosis; Koken et al, 2003, Denver CO, Pulm Heart Disease.

- Hospital Admissions: Respiratory Diseases: Burnett et al, 1997 16 Canadian City; Burnett et al, 1997 Toronto; Yang et al., 2003 Vancouver (<3 yo); Yang et al., 2003 Vancouver (65+ yo).

- Hospital Admissions, Asthma: Burnett et al, 1999, Toronto.

- Hospital Admissions: Other Respiratory Diseases: Burnett et al. 1999, Toronto, Respiratory infection

- Mortality: Total nonaccidental: Vedal et al. 2003, Vancouver

3. EPA's dismissive treatment of the multi-city studies is based on EPA's view that such studies are somehow lacking in probative value unless it is shown that, during the study period, all of the cities in the study would have met the 98th percentile form of the ozone standard at alternative standard levels under consideration. EPA also asserts that there is greater uncertainty in multi-city studies where individual city effect estimates are not presented because it is not possible to assign the multicity health effect associations to the air quality in any one study location or to the air quality in a particular subset of locations. 79 Fed. Reg. 75307. Neither CASAC nor the PA found that the multi-city studies should be discounted on these grounds. Although the PA acknowledges there are uncertainties in multi-city studies, it also points out that the multi-city studies have advantages in terms of statistical power and number of people and variety. Moreover, the PA finds that in four Canadian multicity studies showing positive and statistically significant associations between ozone and hospital admissions or mortality, the "effect estimates are largely influence by locations meeting the current standard," suggesting "a relatively high degree of confidence in the presence of associations with mortality and morbidity for ambient O3 concentrations meeting the current standard." PA 3-63 to -64. See also PA 3-67. The PA further shows that, in two of these studies, the majority of cities would have met a standard of 65 ppb. PA. 4-14. The PA concludes that multicity epidemiologic studies report positive and statistically significant associations with mortality and morbidity based largely on distributions of ambient O3 levels that would have been allowed by alternative standards with levels of 70 or 65 ppb, but not 60 ppb. PA 4-17. The PA also finds that cut-point analyses of the multicity study by Bell suggests that the large majority of air quality distributions that provided the basis for positive and statistically significant associations with mortality (i.e., for the 30 and 35 ppb cut points) would likely have met the current O3 standard. PA 3-72, -73.

4. EPA's discounting of results where the study area would have violated the 98th percentile form of a given standard level is arbitrary. As noted by CASAC member Dr. Ana Diez Roux, the " informativeness of these studies depends on the actual distribution and range of ozone concentrations investigated rather than on whether the standard was or was not met." CASAC 11-26-12 Letter to Administrator at A-12. EPA does not explain why confidence in the association of an 8-hour ozone exposure with adverse effects is somehow undermined merely because the area in which the association is shown would have violated a 98th percentile form of the standard at the same or higher exposure level. EPA does not discount the results of chamber studies based on whether the subjects live in areas that would have violated the relevant standard levels: There is no rational basis for treating epidemiological studies differently.

5. EPA's dismissive treatment of the multi-city ozone studies in this review in sharp contrast to its reliance on such studies for particulate matter See .eg.78 Fed. Reg. 3086, 3150, 3154-55 (2013).

2. A Standard Above 60 ppb Cannot Be Justified On This Record

a. EPA Does Not, and Cannot, Show How Any Level Above 60 ppb Will Protect Sensitive Populations with an Adequate Margin of Safety

As described above, exposure to 8-hour average ozone levels of 60 ppb will cause adverse effects to sensitive populations. D.C. Circuit case law is clear: "NAAQS must protect not only average healthy individuals, but also 'sensitive citizens' such as children, and if a pollutant adversely affects the health of these sensitive individuals, EPA must strengthen the entire national standard." *Coal. of Battery Recyclers Ass'n*, 604 F.3d at 618 (internal quotation marks and alterations omitted) (quoting *American Lung Ass'n*, 134 F.3d at 389, and *Lead Indus. Ass'n*, 647 F.2d at 1152-53). Thus, any level above 60 ppb will unlawfully allow adverse effects to persist.[340]

The evidence shows that a level above 60 ppb would not provide any margin of safety for sensitive populations. The NAAQS, "as required by the [Clean Air Act]," must "provide an adequate margin of safety for vulnerable subpopulations." *Am. Farm Bureau Fed'n*, 559 F.3d at 525-26. A standard that allows adverse effects to persist perforce provides no margin of safety at all. At the very least, EPA has failed to explain --in light of the evidence and scientific recommendations before it—how a standard with a level of 65 to 70 ppb provides an adequate margin of safety for sensitive subpopulations.

b. EPA's Inconsistent Treatment of Adverse Effects is Arbitrary and Capricious

EPA irrationally claims that adverse effects have only been demonstrated as low as 72 ppb, and that it "has less confidence that adverse effects will occur following exposures to O_3 concentrations as low as 60 ppb."[341] In particular, EPA relies on chamber studies' not finding "statistically significant increases in respiratory symptoms" below 70 ppb.[342] Thus, the agency treats lung function decrements as adverse only if combined with respiratory symptoms.[343]

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[340] Further, as discussed below, the 4th-highest form EPA proposes for the NAAQS would allow up to three exposures above 60 ppb to persist every year. Thus, looking at the NAAQS holistically, *see* Proposed Rule at 75,305/2 ("the degree of protection provided by any NAAQS is due to the combination of all of the elements of the standard"), EPA has not justified why a level above or of 60 ppb, coupled with the 4th-highest form, would protect public health.

[341] *See, e.g.*, Proposed Rule at 75,304/3-05/1, 75,309/3.

[342] *Id.* at 75,305/1; *see also id.* at 75,309/3, 75,310/2.

[343] EPA further does not treat the effect as adverse unless the occurrences of both the lung decrements and respiratory symptoms are statistically significant, and the respiratory symptoms are statistically significant when compared with the symptoms observed under filtered air conditions.

This approach conflicts with EPA's own statements elsewhere in the Proposed Rule. EPA itself states that it "considers estimates of the occurrence of O3-induced FEV_1 decrements $\geq$ 10 and 15 percent as surrogates for the occurrence of adverse health outcomes."[344] EPA and the PA further agree that exposure to ozone at 60 ppb causes respiratory effects, including lung function decrements that equal or exceed these 10 percent and 15 percent levels.[345] EPA itself notes that decrements may be adverse in terms of "population risk," where exposure to air pollution increases the risk to the population even though it might not harm lung function to a degree that is, on its own, "clinically important" to an individual:

> Exposure to air pollution that increases the risk of an adverse effect to the entire population is adverse, even though it may not increase the risk of any individual to an unacceptable level. For example, a population of asthmatics could have a distribution of lung function such that no individual has a level associated with clinically important impairment. Exposure to air pollution could shift the distribution to lower levels that still do not bring any individual to a level that is associated with clinically relevant effects. However, this would be considered to be adverse because individuals within the population would have diminished reserve function, and therefore would be at increased risk to further environmental insult (U.S. EPA, 2013a, p. lxxi; and 75 FR at 35526/2, June 22, 2010).[346]

Indeed, ozone exposure may make people more susceptible to respiratory infection, allergies, and asthma.[347] Yet EPA has arbitrarily failed to appropriately consider population risk.

Nor is EPA's proposed approach to the adversity of lung function decrements consistent with EPA's past practice, ATS guidelines, or CASAC's advice, which are the only three sources of guidance EPA cites in discussing what makes an effect adverse.[348] In promulgating a NAAQS for SO_2 in 2010, EPA explained that, under the 2000 ATS guidelines, "diminished reserve lung function in a population that is attributable to air pollution is considered an adverse effect under ATS guidance."[349] In promulgating that NAAQS, unlike here, the lung function decrements were not statistically significant at the group mean level, yet EPA still found the decrements observed there were adverse.[350] In the last ozone NAAQS review, EPA itself also acknowledged that a lung function decrement of 10 percent or greater "represent[s] a level that should be considered adverse for asthmatic individuals."[351] A substantial percentage—at least 10 percent—of healthy, young adults experience such decrement at 60 ppb,[352] and EPA, CASAC, and the PA all agree that at-risk populations, like people with asthma, are likely to experience at least the same decrements at the same level of ozone exposure.[353] Finally, CASAC plainly stated that "FEV_1 decrement of $\geq$10 percent is a scientifically relevant surrogate for adverse health outcomes for

[344] Proposed Rule at 75,306/1.
[345] *Id.* at 75,311/2; Policy Assessment at 4-56; *see also id.*at 75,250-51.
[346] Proposed Rule at 75,263/1.
[347] Policy Assessment at 3-31.
[348] *See id.* at 75,263/1-64/3
[349] 75 Fed. Reg. at 35,526/2 (June 22, 2010).
[350] *Id.*
[351] 73 Fed. Reg. at 16,454/3-55/1 (Mar. 27, 2008).
[352] Proposed Rule at 75,250/2 & n.37 (noting that percentage is an underestimate).
[353] *See, e.g., id.* at 75,265/1, /3, 75,280/1 n.92, 75,287/1, 75,288/3, 75295/3, 75,296/2 & n.127.

people with asthma and lung disease" and that there are "adverse effects, including clinically significant lung function decrements and airway inflammation, after exposures to 60 ppb ozone in healthy adults with moderate exertion."[354] Both EPA's failure to provide any rational explanation for its treatment of what makes an effect adverse here and its unexplained departure from the sole sources of guidance, as well as its own past finding, it has relied on in the past are arbitrary. *See American Lung Ass'n*, 134 F.3d at 392-93.

EPA's irrational treatment of the adverse effects observed at 60 ppb is especially exacerbated by its departure from both the PA and CASAC's findings that the effects observed at 60 ppb are adverse.[355] The PA examines population risk and expressly finds that the decrements found in the chamber studies at or near 60 ppb fit the ATS criteria for adverse effects.[356] As described above, CASAC said that the effects at 60 ppb were adverse. EPA thus has failed its obligations to confront and explain any departures from CASAC's advice, and to address analyses in the PA that are missing in the Proposed Rule. *E.g.*, *Am. Farm Bureau Fed'n*, 559 F.3d at 521 (citing 42 U.S.C. § 7607(d)(3) and *NRDC v. EPA*, 902 F.2d 962, 967-68, 970 (D.C. Cir. 1990), *vacated in other part* 921 F.2d 326 (D.C. Cir. 1991)).

c. EPA's Reliance on Its Exposure Risk Assessment to Reject a Level of 60 ppb Was Arbitrary and Capricious, Particularly Because EPA Is Relying On People Not Going Outdoors to Conclude That the Level of Air Pollution Outdoors Will Not Harm Them

As discussed above, section 109 of the Clean Air Act mandates that EPA set the NAAQS at a level that allows the public to go out whenever and wherever they want and that the air will be clean enough to "protect the public's 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. 42,381 (Dec. 18, 1970) (remarks of Senator Muskie, floor manager of the conference agreement).

> 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. at 8210.

In this case, EPA is unlawfully using the HREA to subvert this mandate. EPA is using the HREA to justify a level of pollution that is unsafe if actually experienced on the ground that people will probably stay indoors. EPA's rejection of a primary standard of 60 ppb relies

[354] CASAC Letter 2014a at 6-7.
[355] *See*, e.g., Policy Assessment at 4-12 ("Thus, 60 ppb is a short-term exposure concentration that may be reasonably concluded to elicit adverse effects in at-risk groups").
[356] *Id*. at 4-52 ("In addition, as discussed in section 3.1.3, such a decrease in mean lung function meets the ATS criteria for an adverse response given that a downward shift in the distribution of FEV_1 would result in diminished reserve function, and therefore would increase risk from further environmental insult."); *see also id*. at 4-9, -10.

heavily upon the exposure risk assessment, which concludes that because people will supposedly not go outside there will be relatively few "exposures of concern" at this level.[357] In other words, EPA is relying on people not going outside to set a higher level of ozone that would be unsafe when people do in fact go outside: EPA reasons that no one will actually be harmed if they just stay indoors.

Even if the EPA's approach was not completely unlawful—which it is—EPA's exposure risk assessment is itself also technically flawed as it is unable to effectively capture key inputs regarding outdoor activity patterns and, as a result, ends up potentially underestimating dangerous exposure incidents by sensitive groups such as children and outdoor workers by over 30 percent.[358] Furthermore, EPA emphasizes the role of averting behavior, noting that it may result in an overestimation of exposures of concern, and cites this behavior (essentially staying indoors or not exercising) in order to reach what it deems an acceptable level of risk. But again, this is inconsistent with the Clean Air Act's mandate that EPA set a level that allows people to go outside and not put themselves at risk by doing so. What this all means is that first, adequate protection of children and others who are active outside, especially during ozone season, is being overlooked, and second, the levels of exposure incidents EPA relies on in its decision-making may only be reached if people choose not to be active outdoors, but instead stay inside to avoid dangerous air pollution.

The Clean Air Act requires that uncertainty be resolved in favor of protecting the public health, and that the precautionary principle be applied. Uncertainties in exposure modeling and data gaps may be unavoidable, but where they do exist, a precautionary and protective approach must be applied. When such uncertainties suggest that it might be unsafe to go outside whenever and wherever desired, or "suggest the possibility that some at-risk groups could experience more frequent exposures of concern than indicated by estimates made"[359], EPA must act in favor of protecting sensitive groups.

i. EPA's Proposed Range of 65 to 70 ppb Undervalues Exposure Risk for Children and Outdoor Workers

According to EPA, "the HREA concludes that exposures of concern could be underestimated for some individuals who are frequently and routinely active outdoors during the warm season," including "outdoor workers and children who are frequently outdoors."[360] Finding that long-term activity records for such groups do not exist, the HREA notes that estimates of high exposures for such groups in the APEX modeling are limited as a result.[361] To better assess this deficiency, EPA performed limited exposure model sensitivity analyses using subsets of activity diaries that were deemed similar to the more active groups. Estimating exposures for children who spend large portions of time outdoors during the summer when

[357] *See e.g.*, Proposed Rule at 75,297: "Based on the HREA results, meeting an O3 standard with a level of 60 ppb would be expected to almost eliminate exposures of concern to O3 concentrations at or above 60 ppb. To the extent lower exposure concentrations may result in adverse health effects in some people, a standard level of 60 ppb would be expected to also reduce exposures to O3 concentrations below 60 ppb."
[358] Proposed Rule at 75,274.
[359] *Id*.
[360] *Id*. at 75,273 (emphasis added), HREA sec. 5.5.
[361] HREA at 5-78.

school is traditionally out of session, EPA found that exposures of concern could be underestimated by 33 percent.[362]

EPA has estimated that all children in urban areas will experience approximately three times more of these "exposures" than adults.[363] At the same time, children also experience some of the greatest physiological sensitivity to ozone pollution (see section IV.B.1 on children's health). Children spend large amounts of time being physically active outdoors, especially during periods (e.g., summers, afternoons) when ozone concentrations are high, and are thus more likely to experience dangerous exposures. During the summer, many children spend significant amounts of time outdoors and being active at camps and at play. Summertime is also when ozone levels are highest, thus putting children at greater risk.

Every year more than 11 million children, and adults, attend camps in the U.S., not to mention over 1.5 million camp workers.[364] Most camps last for at least a week, and many for a month or two, and over 71 percent of registered camps are overnight camps.[365] The majority of campers are under age 12[366] and the majority of camp activities take place outdoors. According to one survey, over 75 percent of both overnight and day camps said campers spent more than seven hours a day outside in the open air and only one camp reported less than two hours a day spent in the open air.[367] Studies of children at summer camps, where they experience sustained outdoor activity, show that short-term ozone exposure is associated with decreased respiratory function,[368] even when restricted for levels above 60 ppb.[369] Studies of children at summer camp with asthma have also shown increased risk of respiratory symptoms on high pollution days as well as decreases in lung function.[370]

Playing outside and participating in athletics are important parts of a child's development. Over 21 million children play team sports on a regular basis[371], although current trends show children beginning to spend too much time indoors being inactive.[372] Compelling children to stay inside to avoid poor air quality and health impacts only further jeopardizes

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[362] *Id*. at 5-49; Proposed Rule at 75,274.
[363] Proposed Rule at 75,272.
[364] American Camp Association, ACA Facts and Trends, *available at* http://www.acacamps.org/media/aca-facts-trends (accessed Mar. 16, 2015).
[365] T. Bennett (2014). Fall 2014 Camp Enrollment and Staff Recruitment Survey, American Camp Association, *available at* http://www.acacamps.org/sites/default/files/images/research/improve/Fall_2014_Enrollment_Survey_Report.pdf.
[366] *See id*.
[367] P.A. James & K.A. Henderson, Camps and Nature Report (2007), *available at* http://www.acacamps.org/sites/default/files/images/research/understand/camps_nature_report.pdf.
[368] *See e.g*., P.L. Kinney, G.D. Thurston, M. & Raizenne (1996). The effects of ambient ozone on lung function in children: A reanalysis of six summer camp studies, *Env. Health Perspect.*, 104(2): 170-174; .D. Thurston, M. Lippmann, M.B. Scott, & J.M. Fine (1997). Summertime haze air pollution and children with asthma, *Am. J. of Resp. and Crit. Care Med.*, 155(2): 654-660.
[369] Spektor, D.M., et al. (1988). Effects of ambient ozone on respiratory function in active, normal children, *Am. Rev. of Resp. Disease*, 137(2): 313-320.
[370] Thurston et al. 1997, *supra* note 367,
[371] The Aspen Institute, Project Play-Facts: Sports Activity and Children, *available at* http://www.aspenprojectplay.org/the-facts.
[372] *See e.g., id.*; Let's Move, Learn the Facts, *available at* http://www.letsmove.gov/learn-facts/epidemic-childhood-obesity.

healthy lifestyles and development. In one highly significant study, McConnell et al. showed that active children who played three or more sports growing up in communities with eight-hour ozone levels ranging from 55.8 to a maximum of 69 ppb were three times more likely to develop asthma than their peers in communities with lower ozone levels, ranging from 30.6 to 50.9 ppb.[373] Even so, the high pollution communities in the study would uniformly be in attainment with EPA's proposed standard of 70 ppb—meaning that EPA's proposed standard of 70 ppb would do nothing to address these exposures and the associated health impacts. The authors found that "new diagnoses of asthma is associated with heavy exercise in communities with high concentrations of ozone, thus, air pollution and outdoor exercise could contribute to the development of asthma in children."[374]

Indeed, even a cursory review of camp programs in the United States makes evident just how many camps emphasize extended outdoor time for children. As is evident in the advertisements for these camps included in Exhibit 7 many of these camps focus on extensive outdoor exercise and sports, including baseball, basketball, football, soccer, field hockey, tennis, swimming, canoeing, kayaking, hiking, and ropes courses. And they cater to a wide age range of children from five and six years old through the teen years. These are precisely the types of conditions that lead to maximum exposure—extended outdoor time, with heavy exercise and ventilation—for one what the scientific literature demonstrates is a highly sensitive population.

Children who are active outdoors aren't the only ones at special risk, although children are particularly vulnerable to the health impacts of ozone exposure due to their life stage and physiological factors. Even healthy adults who are active outdoors face greater exposure and health risks. In one study by Kinney and Lippmann of 72 sophomore cadets from the U.S. Military Academy at West Point during summer training demonstrated a seasonal decline in respiratory function when outdoors in the presence of ozone.[375] Similarly, a study of outdoor farm workers in British Columbia found associations between ozone concentrations and decreases lung function that persisted at mean daily maximum 1-hour ozone concentrations of less than 40 ppb.[376] Another study of healthy, adult, amateur cyclists found ozone associations to be associated with decreases in lung function and increases in respiratory symptoms, even at 8-hour ambient concentrations of less than 60 ppb.[377]

The HREA is also missing activity profiles from outdoor workers, so EPA performed an assessment to estimate what exposures of concern might look like for this group. EPA calculated that approximately 30 percent of outdoor workers, who aren't adequately accounted for in the exposure modeling, would experience at least one exposure of concern over 60 ppb, compared to 5 to 8 percent of the general population of workers.[378] These results "suggest the possibility that

[373] R. McConnell et al. (2002). Asthma in exercising children exposed to ozone: A cohort study, *Lancet*, 359(9304): 386-391.
[374] *Id*.
[375] P.L. Kinney. & M.Lippmann, Respiratory effects of seasonal exposures to ozone and particles, *Arch. of Env. Health*,, 55(3), 210-216 (2000).
[376] M. Brauer, J. Blair, & S. Vedal (1996). Effect of ambient ozone exposure on lung function in farm workers, *Am. J. of Resp. and Crit. Care Med.*, *154*(4): 981-987.
[377] B. Brunekreef et al. (1994). Respiratory effects of low-level photochemical air pollution in amateur cyclists, *Am. J. of Resp. and Crit. Care Med.*, 150(4): 962-966.
[378] HREA at 5-51.

some at-risk groups could experience more frequent exposures of concern than indicated by estimates made using the full database of activity diary profiles."[379]

It should also be noted that exposures may at the same time be underestimated for all groups due to the use of the maximum 8-hour average metric. EPA comments that "that it is entirely possible multiple benchmark exceedances could occur for an individual on certain high ambient O3 concentration days" but that "this is not a practical output for the purposes of this assessment."[380]

ii. EPA Impermissibly Relies on People Staying Indoors to Avoid Repeated or Prolonged Exposure to the Air

At the same time, EPA suggests that exposures are probably overestimated due to people staying indoors.[381] In evaluating alternative primary standards, EPA repeatedly emphasizes the HREA modeling and quantitative exposure assessment, though EPA questions whether the exposure assessment may overestimate exposures, due to people staying indoors to avoid poor air quality, while also underestimating critical exposures for children and others who are active outdoors. EPA suggests that behavioral changes to stay indoors and reduce exposure to poor air quality can "reduce the percentages of children estimated to experience exposures of concern at or above the 60 or 70 ppb benchmark concentrations by approximately 10 to 30 percent, with larger reductions possible for the 80 ppb benchmark."[382] The HREA modeling of averting behavior showed that 15.3 percent of schoolchildren in Detroit spent on average 44 minutes less time outdoors –"resulting in approximately one percentage point or fewer children experienced exposures at or above any of the selected benchmark levels."[383] For asthmatic schoolchildren, this resulted in 20.3 percent spending on average 44 minutes less time outdoors and resulted in approximately two percentage points or fewer experienced exposures at or above any of the selected benchmark levels.[384]

Although, in the HREA, EPA recognizes evidence that many people, including children, avert outdoor activity and that the exposure estimates may be too high due to averting behavior, it is also unclear whether there is double-counting of the impact of averting behavior. In a footnote, EPA notes that "we do not know if any diary day represents the activities of an individual who averted. Thus it is entirely possible that the 'no averting' simulation includes, to an unknown extent, individuals who spent less time outdoors than would have occurred if absolutely no individuals averted."[385] In other words, EPA does not know if the activity data it is using already includes double-counting averting behavior, but chooses to assume it does not in their basis for calculating exposures of concern and, consequently, reduces its estimates of risks. In order to reach what EPA unlawfully and irrationally concludes is justifiably low risk, EPA is discounting the number of events of unsafe air based upon EPA's deeply flawed—and unlawful—analysis that people will for one reason or another not be outside and thus will not be

[379] Proposed Rule at 75,274.
[380] HREA at 5-18.
[381] *See* Proposed Rule at 75,276.
[382] Proposed Rule at 75,274; HREA Fig. 5–15.
[383] HREA at 5-53.
[384] HREA at 5-53 – 5-54.
[385] HREA at 5-53, n. 27.

exposed to the unsafe levels of ozone that EPA is proposing to allow. Putting aside the unlawful nature of EPA's approach, from a purely technical assessment, EPA cannot rely on averting behavior as a measure to lower risk and exposures of concern because it cannot accurately gauge the extent of averting behavior based on the data it has.

EPA's discussion of averting behavior inappropriately, unethically, and unlawfully amounts to advocating for it, and cannot provide an option for protecting public health: "Individuals can reduce their exposure to O3 by altering their behaviors, such as by staying indoors, being active outdoors when air quality is better, and by reducing their activity levels or reducing the time being active outdoors on high-O3 days."[386] EPA's observation that "the results suggest that exposures of concern could be overestimated, particularly in children, if the possibility for averting behavior is not incorporated into estimates" unlawfully and arbitrarily promotes a standard that would effectively compel sensitive populations to remain indoors.[387]

iii. The Sufficiency of the NAAQS to Protect Public Health Cannot Depend upon Sensitive Populations Remaining Indoors

Air quality standards cannot rely on avoidance behavior in order to protect the public health and sensitive groups. The promise of the Clean Air Act is to clean up the air so that anyone can go outside anytime and the air will be safe to breathe, and that assessment of safety will be based on the best assessment of up-to-date science. Thus, meeting the NAAQS does not mean that the air is only safe to breathe part of the time. It would be unlawful for EPA to set the standard at a level that is contingent upon people spending most of their time indoors.

Not only is it unlawful to set the standard based on an expectation that people will spend most of their time indoors, but such an approach carries serious consequences for public health. There are significant health benefits to be gained by spending time outdoors. Forcing sensitive groups to stay indoors in order to avoid unhealthy air quality levels puts them in a position of either avoiding outdoor activity, thus raising the risk of obesity and other health problems, or going outside and running the risk of respiratory disease and asthma attacks. Unfortunately, some opponents of a more protective ozone standard have already argued that staying indoors is an acceptable protection. EPA cannot base its final decision on any aspect of assumed avoidance of exposure.

EPA is effectively discouraging the very types of healthy exercise and outdoor behaviors that we want to be encouraging and that are in such critical jeopardy in this country. In recent years, childhood obesity has skyrocketed in the U.S., more than doubling for children and quadrupling for adolescents over the past three decades.[388] In 2012, over one third of children and adolescents were either overweight or obese, putting them at greater risk for cardiovascular disease, diabetes, bone and joint problems, social and psychological problems, and even cancer

[386] Proposed Rule at 75,269.
[387] Proposed Rule at 75,274.
[388] CDC, Childhood Obesity Facts, *available at* http://www.cdc.gov/healthyyouth/obesity/facts.htm; *see also* Let's Move, *supra* note 371.
[388] CDC, Childhood Obesity Facts, *available at* http://www.cdc.gov/healthyyouth/obesity/facts.htm

in the long-term.[389]

Outdoor activities and spending time in natural environments can help combat the rise of health conditions such as obesity, type 2 diabetes, vitamin D deficiency, and attention-deficit/hyperactivity disorder (ADHD), all of which sedentary indoor lifestyles contribute to.[390] Research shows that both physical activity and exposure to nature promote good physical and mental health and well-being, especially for children.[391] Time spent outdoors is associated with higher levels of physical activity.[392] Children who spend more time outside not only tend to be more physically active, but are also less likely to be overweight.[393] Furthermore, in addition to increasing physical activity and health, time spent outdoors is shown to improve children's sense of well-being and feelings of health, safety, and satisfaction.[394] Time spent outside has cognitive benefits and can help decrease childhood stress, improve attention, and reduce the symptoms of ADHD.[395] Physical activity and contact with nature are also beneficial to psychological well-being, leading to improvements in self-esteem, depression and mood.[396]

Instead of encouraging active, healthy lifestyles, EPA proposed range of 65 to 70 ppb would result in levels of air quality that force people inside. Additional health benefits provided by standards of 65 or 70 ppb are undermined to the extent they depend on lifestyle changes that increase other health risks. The purpose of the Clean Air Act is "to protect and enhance the quality of the Nation's air resources as to promote the public health and welfare and the productive capacity of its population." 42 U.S.C. § 7401(b)(1). As such, it is unacceptable for EPA to rely on people staying indoors in order to deem the level of risk satisfactorily low. Everyone has the right to go outside—and to not experience health risks when doing so.

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[389] *Id.*

[390] L.E. McCurdy, K.E. Winterbottom, S.S. Mehta, & J.R. Roberts (2010). Using nature and outdoor activity to improve children's health, *Current Problems in Pediatric and Adolescent Health Care*, 40(5): 102-117.

[391] G. Godbey (2009). Outdoor Recreation, Health, and Wellness: Understanding and Enhancing the Relationship, Resources for the Future; J. Pretty et al. (2009). Nature, Childhood, Health and Life Pathways. Interdisciplinary Centre for Environment and Society Occasional Paper 2009-02; S.A. Muñoz (2009). Children in the outdoors: a literature review, Sustainable Development Research Centre.

[392] A.R. Cooper et al. (2010). Patterns of GPS measured time outdoors after school and objective physical activity in English children: the PEACH projec, *Int. J. of Behavioral Nutrition and Phys. Activity*, 7(31); A. Nilsson et al. (2009). Correlates of objectively assessed physical activity and sedentary time in children: a cross-sectional study, *BMC Public Health*, 9(322); T. Hinkley, D. Crawford, J. Salmon, A.D. Okely, & K. Hesketh (2008). Preschool children and physical activity - A review of correlates, *Am. J. of Prev. Med*., 34(5): 435-441; J. Sallis, J. Prochaska, & and W. Taylor (2000). A Review of Correlate of Physical Activity of Children and Adolescents, *Med. and Science in Sports and Exercise*, 32(5): 963–75.

[393] V. Clelandet al. (2008). A prospective examination of children's time spent outdoors, objectively measured physical activity and overweight, *Intl.. J. of Obesity*, 32*(*11):1685-1693.

[394] C. Wood, R. Hine, & J. Barton (2011). The health benefits of the Youth Outdoor Experience (YOE) project: University of Essex.

[395] McCurdy et al. 2010, *supra* note 326.; M.G. Berman, J. Jonides, & S. Kaplan, The Cognitive Benefits of Interacting With Nature. *Psychological Science*, 19(12),. 1207-1212 (2008); N.M. Wells, At Home with Nature: Effects of "Greenness" on Children's Cognitive Functioning, *Environment and Behavior*, 32(6), 775-795 (2004); F. Kuo. & A. Taylor, A,A Potential Natural Treatment for Attention-Deficit/Hyperactivity Disorder: Evidence from a National Study, *Am. J. of Public Health*, 94(9), 1580–86 (2004); Godbey 2009, *supra* note 390.

[396] Wood et al. 2011, *supra* note 393.

d. EPA's Risk and Exposure Assessment Has Substantial Limitations that Severely Underestimate the Health Impacts of Exposure

EPA's quantitative HREA is intended to elucidate the potential public health implications of current and proposed alternative standards. With any such analysis, multiple analytical assumptions can greatly affect the result and multiple uncertainties can diminish the reliability of those results. The selection of factors such as risk coefficients used, health impacts analyzed, years of air quality monitoring utilized, exposure assumptions, and other variables can have a big impact on the outcomes. Although the HREA clearly demonstrates three key aspects: the burden that current concentrations of ozone pose to public health; the inadequacy of the current standard of 75 ppb to provide the legally required protection; and the evidence that the lower the standard, the greater the reductions in exposure and risk that could be achieved. However, several factors and limitations in the scope of the risk assessment result in severe underestimates of exposures of concern and of health impacts.

i. Infants and Small Children Are Excluded

First, children aged zero to five are among the most susceptible populations, but they are not included in the quantitative risk and exposure assessment examining lung function decrements in children. This is a serious omission. Approximately 20 million children in the U.S. fall within the 0-5 age group, yet they are completely disregarded in the risk estimates.

We know that the lungs are not fully developed at birth, and that ozone exposure can affect the post-natal development of the lungs. Infants are exposed to outdoor air at an early age and they are active outdoors from the time they are mobile. They experience higher exposures than adults because of their increased breathing rate and activity level. By excluding infants and young children from the analysis, EPA greatly underestimates exposures of concern and risks.

ii. A Very Narrow Subset of Health Endpoints are Evaluated

Second, the health endpoints considered in the REA are extremely limited, and do not represent the comprehensive array of health effects attributable to ozone exposure. For instance, the analysis mainly looks at lung function decrements, respiratory hospitalizations, and mortality. Respiratory emergency room visits are considered in only two cities, and respiratory symptoms in only one city. Because of the quantitative nature of the results, the available effect estimates are emphasized in the proposal, to the exclusion of the vast array of other health endpoints of concern. As a result, EPA understates risks.

iii. Exposures of Concern are Evaluated for Limited Scenarios

Third, the analysis only considers alternative levels the standard of 70, 65, and 60 ppb. At 60 ppb, we know that healthy adults experience lung function declines and inflammation with 6.6 hour exposures. Children and those with respiratory disease are considered sensitive

populations and would likely experience effects at lower concentrations. Further, the risk function is based on a 6.6 hour exposure, whereas the standards are based on 8 hours. For these reasons, by failing to consider exposures of concern of 55 ppb, the exposure assessment underestimates risks.

iv. A Risk Function Derived from Study of Healthy Adults Is Inappropriately Applied to Children

The HREA uses a risk function derived from a controlled human exposure study of healthy young adults to estimate lung function decrements in children, including children with asthma. This assumption could result in an underestimate of risk.

v. The Geographic Scope of the Analysis is Limited

The HREA is too limited in its geographic scope. Respiratory admissions are estimated for only one city, Boston. Respiratory function declines are estimated for only 15 cities (and only 14 cities for the 60 ppb standard level, *e.g.*, 79 Fed. Reg. at 75,297 n.135), and respiratory emergency department visits are assessed for 2 cities, Atlanta and New York. Mortality estimates are made for just 12 cities. Effects on rural populations are not estimated, even though some rural areas experience higher ozone concentrations than urban areas.

vi. EPA Looks at Just Two 3-Year Periods to Estimate Air Quality

The EPA analysis uses air quality data from two 3-year periods: 2006 to 2008; and 2008 to 2010. The results for these two periods vary considerably, depending on which time frame had better air quality. The proposal presents results from an average of these two periods. From a standpoint of evaluating public health protection, it is more appropriate to place greater weight on the years with poorer air quality.

vii. The Emission Control Strategies Modeled Are Limited

Finally, it should be emphasized that the emissions control strategies modeled in the draft HREA are limited. Localities will consider many additional factors such as updated emissions inventories and a variety of NO_x and VOC control measures that were not analyzed in the risk assessment. By focusing on NO_x reductions, the risk assessment results are distorted for some areas of the country.

e. Despite These Limitations, the Health Risk and Exposure Assessment and Regulatory Impact Analysis Support Setting the Standard at 60 ppb

As described elsewhere in these comments, EPA misuses the HREA to try to justify a weaker standard. Yet that document, even with its underestimates of exposures and health effects, actually undercuts EPA's efforts. So too does the Regulatory Impact Assessment, where

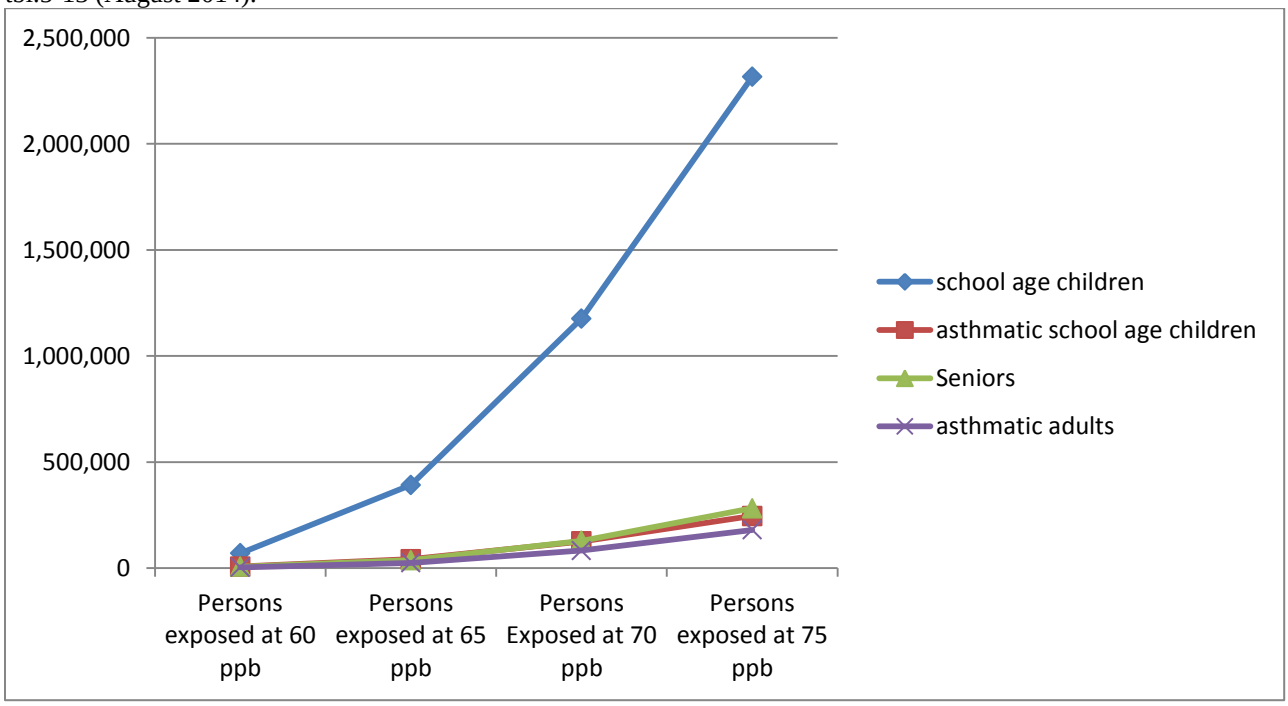
it estimates the health benefits resulting from different levels of the standard. What follows is a sampling of data derived from those documents. More supportive data can be found in them.

Controlled human exposure studies have shown, as discussed elsewhere, that exposure to 60 ppb of ozone over a 6.6 hour period causes adverse effects in healthy adults. Sensitive subjects, like children, asthmatics, and seniors, are more vulnerable. Table 6and figure Y1 below demonstrate not only that the current standard is much weaker than required, but also that only a standard of 60 ppb brings the occurrence of these dangerous exposures to anywhere approaching zero—which is what the Clean Air Act demands.

TABLE 6 **Mean number of people with at least one daily maximum 8-hr average O_3 Exposure at or above 60ppb while at moderate or greater exertion** (includes 15 urban area studies)
(Data derived from EPA, EPA-452/R-14-004a, Health Risk and Exposure Assessment for Ozone: Final Report 5-87 tbl.5-13 (August 2014).

Standard 8-hr level	School age children	Asthmatic school-age children	Asthmatic adults	Seniors (aged 65-95)
60 ppb	70,000	7,700	4,100	6,800
65 ppb	392,000	42,000	25,000	38,000
70 ppb	1,176,000	126,000	83,000	129,000
75 ppb	2,316,000	246,000	180,000	282,000

FIGURE 6 **Increased Exposure for At-Risk Groups According to Ozone Standard** (with at least one daily 8-hr average O3 exposure at or above 60ppb while at moderate or greater exertion)
(Data derived from EPA, EPA-452/R-14-004a, Health Risk and Exposure Assessment for Ozone: Final Report 5-87 tbl.5-13 (August 2014).



Similarly, Tables 7 and 8 and Figures 7 and 8 below show that a standard of 60 ppb is necessary to truly reduce occurrence of adverse lung function decrements in children to levels near what the Clean Air Act calls for.

TABLE 7 **Maximum Percentage Children (ages 5-18) Experiencing Decreased Lung Function According to Ozone Level** (During the O_3 Season)
(Data derived from EPA, EPA-452/R-14-004a, Health Risk and Exposure Assessment for Ozone: Final Report 6-22 to -27 tbls.6-4, 6-5 (August 2014).

Standard	Percent of Children Experiencing 1 Day or More of Lung Function Decrement ≥10%	Percent of Children Experiencing 1 Day or More of Lung Function Decrement ≥ 15%	Percent of Children Experiencing 6 Days or More of Lung Function Decrement ≥10%
60 ppb	13%	3%	3%
65 ppb	18%	4%	4%
70 ppb	20%	5%	5%
75 ppb	22%	6%	6%

FIGURE 7 **Increase in Maximum Percentage of Children (ages 5-18) to Experience Lung Function Decrement Arranged According to Alternate Ozone Standards**
(Data derived from EPA, EPA-452/R-14-004a, Health Risk and Exposure Assessment for Ozone: Final Report 6-22 to -27 tbls.6-4, 6-5 (August 2014).

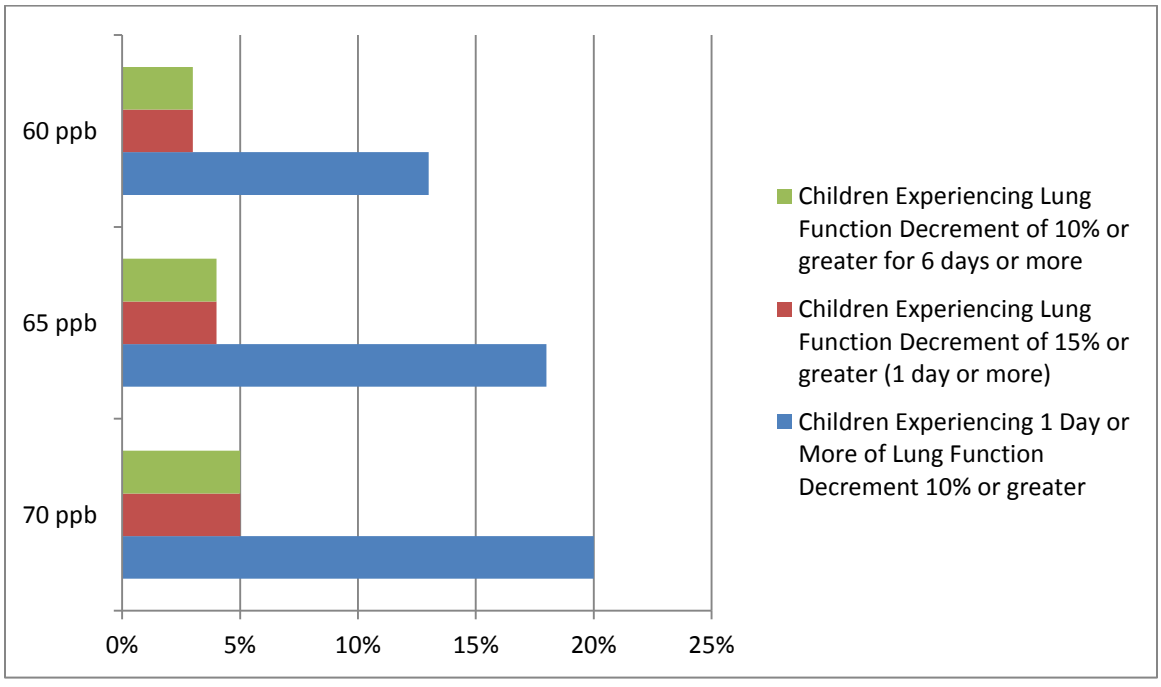
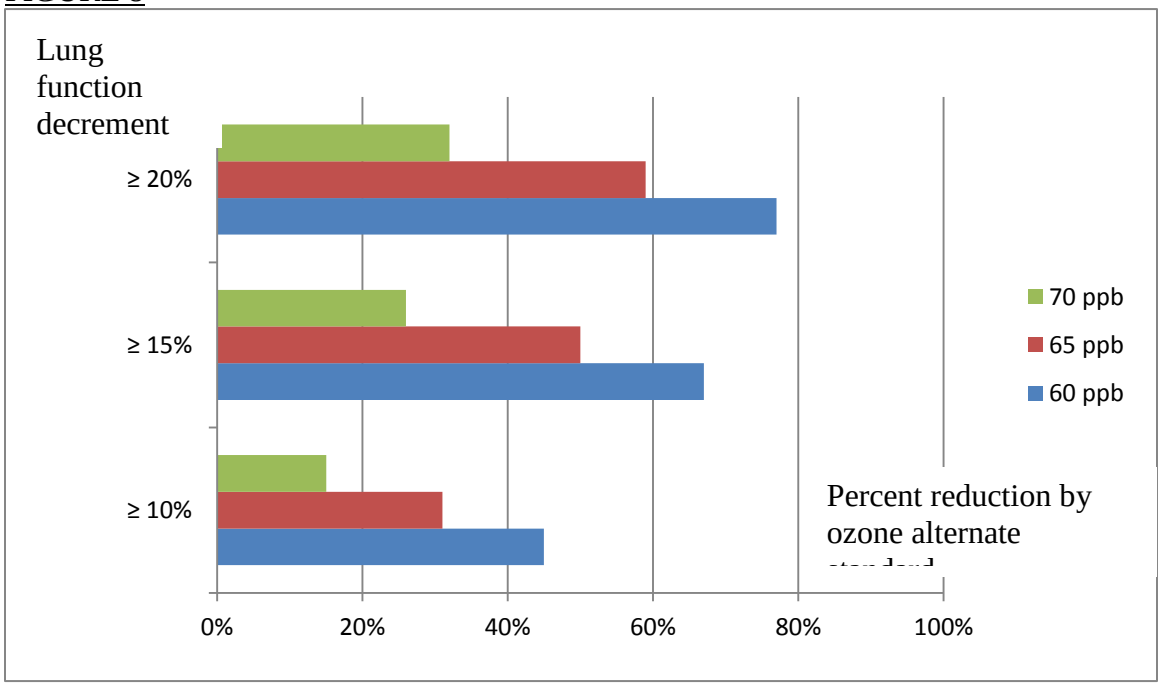


TABLE 8 **Lung Function Decrements for Alternate Ozone Standards for Children (ages 5-18) Experiencing One or More Decrements Per Season**
(Data derived from 79 Fed. Reg. at 75,298 tbl.5)

Decreased Lung Function	Alternate Ozone Standard	Number of children experiencing decrement	Average % reduction from current standard
≥10%	60 ppb	1,404,000	45%
	65 ppb	1,896,000	31%
	70 ppb	2,527,000	15%
≥15%	60 ppb	225,000	67%
	65 ppb	356,000	50%
	70 ppb	562,000	26%
≥20%	60 ppb	57,000	77%
	65 ppb	106,000	59%
	70 ppb	189,000	32%

FIGURE 8

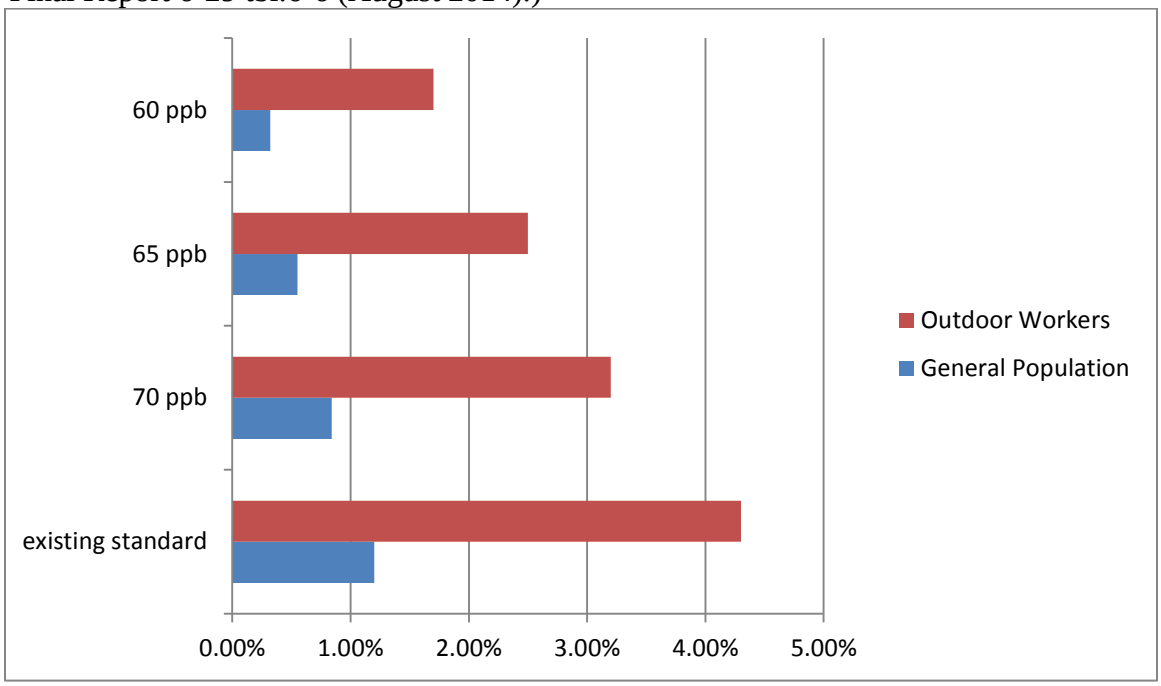


Percent reductions in each urban study area were calculated and averaged across areas (excluding NY for 60ppb) (Data derived from 79 Fed. Reg. at 75,297-98 tbl.4)

In the Proposed Rule, EPA declines to highlight the benefits of more protective standards for outdoor workers, who form another sensitive population. As summarized in Figure 9 below, EPA's quantitative assessment of those benefits again demonstrates the virtue of the 60 ppb standard, both for outdoor workers and the general population, in terms of preventing lung function decrements of ≥15%.

FIGURE 9 **Percentage of Outdoor Workers (age 19-35) Experiencing One or More Lung Function Decrement of at least 15%**

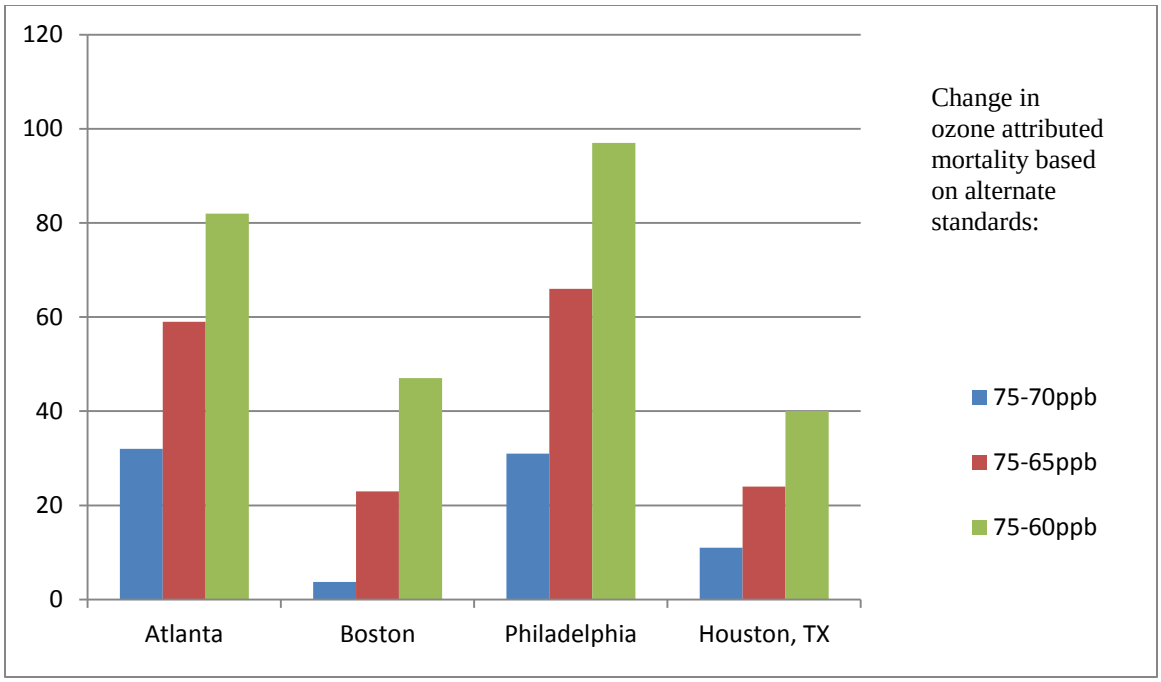
(Data derived from EPA, EPA-452/R-14-004a, Health Risk and Exposure Assessment for Ozone: Final Report 6-29 tbl.6-6 (August 2014).)



A more protective ozone standard would also prevent deaths from long-term ozone exposure. Figure 10 below compares how many lives would be saved at a 60 ppb level (over the current standard of 75 ppb) against the numbers saved at 65 ppb and 70 ppb (both also over the current standard) in just a few of the cities EPA assessed. This shows that a 70 ppb standard would have only a slight effect, and a 60 ppb standard would be many times more effective.

FIGURE 10 **Long-term Respiratory Mortality in Certain Cities attributed to Ozone based on 2009 Air Quality Data**

(Data derived from EPA, EPA-452/R-14-004a, Health Risk and Exposure Assessment for Ozone: Final Report 7-62 tbl.7-12 (August 2014))



In the Regulatory Impact Analysis, EPA modeled a broader range of health benefits resulting from more protective ozone standards. These data allow calculations of the benefits—shown in Table 9 and Figure 11 below—foregone by establishing unlawfully and irrationally under-protective standards, as opposed to a 60 ppb standard. These show that a 60 ppb standard would reap more than twice the benefits of a 65 ppb standard, compared to a 70 ppb standard. EPA must set the level at 60 ppb.

TABLE 9 **Yearly Death and Morbidity Rates According to Ozone Standards: 2025 National Benefits (except California)**

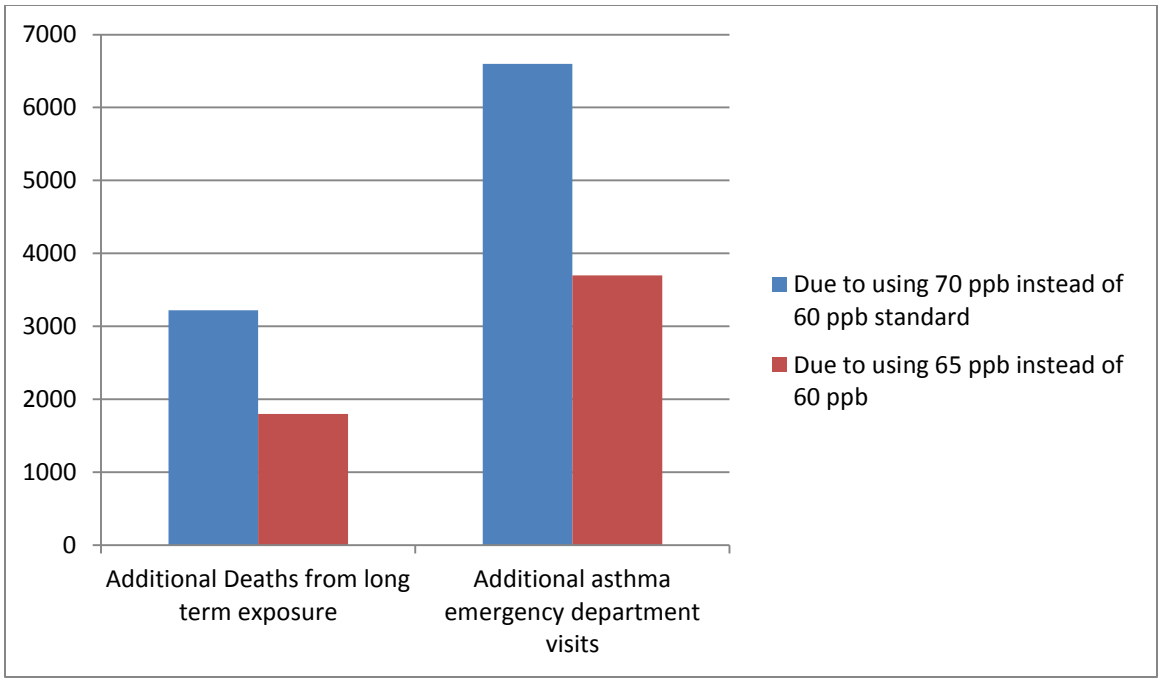
(Data derived from EPA, EPA-452/P-14-006, Regulatory Impact Analysis of the Proposed Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone at ES-14 to -15 tbl.ES-7 (November 2014))

	Alternative Standard Levels		
	60ppb versus 70ppb: Additional persons protected	60 ppb versus 65 ppb: Additional persons protected	65ppb versus 70ppb: additional persons protected:
Short-term exposure related premature deaths avoided*	**1560**	900	660
Long-term exposure-related premature deaths avoided (age 30+)	**3220**	1800	1420

Non-fatal heart attacks (18-99)*	**2500**	1400	1100
Respiratory Hospital Admissions (age 0-99)	**2390**	1400	990
Cardiovascular hospital admissions (age 8-99)	**770**	420	350
Asthma emergency department visits (age 0-99)	**6600**	3700	2900
Acute bronchitis (age 8-12/3rd to 7th grade children)	**3310**	1800	1510
Asthma exacerbation (age 6-18)	**1,480,000**	840,000	640,000
Lost Work Days (age 18-65)	**275,000**	160,000	115,000
Minor restricted activity days (age 18-65)	**6,000,000**	3,300,000	2,700,000
Upper & lower respiratory symptoms (children 7-14)	**106,000**	60,000	46,000
School loss days (ages 5-17/kindergarten to 12th grade	**1,570,000**	900,000	670,000

*Maximum amount avoided death or morbidity rates found for each alternative standard used as a basis for comparison.

FIGURE 11 **Increase in Deaths and Asthma-Induced Trips to the Emergency Room Between Alternate Ozone Standards.**
(Data derived from EPA, EPA-452/P-14-006, Regulatory Impact Analysis of the Proposed Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone at ES-14 to -15 tbl.ES-7 (November 2014))



3. EPA Proposes a Form of the NAAQS-- the Fourth Highest 8-hour Maximum Averaged Over 3 Years--That Will Fail to Deliver the Public Health Protection that EPA Itself Ultimately Concludes is Necessary; Whatever Nominal Level EPA Adopts, the Form Will Allow Repeated Exceedances of the Standard Each Season Without Triggering a Nonattainment Designation or Any Obligation to Clean up the Air and EPA Must Compensate For This By Setting An Accordingly Lower Nominal Level Of The Standard

In light of the form of the standard proposed, EPA's basis for rejecting a level of 60 ppb and instead proposing a level in the range of 65 to 70 ppb is based on a flawed characterization of the relationship between the level of the standard and actual occurrences of ozone levels in the ambient air. EPA purports to reject a standard of 60 ppb on the basis that it "would place a large amount of weight on the potential public health importance of virtually eliminating even single occurrences of exposures of concern at and above 60 ppb."[397] Instead "the Administrator focuses on the extent to which a revised standard would be expected to protect populations from experiencing two or more O_3 exposures of concern (i.e., as a surrogate for repeated exposures)."[398] Yet given the proposed form of the standard—the 3-year average of 4th high daily maximum 8-hour ozone values—the standard is far less protective of human health than its nominal level suggests. Indeed a standard with a nominal level of 65 or 70 ppb would expressly authorize numerous occurrences of and exposures to 8 or more hours to levels in excess of these levels each year without triggering a nonattainment designation or other ameliorative requirements under the Clean Air Act and requirements to clean the air. Given EPA's

[397] Proposed Rule at 75,309.
[398] *Id*. at 75,305.

recognition that even single exposures[399]—and certainly multiple exposures[400]—to levels of 70 ppb are potentially adverse, a standard of 70 ppb unlawfully fails to protect the public from acknowledged adverse effects. Likewise, empirical data show that a standard of 65 ppb would allow numerous exposures of concern each year. Because multiple 8 hour occurrences of air quality levels above the NAAQS will regularly occur in areas meeting the 4th high form of the standard, a lower level of the NAAQS is required to achieve EPA's claimed level of health protection.

a. Multiple Aspects of the NAAQS Define the Stringency of the Standard

Several elements define each National Ambient Air Quality Standard. The standard has a form, an averaging time, an indicator, and a level. Each of these elements, as well as other factors such as monitoring regime affects the stringency of the standard. For example, EPA's original primary NAAQS for photochemical oxidants established in 1971 differed from the current and proposed standard in several of the elements, which rendered the 1971 standard significantly more health protective. While the level of the original 1971 NAAQS—0.08 ppm—was slightly higher than the current 0.075 ppm level, each of the other elements worked together to make the standard far more protective of health. Rather than using ozone only as an indicator, the 1971 standard used total photochemical oxidants, a broader category of pollutants and thus one that occurs in higher concentrations than ozone alone. Rather than using an 8-hour averaging time, the 1971 standard employed a 1-hour averaging time. And rather than rely on a statistical form that considers the 4th high 8-hour daily max averaged over three years, the 1971 standard used a deterministic "not to be exceeded more than one hour per year" form. Taken together, the four elements of the 1971 NAAQS rendered it far stronger than the 2008 standard now in effect.

EPA's proposed revision of the standard is flawed because it attempts to divorce its analysis of each of the elements of the standard and consider each in isolation. Thus, EPA discusses the scientific research on ozone in the context of the level of the standard only. But every element of the form that EPA has proposed then renders the nominal numerical level far less protective than—and further divorced from—the underlying science. Thus EPA's attempt to bestow the imprimatur of CASAC approval on its election to use the 4th high 8-hour daily max form is deeply problematic. CASAC supported the form as part of a complete standard with level, indicator, and averaging time. Altering any one of these elements changes the overall protectiveness of the standard. Selecting a 4th high 8-hour daily max form necessitates that EPA recognize that it is far less protective than a deterministic 1st high or even a three-year average 1st high 8-hour daily max form.

[399] In the proposed rule EPA acknowledges that "single exposures of concern could be adverse for some people, particularly for the higher benchmark concentrations (70, 80 ppb) where there is stronger evidence for the occurrence of adverse effects." *Id*. at 75,289.

[400] EPA acknowledges moderate lung function decrements at 70 ppb. And EPA acknowledges that "Thus it has been judged that repeated occurrences of moderate responses, even in otherwise healthy individuals, may be considered to be adverse since they could well set the stage for more serious illness." *Id*. at 75,264.

Stated another way, if EPA wants to ensure that people in fact are not exposed to levels of 70 ppb for 8 hours more than once, and yet EPA wants to use the proposed form of the 4th highest averaged over three years—which dismisses the first three occurrences each year—EPA will have to set the nominal numerical standard far below 70 ppb. Indeed, as discussed below, empirical data from the last 13 years shows a very consistent pattern: the 1st high averaged over three years is approximately 7.6 ppb higher than the 4th high, the second high is approximately 3.8 ppb over the 4th high. As a result, if EPA adopts the form it has proposed, a nominal level of 70 ppb is actually equivalent to a level of approximately 78 ppb, which is clearly far in excess of what all the science, and EPA itself, says is a safe level of the NAAQS. However, as discussed below, each element of the form adds a further weakening the standard.

b. EPA's Proposed Form and Averaging Time of the NAAQS Requires that the Level of the Standard be Lowered to Achieve EPA's Claimed Level of Health Protection.

i. An 8-hour Averaging Time is Longer than the Controlled Human Exposure Studies Relied upon by EPA; Extrapolating the Results of These Studies to 8 Hours Would Produce Larger and More Pervasive Impacts to Test Subjects, Necessitating a Lower Level of the NAAQS.

Despite discussing at length the controlled human exposure studies identifying acknowledged adverse effects (combination of lung function decrements and symptoms) at 72 ppb based on exposures in healthy individuals for 6.6 hours, EPA proposes a form of the standard with a longer averaging time than the studies it bases its analysis on. Ozone levels that EPA identifies as being of concern based on exposures of 6.6 hours will pose even greater threats to health under longer 8-hour exposures. Indeed, as discussed above, it is well established by now that there is a dose response curve to ozone. In order to protect public health with an adequate margin of safety, EPA cannot simply treat the results of human controlled studies based on 6.6- hour exposures as determinative of impacts based upon 8-hour exposures. Indeed, an 8 hour exposure period will result in a 21 % increase in exposure time over the 6.6 exposures in the clinical trials. Stated another way, if EPA sets a level of 70 ppb, but uses an 8-hour exposure timeframe instead of the 6.6 hour timeframe used in the controlled human exposure studies that underpin EPA's standard, EPA must first identify the effect that that has on overall dose response outcomes and account for it in setting the nominal numerical level of the standard. Simply put, if EPA is going to increase dose time, it needs to understand the effects of doing so and then build additional protection into the standard by adopting a level that accounts for the less protective form and averaging time of the proposed standard.

Moreover, the form of the standard considers ozone levels only during one 8-hour period each day. Elevated ozone concentrations outside this peak period are wholly disregarded under the proposed form of the standard. Unsafe ozone levels could occur for 24 straight hours on one day at average levels above the NAAQS and yet that day would be treated no differently from a day during which elevated ozone concentrations were limited to a period of only eight hours.

In using an 8-hour averaging time while relying on scientific studies with exposure times of only 6.6 hours, EPA must adjust the level of the standard downward from what the scientific studies directly concluded in order to account for the longer averaging time of the proposed standard.

ii. Because of EPA's Truncating Conventions, Any Ozone Standard Proposed is, as a Practical Matter, a Full Part Per Billion Higher; EPA Must Account for this Under-Protection in Setting the Level of the Standard

EPA's truncating conventions result in almost a full part per billion of under-protection in the level of the NAAQS that needs to be accounted for when setting the new standard. EPA assiduously documents throughout its proposal that the Schelegle et al. study identifying adverse effects in healthy individuals at "70 ppb" actually had an average exposure concentration of 72 ppb, attempting to draw a meaningful distinction between levels of 72 and 70 ppb. But EPA fails to acknowledge anywhere in its proposal that because of its truncating conventions, an area can have 8-hour daily maximum ozone levels and design values almost a full part per billion in excess of the level of the NAAQS without triggering a nonattainment designation. That is, built into EPA's calculation of design values is a full part per billion of under-protection because both 8-hour daily maximum concentrations and 3-year averages of 4th highest maximum 8-hour average concentrations are recorded in parts per million and truncated after the third decimal digit.[401] The practical consequence is that a standard of 70 ppb is effectively a standard of 71 ppb and a standard of 65 ppb is effectively a standard of 66 ppb. EPA must take into account this under-protectiveness by setting the level of the standard one part per billion lower than would otherwise be required by the science.

iii. Because of the Real-World Relationship Between the 3-Year Average of 1st, 2nd and 4th Highest 8-hour Daily Monitored Maximum Ozone Concentrations, Use of a 4th Highest Form Is Equivalent to Use of a 1st Highest Form with a Level 7.5 to 8 ppb Higher and to a 2nd Highest Form with a Level 3.5 to 4 ppb higher; EPA Must Account for this Under-Protection in Setting the Level of the Standard

EPA must reconcile its choice of a 4th highest form for the proposed standard with its identified concerns regarding single and multiple exposures to elevated levels of ozone. Even

[401] *See id*. at 75,352 ("The EPA is proposing to maintain the requirement that hourly O3 concentration data be reported in parts per million (ppm) to three decimal places. Any decimal digits reported beyond three decimal digits will be truncated, consistent with past practice (40 CFR part 50, Appendix P, section 2.1) and the typical measurement uncertainty associated with most O3 monitoring instruments."); *see also* 73 Fed. Reg. 16,501 (Mar. 21, 2012) ("Consistent with the current approach for computing 8-hour averages, in calculating 8-hour average O3 concentrations from hourly data, any calculated digits beyond the third decimal place would be truncated, preserving the number of digits in the reported data. In calculating 3-year averages of the fourth highest maximum 8-hour average concentrations, digits to the right of the third decimal place would also be truncated, preserving the number of digits in the reported data.")

without accounting for the use of averaging across three years, which adds a significant additional degree of under-protection, a 4th highest standard by definition allows three days per year during when ozone levels are unconstrained and are not considered in classifications of attainment. If ozone levels on these peak days are appreciably higher than on the 4th highest day, given EPA's acknowledged concerns regarding single or multiple (defined by EPA as 2 or more) exposures to elevated ozone concentrations, EPA must account for the degree of under-protection in setting the level of the NAAQS. Stated another way, as discussed below, if EPA adopts a nominal level of 70 ppb but uses a fourth highest form, the result is a standard that is effectively a 77-78 ppb standard.

To determine the relationship between 1st, 2nd and 4th high ozone concentrations, we calculated the "design values" for five different three-year periods for each Core Based Statistical Area (CBSA) in the country as follows. For every ozone monitor in the United States, we pulled the 1st through 4th maximum 8-hour daily ozone levels for each year from 2008 to 2014. For each CBSA and each three-year period (i.e., 2008-2010, 2009-2011, up to 2012-2014) we identified the "design value" for the CBSA under three different assumed forms of the standard: (1) the current 4th highest form (i.e., 3-year average of 4th highest 8-hour daily maximums using the monitor site in the CBSA for which this value is highest); (2) a 1st highest form (i.e., 3-year average of 1st highest 8-hour daily maximums for the monitor site in the CBSA for which this value is highest; and (3) a 2nd highest form (i.e., 3-year average of 2nd highest daily 8-hour maximums for the monitor site in the CBSA for which this value is highest). This provided three different design values for each three-year period from 2008-2010 to 2012-2014– one based on the first highest, one based on the second highest, and one based on the fourth highest. For each of these three-year periods, we then examined both the ratio of the 1st , 2nd and 4th highest design value forms for each CBSA and also the absolute ppb difference between the 1st , 2nd and 4th highest design value forms for each CBSA. The tables below show the averages and standard deviations for the ratio and for the difference (see Exhibit 8).

Figure 12

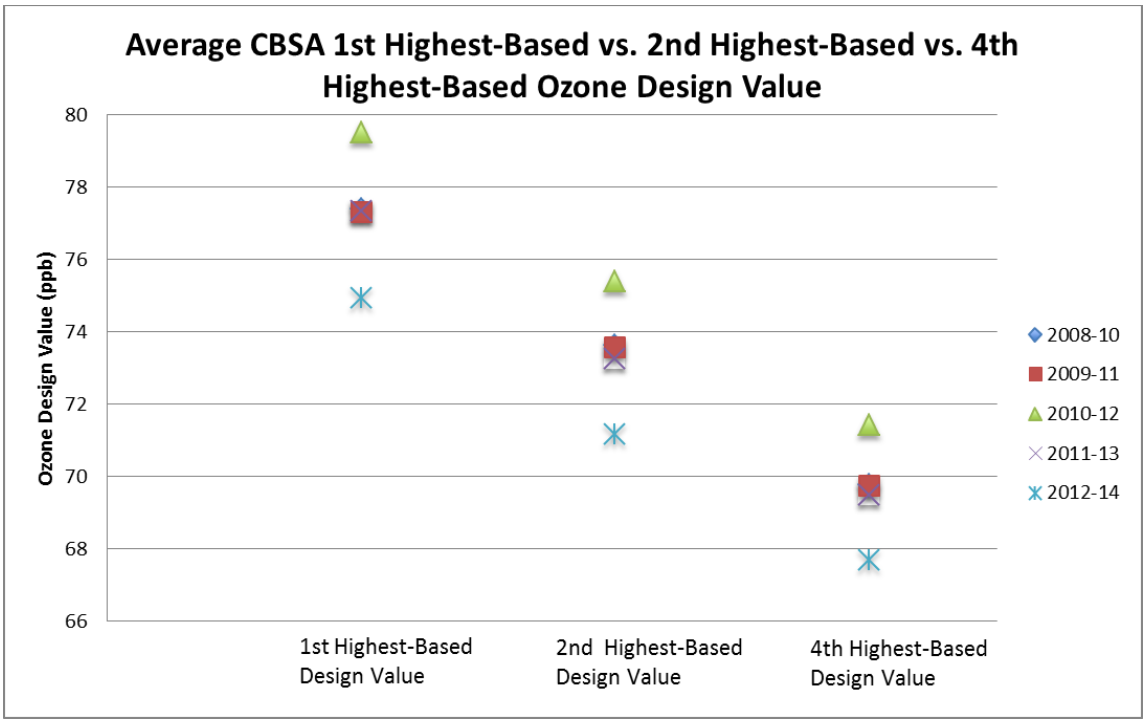


Table 10

2010	Average	Diff from 4th	Standard Deviation
4th highest CBSA design value	69.7672		
1st highest CBSA design value	77.3780	7.6108	3.4602
2nd highest CBSA design value	73.6117	3.8445	2.0162

	Average	Standard Deviation
Ratio of 1st to 4th highest CBSA design value	1.1083	0.0467
Ratio of 2nd to 4th highest CBSA design value	1.0548	0.0279

Table 11

2011	Average	Diff from 4th	Standard Deviation
4th highest CBSA design value	69.7518		
1st highest CBSA design value	77.3016	7.5498	3.4341
2nd highest CBSA design value	73.5679	3.8161	1.9936

	Average	Standard Deviation
Ratio of 1st to 4th highest CBSA design value	1.1081	0.0465

Ratio of 2nd to 4th highest CBSA design value	1.0546	0.0278

Table 12

2012	Average	Diff from 4th	Standard Deviation
4th highest CBSA design value	71.4171		
1st highest CBSA design value	79.5074	8.0903	3.4710
2nd highest CBSA design value	75.3799	3.9628	1.8935

	Average	Standard Deviation
Ratio of 1st to 4th highest CBSA design value	1.1126	0.0448
Ratio of 2nd to 4th highest CBSA design value	1.0551	0.0242

Table 13

2013	Average	Diff from 4th	Standard Deviation
4th highest CBSA design value	69.4930		
1st highest CBSA design value	77.3419	7.8489	3.4064
2nd highest CBSA design value	73.2508	3.7578	1.7766

	Average	Standard Deviation
Ratio of 1st to 4th highest CBSA design value	1.1121	0.0444
Ratio of 2nd to 4th highest CBSA design value	1.0539	0.0236

Table 14

2014	Average	Diff from 4th	Standard Deviation
4th highest CBSA design value	67.6946		
1st highest CBSA design value	74.9183	7.2237	3.0649
2nd highest CBSA design value	71.1688	3.4742	1.5290

	Average	Standard Deviation
Ratio of 1st to 4th highest CBSA design value	1.1065	0.0428

Ratio of 2nd to 4th highest CBSA design value	1.0514	0.0221

The results are remarkably consistent across time.[402] For all three-year periods reviewed, the 4th highest design value is 7.2 to 8.1 ppb lower on average across CBSAs than a 1st highest "design value" and 3.5 to 4.0 ppb lower on average across CBSAs than a 2nd highest "design value."

The consequences for public health protection are massive. Retaining the current form of the NAAQS and setting the level at 70 ppb is equivalent to establishing a 1st max standard of between 77.2 and 78.1 ppb. That means, under a standard of 70 ppb using the current form, occurrences of and exposures to 8-hour concentrations in the range of 77.2 to 78.1 ppb would be anticipated annually in areas just meeting the NAAQS. And likewise, with a 4th highest standard of 65 ppb, occurrences of and exposures to 8-hour concentrations of 72.2 to 73.1 ppb (as well as multiple exposures between 70 and 77.2) would be anticipated annually in areas just meeting the NAAQS. Individual exposures to all of these concentrations for only 6.6 hours were found to produce both lung function decrements and symptoms, a fact that EPA does not dispute and a combination of effects that EPA acknowledges to be adverse. Consequently, given the proposed form of the standard, EPA cannot support a level in the range of 65 to 70 ppb.

Moreover, the above results illustrate that there will be multiple occurrences of ozone levels corresponding to exposures of concern in areas meeting a NAAQS of 65 to 70 ppb. The average 2nd highest "design value" for CBSAs ranges from 3.5 to 4 ppb higher than the 4th highest design values. Consequently, for areas just meeting a standard of 70 ppb, there would be anticipated to be multiple 8-hour occurrences of levels of 73.5 ppb or higher each year—exposures to which EPA acknowledges to be adverse based on controlled human exposures studies using exposures for only 6.6 hours.

For the urban areas included in EPA's Health Risk and Exposure Assessment, the differences between 1st, 2nd and 4th highest design values were even greater. Table 15 identifies the difference between 1st and 4th highest 3-year average design values for the three-year periods 2008-2010 through 2012-2014. Table 16 identifies the difference between 2nd and 4th highest 3-year average design values for the same five three-year periods. As the tables illustrate, the average difference between 1st and 4th highest design value across the five periods is 12.5 ppb. And the average difference between 2nd and 4th highest design value across the five periods is 6.1 ppb. These data show that in order to account for the form of the standard, in these areas, the level of the standard would need to be set 12.5 ppb lower to avoid individual exposures to the level of the NAAQS and 6.1 ppb lower to avoid multiple exposures to that level.

Table 15: Differences Between 1st and 4th Highest-Based Design Values for EPA HREA Study Areas, 2008 to 2014

Area	1st -4th Design	1st – 4th Design	1st – 4th Design	1st – 4th Design	1st – 4th Design	Average 1st – 4th Design

[402] These results are also consistent with California's testimony that 70 ppb not to be exceeded standard is equivalent to a 60 ppb standard.

	Value 2008-2010 (ppb)	Value 2009-2011 (ppb)	Value 2010-2012 (ppb)	Value 2011-2013 (ppb)	Value 2012-2014 (ppb)	Value for 2008 to 2014
Atlanta-Sandy Springs-Marietta, GA	13.0	12.0	16.0	16.7	16.7	**15.1**
Baltimore-Towson, MD	18.0	18.7	16.7	14.7	19.3	**17.5**
Boston-Cambridge-Quincy, MA-NH	8.7	10.0	13.0	12.0	10.0	**10.7**
Chicago-Naperville-Joliet, IL-IN-WI	10.3	12.7	11.0	10.3	6.7	**10.2**
Cleveland-Elyria-Mentor, OH	10.0	11.3	12.3	14.3	12.7	**12.1**
Dallas-Ft. Worth-Arlington, TX	10.7	7.0	9.3	n/a	n/a	**9.0**
Denver-Aurora, CO	7.3	12.3	14.0	12.0	6.7	**10.5**
Detroit-Warren-Livonia, MI	13.0	13.0	13.7	15.3	17.3	**14.5**
Houston-Sugar Land-Baytown, TX	15.7	15.0	15.3	18.0	19.0	**16.6**
Los Angeles-Long Beach-Santa Ana, CA	16.0	18.7	n/a	n/a	n/a	**17.3**
New York-Northern New Jersey-Long Island, NY-NJ-PA	8.7	12.7	14.7	16.0	14.3	**13.3**
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	11.0	8.7	9.3	7.0	9.7	**9.1**
Sacramento-Arden Arcade-Roseville, CA	13.7	9.3	9.3	6.7	6.3	**9.1**
St. Louis, MO-IL	10.0	9.3	9.7	13.7	10.0	**10.5**
Washington-Arlington-Alexandria, DC-VA-MD-WV	13.7	10.3	13.7	10.3	9.0	**11.4**

Table 16: Differences Between 2nd and 4th Highest-Based Design Values for EPA HREA Study Areas, 2008 to 2014

Area	2nd -4th Design Value 2008-2010 (ppb)	2nd – 4th Design Value 2009-2011 (ppb)	2nd – 4th Design Value 2010-2012 (ppb)	2nd – 4th Design Value 2011-2013 (ppb)	2nd – 4th Design Value 2012-2014 (ppb)	Average 2nd – 4th Design Value for 2008 to 2014
Atlanta-Sandy Springs-Marietta, GA	9.7	9.3	5.3	7.0	5.7	**7.4**
Baltimore-Towson, MD	8.3	7.3	7.7	8.0	8.7	**8.0**
Boston-Cambridge-Quincy, MA-NH	5.7	6.7	4.7	3.7	3.7	**4.9**
Chicago-Naperville-Joliet, IL-IN-WI	6.0	7.3	5.3	4.0	3.0	**5.1**
Cleveland-Elyria-Mentor, OH	8.0	8.7	5.3	5.0	3.7	**6.1**
Dallas-Ft. Worth-Arlington, TX	5.0	5.0	7.0	n/a	n/a	**5.7**
Denver-Aurora, CO	3.7	4.0	5.7	5.7	3.7	**4.5**
Detroit-Warren-Livonia, MI	5.3	4.7	5.3	6.3	5.3	**5.4**
Houston-Sugar Land-Baytown,	8.0	11.7	9.0	10.7	6.7	**9.2**

TX						
Los Angeles-Long Beach-Santa Ana, CA	4.3	7.0	n/a	n/a	n/a	**5.7**
New York-Northern New Jersey-Long Island, NY-NJ-PA	5.0	8.3	10.3	10.0	4.7	**7.7**
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	6.3	4.7	7.7	3.7	4.7	**5.4**
Sacramento-Arden Arcade-Roseville, CA	6.7	5.7	5.3	4.7	4.3	**5.3**
St. Louis, MO-IL	5.0	6.3	6.0	6.7	5.3	**5.9**
Washington-Arlington-Alexandria, DC-VA-MD-WV	5.3	5.0	7.7	3.3	2.3	**4.7**

c. Under EPA's Proposed Form of the Standard, a Level of 65 to 70 ppb Would Impermissibly Allow, in a Single Ozone Season, Numerous Exposures to Levels Far in Excess of Those Acknowledged to be of Concern without Triggering a Nonattainment Designation

The statistical analysis in the previous section is made concrete by considering monitored ozone data from CBSAs presently attaining standards of 70 and 65 ppb. Table 17 provides CBSAs with design values between 66 and 70 ppb —CBSAs that would be judged in attainment with a 70 standard--routinely record numerous days with maximum 8-hour concentrations of 70 ppb or higher in a single year, and almost uniformly record multiple levels of 70 ppb or higher in a single calendar year.[403] The Columbia, South Carolina area, for example, recorded up to 20 days with 8-hour maximum concentrations of 70 ppb or higher in one year, yet still achieved a 3-year design value of 69 ppb. Vineland-Millville-Bridgeton, New Jersey recorded 19 days in a single ozone season with 8 hour concentrations of 70 ppb or above while attaining a 3-year design value of 70 ppb. Cadillac, Michigan recorded 17 days in a single ozone season with 8 hour levels of 70 ppb or higher but met a standard of 70 ppb.

Indeed, across the nation there are untold numbers of areas that would be judged to be in compliance with a 70 ppb standard using the form that EPA is proposing—and that therefore would never have to clean its air or reduce pollution--but that would nonetheless regularly subject residents to 8 hour concentrations above 70 ppb standard: Pensacola, Florida recorded 16 days of 70 ppb or above, Jefferson City, Missouri, 15 days, Athens, Georgia, Brigham City Utah, Clarksville, Kentucky, Columbia, Missouri, and Lafayette, Indiana all recorded 14 days, Clinton, Iowa and Omaha-Council Bluffs, Nebraska, 13 days, Madison, Wisconsin, Huntington-Ashland, West Virginia-Kentucky-Ohio and Huntsville, Alabama 12 days—yet all of these CBSAs had maximum design values of 70 ppb or lower.

Nor are repeated occurrences in areas currently meeting a standard of 70 ppb limited to concentrations near 70 ppb. Millville, New Jersey registered 12 days with maximum 8-hour concentrations of 75 ppb or higher in a single ozone season while meeting a standard of 70 ppb.

[403] See Exhibit 10.

Columbia, South Carolina recorded 10 days with maximum 8-hour concentrations of 75 ppb or higher in the same ozone season while meeting a 3-year average of 69 ppb. As a result, Columbia, South Carolina would be judged to be in attainment with a 70 ppb, would be allowed to maintain that level of ozone air pollution forever, and would not have to take any steps to reduce levels of ozone--despite the fact that residents would be repeatedly exposed to 8 hour ozone levels above 70 ppb which EPA has judged to be unsafe. Many areas recorded at least six days in a single ozone season with levels of 75 ppb or higher for 8 hours while still attaining at a level of 70 ppb including Akron, Ohio, Cadillac, Michigan, Clinton, Iowa, Columbia, Missouri, Fayetteville, North Carolina, Huntington-Ashland, WV-KY-OH, Huntsville, Alabama, Omaha-Council Bluffs, Nebraska, Pensacola, Florida.

Of even greater concern, many areas recorded multiple days with maximum 8-hour concentrations of 80 ppb or higher in a single season (Akron, OH; Anderson, SC; Athens, GA; Cadillac, MI; Clarksville, KY; Columbia, MO; Durham, NC; Elizabethtown, KY; Fayetteville, NC; Florence, SC; Gulfport-Biloxi, MS; Huntington-Ashland, WV-KY-OH; Huntsville, AL; Jackson, MS; Johnstown, PA; Lakeland, FL; Madison, WI; Muncie, IN; Parkersburg-Marietta-Vienna, WV-OH; Pensacola, FL; Poughkeepsie, NY; Quincy, IL-MO; Watertown-Ft. Drum, NY), yet attained a standard of 70 ppb. Columbia, South Carolina had 4 days in a single ozone season with 8 hour concentrations of 80 ppb or higher yet its design value was only 69 ppb. And Millville, NJ recorded 7 days in a single season with 8-hour ozone levels of 80 ppb or higher and attained a standard of 70 ppb.

Indeed, some areas meeting a standard of 70 ppb even recorded levels of 85 ppb or higher.[404] This was true in at least 38 CBSAs with design values between 66 and 70 ppb, including Millville, NJ, which recorded 3 days with 8 hour concentrations of 85 ppb or higher while attaining a standard of 70 ppb. Elkhart, Indiana recorded an 8-hour ozone concentration of 111 ppb in 2012 yet attained a design value below 70 ppb for 2011 to 2013.

[404] See Exhibit 9.

Table 17: 2011-2013 Ozone Monitor Data for CBSAs with Design Value 0.066 to 0.070 ppm (Monitors with Design Value 0.066 to 0.070 ppm)

CBSA	State	County	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)
Akron	OH	Portage	Rockwell	391331001	0.067	11(8)	4(3)	1(1)	1(1)
Akron	OH	Summit	Patterson Park	391530020	0.068	14(9)	7(6)	3(2)	2(1)
Albany-Schenectady-Troy	NY	Albany	Loudonville	360010012	0.067	9(8)	3(2)	2(1)	0(0)
Anderson	IN	Madison	n/a	180950010	0.069	15(11)	3(3)	1(1)	1(1)
Anderson	SC	Anderson	n/a	450070005	0.068	14(9)	7(4)	3(2)	2(1)
Asheville	NC	Haywood	Purchase Knob	370870036	0.067	6(4)	3(2)	0(0)	0(0)
Athens	GA	Clarke	Fire Station #7	130590002	0.068	18(14)	8(5)	2(2)	1(1)
Augusta-Richmond	GA	Richmond	Bungalow Rd.	132450091	0.069	14(8)	5(3)	1(1)	0(0)
Augusta-Richmond	GA	Columbia	Riverside Park	130730001	0.068	13(9)	6(3)	2(2)	1(1)
Baraboo	WI	Sauk	Devils Lake SP	551110007	0.067	10(10)	2(2)	0(0)	0(0)
Berlin	NH	Coos	Mt. Washington	330074001	0.069	12(6)	2(2)	0(0)	0(0)
Boise City-Nampa	ID	Ada	n/a	160010017	0.068	10(5)	3(2)	1(1)	0(0)
Brigham City	UT	Box Elder	Brigham City	490030003	0.069	19(14)	3(2)	1(1)	0(0)
Cadillac	MI	Missaukee	n/a	261130001	0.070	21(17)	9(8)	2(2)	0(0)
Chambersburg	PA	Franklin	Methodist Hill	420550001	0.068	8(6)	2(1)	0(0)	0(0)
Clarksville	KY	Christian	Hopkinsville	210470006	0.069	20(14)	5(5)	0(0)	0(0)
Clarksville	KY	Trigg	Dover Rd.	212219991	0.070	14(12)	5(4)	3(3)	1(1)
Clinton	IA	Clinton	Rainbow Park	190450021	0.068	15(13)	7(7)	0(0)	0(0)
Columbia	MO	Boone	Finger Lakes	290190011	0.069	17(14)	7(7)	2(2)	1(1)

CBSA	State	County	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)
Columbia	SC	Richland	Sandhill Exp. Stn.	450791001	0.069	23(20)	11(10)	4(4)	1(1)
Corpus Christi	TX	Nueces	Corp. Christi West	483550025	0.070	9(6)	5(4)	0(0)	0(0)
Corpus Christi	TX	Nueces	CC Toluso	483550026	0.069	9(7)	4(3)	3(2)	1(1)
Dalton	GA	Murray	Ft. Mtn.	132130003	0.068	12(8)	2(2)	0(0)	0(0)
Daphne-Fairhope-Foley	AL	Baldwin	Fairhope	010030010	0.067	12(10)	2(2)	0(0)	0(0)
Decatur	AL	Morgan	Decatur	11030011	0.068	12(7)	4(3)	0(0)	0(0)
Deming	NM	Luna	Airport Rd.	350290003	0.067	6(5)	1(1)	0(0)	0(0)
Durham	NC	Person	Bushy Fork	371450003	0.069	8(6)	5(5)	2(2)	0(0)
Durham	NC	Durham	Durham Armory	370630015	0.068	11(7)	4(3)	1(1)	1(1)
Effingham	IL	Effingham	Central Jr. High	170491001	0.067	12(11)	2(2)	1(1)	1(1)
Elizabethtown	KY	Hardin	n/a	210930006	0.070	11(8)	3(3)	2(2)	1(1)
Elkhart-Goshen	IN	Elkhart	Bristol	180390007	0.067	14(9)	7(4)	2(1)	1(1)
Fayetteville	NC	Cumberland	Golfview	370511003	0.069	14(10)	7(6)	4(3)	0(0)
Fayetteville	NC	Cumberland	n/a	370510008	0.067	11(8)	3(2)	1(1)	0(0)
Fernley	NV	Lyon	Fernley Intermed. School	320190006	0.069	10(6)	3(2)	1(1)	0(0)
Florence	SC	Darlington	Pee Dee Exp. Stn.	450310003	0.066	11(7)	3(2)	2(2)	0(0)
Fort Payne	AL	DeKalb	Sand Mtn.	010499991	0.066	6(5)	1(1)	0(0)	0(0)
Fort Wayne	IN	Allen	Amstutz Rd.	180030002	0.069	12(8)	4(3)	1(1)	1(1)
Fort Wayne	IN	Allen	N. Beacon	180030004	0.069	15(8)	5(3)	1(1)	1(1)
Grand Junction	CO	Mesa	Rapid Creek Rd.	080770020	0.067	4(4)	2(2)	0(0)	0(0)
Greenville	NC	Pitt	Pitt Ag. Ctr.	371470006	0.069	16(10)	5(3)	2(1)	1(1)
Greenville-Mauldin-Easley	SC	Greenville	Hillcrest Middle School	450450016	0.067	7(4)	2(1)	1(1)	0(0)
Greenville-Mauldin-	SC	Pickens	Clemson CMS	450770002	0.067	11(10)	4(4)	1(1)	0(0)

CBSA	State	County	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)
Easley									
Gulfport-Biloxi	MS	Harrison	Gulfport Youth Court	280470008	0.069	18(11)	4(2)	2(2)	0(0)
Gulfport-Biloxi	MS	Hancock	Waveland	280450003	0.066	5(3)	1(1)	0(0)	0(0)
Harrison	AR	Newton		051010002	0.067	5(3)	2(2)	1(1)	0(0)
Hobbs	NM	Lew	Hobbs-Jefferson	350250008	0.066	7(5)	0(0)	0(0)	0(0)
Huntington-Ashland	WV-KY-OH	Boyd	Ashland Primary (FIVCO)	210190017	0.069	12(7)	6(3)	1(1)	0(0)
Huntington-Ashland	WV-KY-OH	Greenup	Worthington	210890007	0.069	17(12)	8(6)	3(2)	2(1)
Huntington-Ashland	WV-KY-OH	Cabell	Henderson Center/Marshall University	540110006	0.069	17(10)	8(5)	3(2)	1(1)
Huntington-Ashland	WV-KY-OH	Lawrence	ODOT (Ironton)	390870012	0.068	10(8)	5(4)	1(1)	1(1)
Huntsville	AL	Madison	Huntsville Old Airport	010890014	0.070	17(12)	8(6)	2(2)	0(0)
Jackson	MS	Hinds	Jackson FS19	280490010	0.066	6(4)	4(3)	2(2)	1(1)
Jefferson City	MO	Callaway	New Bloomfield	290270002	0.068	17(15)	4(4)	1(1)	0(0)
Johnstown	PA	Cambria		420210011	0.070	15(9)	5(2)	2(2)	2(2)
Kinston	NC	Lenoir	Lenoir Co. Comm. Coll.	371070004	0.067	6(3)	1(1)	1(1)	0(0)
Lafayette	IN	Carroll	Flora-Flora Airport	180150002	0.069	15(14)	5(4)	1(1)	1(1)
Lafayette	LA	Lafayette	Lafayette / USGS	220550007	0.069	13(6)	5(3)	1(1)	0(0)
Lake Charles	LA	Calcasieu	Vinton	220190009	0.070	10(6)	5(3)	1(1)	0(0)
Lake Charles	LA	Calcasieu	Carlyss	220190002	0.069	15(8)	2(1)	0(0)	0(0)
Lake Charles	LA	Calcasieu	Westlake	220190008	0.067	6(3)	1(1)	1(1)	0(0)
Lakeland	FL	Polk		121056005	0.068	10(5)	3(2)	1(1)	1(1)

CBSA	State	County	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)
Lakeland	FL	Polk	Baptist Childrens' Home	121056006	0.068	10(5)	5(3)	4(3)	2(1)
Logan	UT-ID	Cache	Logan #4	490050004	0.067	7(6)	1(1)	0(0)	0(0)
Madison	WI	Columbia	Columbus	550210015	0.069	15(12)	2(2)	1(1)	1(1)
Madison	WI	Dane	Madison East	550250041	0.069	11(10)	3(3)	2(2)	2(2)
Manchester-Nashua	NH	Hillsborough	Gibson Road	330111011	0.067	4(2)	3(2)	2(1)	2(1)
Manchester-Nashua	NH	Hillsborough	Miller State Park	330115001	0.067	10(6)	3(2)	0(0)	0(0)
Minneapolis-St. Paul-Bloomington	MN	Anoka	Anoka Airport	270031002	0.067	7(4)	4(2)	2(1)	1(1)
Minneapolis	MN	Anoka	Cedar Creek	270031001	0.067	7(4)	2(2)	1(1)	1(1)
Mobile	AL	Mobile	Chickasaw	010970003	0.066	7(4)	2(1)	0(0)	0(0)
Morgantown	WV	Monongalia		540610003	0.068	12(9)	4(2)	1(1)	1(1)
Muncie	IN	Delaware	Albany- Albany Elem. Sch.	180350010	0.068	15(11)	4(4)	2(2)	0(0)
Omaha-Council Bluffs	IA	Harrison	Pisgah	190851101	0.069	15(13)	6(6)	0(0)	0(0)
Omaha-Council Bluffs	IA	Harrison	Woolworth	190850007	0.068	11(10)	4(4)	1(1)	0(0)
Omaha-Council Bluffs	NE	Douglas	n/a	310550019	0.067	8(4)	0(0)	0(0)	0(0)
Panama City-Lynn Haven	FL	Bay	St. Andrews State Park	120050006	0.066	7(5)	0(0)	0(0)	0(0)
Parkersburg-Marietta-Vienna	WV-OH	Washington	Marietta Twp.	391670004	0.069	15(9)	5(4)	2(1)	1(1)
Parkersburg-	WV-OH	Wood	Neale Elementary	541071002	0.068	14(10)	6(5)	3(2)	0(0)

CBSA	State	County	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)
Marietta-Vienna			School						
Pascagoula	MS	Jackson	Pascagoula	280590006	0.070	23(11)	3(2)	0(0)	0(0)
Pensacola-Ferry Pass-Brent	FL	Escambia	n/a	120330018	0.070	21(16)	3(2)	0(0)	0(0)
Pensacola-Ferry Pass-Brent	FL	Santa Rosa	n/a	121130015	0.069	14(12)	6(6)	2(2)	0(0)
Pensacola-Ferry Pass-Brent	FL	Escambia	Ellyson Industrial Park	120330004	0.067	6(5)	1(1)	0(0)	0(0)
Poughkeepsie-Newburgh-Middletown	NY	Dutchess	Millbrook	360270007	0.070	10(5)	7(4)	2(2)	1(1)
Prescott	AZ	Yavapai	Prescott College AQD	040258033	0.069	15(9)	1(1)	0(0)	0(0)
Price	UT	Carbon	n/a	490071003	0.069	15(11)	4(3)	0(0)	0(0)
Quincy	IL-MO	Adams	John Wood Comm. Coll.	170010007	0.068	8(6)	4(4)	2(2)	1(1)
Redding	CA	Shasta	Lassen Volcanic Park	60893003	0.068	7(4)	3(3)	1(1)	0(0)
Redding	CA	Shasta	Shasta Lake	60890009	0.067	7(5)	1(1)	0(0)	0(0)
Reno-Sparks	NV	Washoe	Sparks	320311005	0.068	7(5)	3(2)	1(1)	0(0)
Reno-Sparks	NV	Washoe	South Reno	320310020	0.068	7(5)	1(1)	0(0)	0(0)
Reno-Sparks	NV	Washoe	Lemmon Valley	320312009	0.067	9(6)	3(2)	0(0)	0(0)
Reno-Sparks	NV	Washoe	Reno 3	320310016	0.067	9(6)	1(1)	0(0)	0(0)
Reno-Sparks	NV	Washoe	Toll	320310025	0.066	3(1)	0(0)	0(0)	0(0)
Riverton	WY	Fremont	n/a	560130232	0.066	4(2)	0(0)	0(0)	0(0)

CBSA	State	County	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)
Rock Springs	WY	Sweetwater	Moxa	560370300	0.066	3(2)	0(0)	0(0)	0(0)
Rockford	IL	Winnebago	Maple Elem. Schl.	172012001	0.068	9(7)	3(2)	0(0)	0(0)
Rockland	ME	Knox	Marshall Point Lighthouse	230130004	0.068	8(5)	5(4)	1(1)	0(0)
Rocky Mount	NC	Edgecombe	Leggett	370650099	0.069	13(8)	3(2)	1(1)	1(1)
Santa Fe	NM	Santa Fe	n/a	350490021	0.066	5(3)	0(0)	0(0)	0(0)
Scranton-Wilkes Barre	PA	Luzerne	n/a	420690101	0.070	6(4)	3(3)	1(1)	0(0)
Scranton-Wilkes Barre	PA	Luzerne	n/a	420692006	0.069	6(4)	3(3)	0(0)	0(0)
Show Low	AZ	Navajo	Petrified Forest National Park, South Entrance	040170119	0.070	12(8)	2(2)	0(0)	0(0)
Sioux Falls	SD	Minnehaha	SD School for the Deaf	460990008	0.068	11(10)	1(1)	0(0)	0(0)
Somerset	KY	Pulaski	Somerset	211990003	0.067	7(5)	4(4)	1(1)	0(0)
Syracuse	NY	Onondaga	East Syracuse	360671015	0.069	10(8)	3(2)	1(1)	0(0)
Syracuse	NY	Oswego	Fulton	360750003	0.067	6(4)	3(2)	0(0)	0(0)
Terre Haute	IN	Vigo	Terre Haute CAAP/ McLean High School	181670018	0.067	14(13)	5(4)	1(1)	0(0)
Vallejo-Fairfield	CA	Solano	Ground in shelter	060953003	0.067	10(5)	1(1)	1(1)	0(0)
Victoria	TX	Victoria	Victoria	484690003	0.067	7(6)	2(2)	1(1)	0(0)
Vineland-Millville-Bridgeton	NJ	Cumberland	Millville	340110007	0.070	21(19)	14(12)	7(7)	3(3)
Watertown-Fort Drum	NY	Jefferson	Perch River	360450002	0.070	13(10)	7(5)	2(2)	0(0)

CBSA	State	County	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)
Williamsport	PA	Lycoming	Montoursville	420810100	0.066	8(7)	0(0)	0(0)	0(0)
Worcester	MA	Worcester	Uxbridge	250270024	0.068	10(4)	3(2)	1(1)	1(1)
Worcester	MA	Worcester	Worcester Airport	250270015	0.067	9(4)	3(2)	2(1)	2(1)

Setting the standard at 65 ppb does not eliminate occurrences of 70 ppb, or even multiple occurrences of 70 ppb in a single ozone season. Numerous CBSAs with design values of 65 ppb and below record multiple 8-hour concentrations of 70 ppb or higher in a single ozone season. As illustrated in Table 18 below, Sault Ste. Marie, Michigan, recorded 9 days with 8-hour average ozone concentrations of 70 ppb or higher in a single ozone season yet had a 2011-2013 design value of only 0.064 ppm. Likewise, Florence-Muscle Shoals, Alabama and Gillette, Wyoming recorded 8 days in a single season with 8-hour concentrations of 70 ppb or above yet still attained a standard of 65 ppb. Sault Ste. Marie, Florence-Muscle Shoals, and Seattle, Washington, all recorded multiple days with 8-hour concentrations of 80 ppb or above in a single ozone season while attaining a standard of 65 ppb or lower for 2011-2013.

Not only would a standard of 65 fail to eliminate multiple occurrences of 70 and even 80 ppb during a single year, a standard of 65 ppb would do even less to eliminate instances of multiple exposures to 8-hour ozone concentrations of 60 ppb, contrary to EPA's suggestion.[405] As Table 18 identifies, CBSAs with 2011-2013 design values that would be meeting a standard of 65 ppb (including all CBSAs with design values of exactly 65 ppb) routinely record dozens of days in a season with peak 8-hour concentrations of 60 ppb or above. Gillette, Wyoming had as many as 51 days in a single ozone season on which ozone concentrations were 60 ppb or above; yet Gillette still attained a standard of 65 ppb. Gillette is by no means alone. Evanston, Illinois had 46 days in a single season of 8-hour ozone levels of 60 ppb or higher; Cedar Rapids, Iowa had 40 days; Roanoke, Virginia had 34 days; Winchester VA-WV had 32.

[405] See Exhibit 11.

Table 18: 2011-2013 Ozone Monitor Data for CBSAs with Design Values 0.062 ppm - 0.065 ppm (Monitors with Design Values 0.062 ppm - 0.065 ppm)

CBSA	State	County	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 60+ ppb (max/yr)	Total days 65+ ppb (max/yr)	Total days 70+ ppb (max /yr)	da (n
Cedar Rapids	IA	Linn	Kirkwood	191130028	0.065	57(40)	24(19)	6(6)	1(
Charlottesville	VA	Albemarle	Albemarle High School	391530020	0.065	45(23)	12(6)	3(2)	1(
Columbus	GA-AL	Russell	Ladonia, Phenix City	011130002	0.065	54(27)	21(10)	3(2)	1(
Des Moines-West Des Moines	IA	Warren	Gravel Road in Lake Aquabi State Park	191810022	0.064	43(32)	17(16)	7(7)	
Evanston	WY	Uinta	Murphy Ridge	560410101	0.065	66(46)	16(8)	3(3)	1(
Florence-Muscle Shoals	AL	Colbert	Muscle Shoals	10331002	0.064	41(23)	15(13)	8(8)	2(
Gillette	WY	Campbell	Thunder Basin	560050123	0.064	68(51)	30(27)	8(8)	
Hickory-Lenoir-Morganton	NC	Alexander	Waggin` Trail	370030004	0.065	47(22)	15(6)	6(3)	
Huntington	IN	Huntington	Roanoke- Roanoke Elem. School	180690002	0.065	39(19)	15(9)	6(3)	2(
Jackson	WY-ID	Teton	Yellowstone National Park, Water Tank	560391011	0.065	66(27)	10(6)	3(2)	
Marshall	MN	Lyon	Marshall Airport	270834210	0.065	36(15)	13(6)	6(3)	
Montgomery	AL	Montgomery	MOMS, ADEM	11011002	0.065	54(31)	24(17)	5(5)	
Roanoke	VA	Roanoke	East Vinton Elementary School	511611004	0.064	64(34)	23(12)	6(4)	1(
Rochester	NY	Wayne	Williamson	361173001	0.065	42(25)	25(16)	7(7)	
Santa Barbara-Santa Maria-Goleta	CA	Santa Barbara	Paradise Road	60831014	0.065	43(16)	16(7)	6(3)	2(
Santa Barbara-Santa Maria-Goleta	CA	Santa Barbara	Los Flores Canyon #1	060831025	0.063	20(11)	9(5)	5(3)	2(
Sault Ste. Marie	MI	Chippewa	North of Easterday Avenue	260330901	0.064	24(17)	12(10)	9(9)	8(
Seattle-Tacoma-Bellevue	WA	Warren	Enumclaw-Mud Mtn	530330023	0.062	15(10)	10(7)	6(5)	2(

Sebastian-Vero Beach	FL	Indian River	Indian River Lagoon	120619991	0.065	32(11)	11(4)	1(1)	
Seymour	IN	Jackson	Brownstown- 225 W & 200 N. Water facility	180710001	0.065	30(12)	10(4)	3(1)	1(
Somerset	PA	Somerset	Laurel Hill	421119991	0.065	41(19)	20(10)	4(2)	2(
Summerville	GA	Chattooga	Summerville-DNR Fish Hatchery	130550001	0.065	41(20)	16(9)	5(3)	1(
Tallahassee	FL	Leon	Tallahassee Community College	120730012	0.065	39(19)	17(9)	1(1)	
Wausau	WI	Marathon	Lake Dubay	550730012	0.065	36(19)	13(8)	4(3)	
Winchester	VA-WV	Frederick	Rest	510690010	0.065	51(32)	23(14)	8(6)	1(

EPA's proposal tries to avoid the empirical data that show repeated (and under the proposed form, permissible) 8-hour events of unsafe ozone levels over and above whatever level of ozone that EPA ultimately decides the health studies demonstrate will cause harm. Rather than determining what level of ozone is safe and creating a numerical standard and form that requires the country to achieve that level everywhere, all the time, by eliminating ozone events that exceed that level, EPA introduces the concept of "exposures" and swaps out "events" (of "occurrences") for "exposures." That is, EPA is proposing a standard that ensures that there are repeated, "permissible" 8 hour events of unsafe ozone levels—that is, ozone that is above whatever level EPA ultimately decides is the safe level based on the science--but EPA asserts this is acceptable because regardless if the air is in fact unsafe to breathe, no one will really be outside to be "exposed" to the events.

To this end, EPA explains that "the Administrator places the most weight on estimates of two or more exposures of concern (i.e., as a surrogate for the occurrence of repeated exposures), though she also considers estimates of one or more, particularly for the 70 and 80 ppb benchmarks."[406] In proposing a range of 65 to 70 ppb, EPA notes that selecting 65 would place greater weight on, among other things, "[e]liminating almost all exposures of concern (even single occurrences) at or above 70 and 80 ppb; even in worst-case years and locations," and "almost eliminating the occurrence of two or more exposures of concern at or above 60 ppb."[407] Whereas, EPA notes that selecting a level at or near 70 ppb gives greater weight, among other things, to "[a]lmost eliminating the occurrence of two or more exposures of concern at or above 70 and 80 ppb, even in the worst-case year and location."[408]

As the above tables illustrate, a standard of 65 ppb, for example, would neither eliminate almost all events of ozone levels at or above 70 and 80 ppb, nor eliminate occurrence of two or more events at or above 60 ppb. Indeed, events of 70 and 80 ppb would regularly occur at a nominal standard of 65 using EPA's proposed form, and areas meeting a level of 65 ppb also routinely record numerous concentrations of levels above 60 ppb in a single year.

The only way for EPA to reconcile the empirical data of the number of 8 hour events with unsafe levels of ozone—relative to whatever ultimate numerical standard EPA decides is safe—is to substitute regulation of the amount of air pollution in the air and the number of events of unsafe air with the number of times EPA estimates people will be exposed to that unsafe air. Even if this was not entirely unlawful, as discussed above, the bases for EPA's exposure estimates is technically flawed and based on speculation associated with a small set of diaries as to whether people were or were not engaging in protective behavior when they made entries, and whether the individuals' experiences were reflective of sensitive populations such as outdoor workers or children at outdoor summer camps.

Likewise, EPA's assertion that a level of 70 ppb would "[a]most eliminat[e] the occurrence of two or more exposures of concern at or above 70 and 80 ppb, even in the worst-case year and location," is wholly inconsistent with the empirical data of the number of events,

[406] Proposed Rule at 75,290.
[407] *Id*. at 75,309.
[408] *Id*.

and is based on risk and exposure assessments that are flawed, CBSAs attaining a level of 70 ppb routinely record numerous events of 8-hour concentrations of 70 ppb or higher in a single year (indeed up to 20 such occurrences), and frequently record occurrences of concentrations of 80 or even 85 ppb. And EPA admits that its estimates of exposure could be off by wide margins—as much as 30% for various sensitive populations—and that it does not know to what extent, for example, averting behaviors were engaged in in the dairies it relied upon. Based on the form of the standard, EPA is simply incorrect to suggest that a standard of 70 ppb would "almost eliminate" events of concern at or above 70 and 80 ppb, and it is engaging in inadequately supported estimates in its exposure analysis.

Nor can the extraordinary number of occurrences of concentrations of 70 ppb and above in areas meeting cannot be attributed to the fact that 70 ppb is below the current NAAQS. Replicating the analysis for areas just meeting the current NAAQS—i.e., those CBSAs with maximum 2011-2013 design values of 71 to 75 ppb robustly illustrates this point. Achieving the current standard fails to eliminate occurrences of multiple exposures to levels at and above the NAAQS, as EPA suggests would occur under a standard of 65 or 70 ppb. Nor does it eliminate, or even meaningfully limit, occurrences of multiple exposures to levels 5 ppb below the NAAQS, as EPA suggests would occur under a standard of 65 ppb. The table below starkly illustrates this fact.

As is apparent across CBSAs, attaining the current 75 ppb standard fails to limit multiple exposures in a single ozone season to levels of 75 ppb and does not meaningfully constrain levels of 70 ppb. No CBSA had a 2011-2013 design value between 71 and 75 ppb and did not have multiple 8-hour daily maximum ozone levels of 75 or above in a single year. Indeed, most areas had numerous days with maximum 8-hour ozone levels of 75 or above. For example, Shreveport-Bossier City, LA had 17 in a single season with maximum 8-hour ozone concentrations of 75 ppb or higher and Dover, DE, Adrian, MI, and Las Cruces, NM had up to 16 such days in a season. Table 19 identifies the 25 CBSAs that had at least 10 days in a single ozone season with maximum 8-hour ozone concentrations of 75 ppb or above that nevertheless attained the current 75 ppb ozone standard and therefore are not required to further clean up their emissions. And similar results follow from more recent 3 year design values such as 2012-2014.

Table 19: Maximum days during single ozone season with 8-hour ozone levels of 75 ppb or higher among CBSAs with a 2011-2013 design value between 71 and 75 ppb

Days in single ozone season with 8-hour ozone level of 75 ppb or higher	CBSA
17	Shreveport-Bossier City, LA
16	Dover, DE Adrian, MI Las Cruces, NM
15	Flint, MI Kalamazoo-Portage, MI Traverse City, MI
14	Mount Vernon, IL Lima, OH Indiana, PA

13	Paducah, KY-IL Ann Arbor, MI Grand Rapids-Wyoming, MI Salisbury, NC
12	Indianapolis-Carmel, IN Erie, PA Lancaster, PA Knoxville, TN Beaumont-Port Arthur, TX
11	Springfield, MO Jamestown-Dunkirk-Fredonia, NY
10	Payson, AZ Peoria, IL Bowling Green, KY Cambridge, MD

As Table 22 illustrates, attaining a standard of 75 ppb does not eliminate the occurrence of multiple exceedances in a single season of 8-hour ozone levels 5, 10 and even 15 or more ppb above the standard. Dover, DE and Ann Arbor, MI both have 10 days in a single season with 8-hour ozone levels of 80 ppb or above yet have attained a standard of 75 ppb. Table 20 illustrates the 17 CBSAs attaining a standard of 75 ppb that nevertheless had at least six days in a single ozone season with 8-hour ozone levels of 80 ppb or above. Yet none of these areas are under any obligation to take further steps to reduce their pollution levels any further—and residents of these areas will therefore be repeatedly subjected to events with 8 hour ozone levels above 75 ppb as the status quo going forward--despite the fact that EPA has determined that levels above 75 ppb for more than 8 hours is harmful.

Stated another way, EPA determined in 2008 that the health science indicates that exposures to ozone levels above 75 ppb for longer than 8 hours will result in adverse health impacts. EPA nonetheless created a 2008 NAAQS standard with a nominal numerical level of 75 ppb but with a form—the same form EPA proposes to use again in this proceeding—that ensures numerous 8 hour ozone events far above that level with no further steps required by EPA to reduce that air pollution. And EPA is proposing to do the same thing again in this proceeding.

Table 20: Maximum days during single ozone season with 8-hour ozone levels of 80 ppb or higher among CBSAs with a 2011-2013 design value between 71 and 75 ppb

Days in single ozone season with 8-hour ozone level of 80 ppb or higher	CBSA
10	Dover, DE Ann Arbor, MI
9	Flint, MI Indiana, PA
8	Adrian, MI
7	Paducah, KY-IL Shreveport-Bossier City, LA Grand Rapids-Wyoming, MI

	Traverse City, MI Knoxville, TN Beaumont-Port Arthur, TX
6	Mount Vernon, IL Indianapolis-Carmel, IN Baton Rouge, LA Erie, PA Lancaster, PA

Multiple single season events in areas meeting the current standard of 75 ppb are not limited to levels of 80 ppb and up. Eight CBSAs had four or more days in a single ozone season with maximum 8-hour ozone levels of 85 ppb or above: Dover, DE (7 days); Paducah, KY-IL and Adrian, MI (6 days); Shreveport-Bossier City, LA (5 days); Mount Vernon, IL, Ann Arbor, MI. Flint, MI, and Green Bay, WI (4 days). And 11 CBSA had multiple days in a single ozone season with maximum 8-hour ozone levels of 90 ppb or above, yet still attained the current 75 ppb NAAQS during 2011 to 2013. These areas were Paducah, KY-IL, Morristown, TN, Beaumont-Port Arthur, TX, and Charleston, WV (3 days of 90 ppb or above in a single ozone season); and Dover, DE, Adrian, MI, Flint, MI, Kalamazoo-Portage, MI, Altoona, PA, Richmond, VA, and Green Bay, WI (2 days of 90 ppb or above in a single ozone season). CBSAs attaining a standard of 75 ppb even had days during the ozone season where maximum 8-hour ozone concentrations were as high as 111[409] and 112 ppb.[410] These levels are 36 and 37 ppb above the level of the NAAQS.

Finally, although EPA contends that setting a NAAQS of 65 ppb will largely eliminate exposures of even 60 ppb, this is flatly contradicted by empirical data for areas meeting a standard of 75 ppb that show repeated events of 70 ppb and above. EPA must then rely on its flawed exposure analysis to try to reconcile the difference between how safe the air needs to be and how safe EPA's proposed standard would actually make it. EPA is saying that meeting a standard of 65 ppb will largely eliminate exposures to levels 5 ppb lower. But for CBSAs with a design value between 71 and 75 ppb—i.e., those areas meeting a standard of 75 ppb—there can be dozens of days on which maximum 8-hour ozone concentrations are 70 ppb or higher. At least five CBSAs had more than 30 days in a single ozone season during which maximum 8-hour concentrations were 70 ppb or above, and an additional 26 CBSAs with design values between 71 and 75 ppb had 20 or more days during a single season on which 8-hour ozone concentrations were 70 ppb or above. Table 21 below characterizes these areas.

Table 21: Maximum days during single ozone season with 8-hour ozone levels of 70 ppb or higher among CBSAs with a 2011-2013 design value between 71 and 75 ppb

Days in single ozone season with 8-hour ozone level of 70 ppb or higher	CBSA
36	Knoxville, TN
35	Las Cruces, NM
33	Payson, AZ

[409] Salisbury, NC 8-hour daily maximum in 2012; Morristown, TN 8-hour daily maximum in 2012.
[410] Beaumont-Port Arthur, TX 8-hour daily maximum in 2012.

	Shreveport-Bossier City, LA
31	Adrian, MI
28	Chico, CA Vicennes, IN
27	Mount Vernon, IL
26	Peducah, KY-IL
25	Indianapolis-Carmel, IN Topeka, KS Traverse City, MI Lima, OH McAlester, OK Ogden-Clearfield, UT
23	Springfield, OH
22	Bloomington-Normal, IN Bishop, CA Baton Rouge, LA
21	Dover, DE Ann Arbor, MI Grand Rapids-Wyoming, MI Kalamazoo-Portage, MI Springfield, MO Albuquerque, NM Salisbury, NC Tyler, TX
20	Lexington-Fayette, KY Cambridge, MD Beaumont-Port Arthur, TX Waco, TX

Nor does the fact that the 75 ppb 2008 NAAQS is not fully attained diminish the import of these empirical data. Again, areas presently meeting the 75 ppb NAAQS are not required to further improve their air quality under the present standard. Moreover, it is worth noting that NAAQS are not attained overnight. Eighteen years out from the 1997 NAAQS, this standard is still not fully attained, even in areas that were supposed to attain the NAAQS much earlier based on the timelines set forth in the Clean Air Act.

Table 22: 2011-2013 Ozone Monitor Data for CBSAs with Design Value 0.071 to 0.075 ppm (Monitors with Design Value 0.071 to 0.075 ppm)

State	County	CBSA	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max /yr)	Total days 85+ ppb (max /yr)	Total days 90+ ppb (max /yr)
AZ	Coconino	Flagstaff, AZ	Grand Canyon National Park, The Abyss	040058001	0.072	26 (15)	7 (3)	1	0	0
AZ	Gila	Payson, AZ	TONTO NM	040070010	0.075	43 (33)	17 (10)	3 (1)	0	0
AZ	Cochise	Sierra Vista-Douglas, AZ	CHIRICAHUA NATIONAL MONUMENT	040038001	0.073	32 (15)	6 (4)	0	0	0
AZ	Pima	Tucson, AZ	SAGUARO PARK	040190021	0.073	24 (13)	6 (4)	1	0	0
AZ	Pima	Tucson, AZ	FAIRGROUNDS	040191020	0.071	14 (8)	2 (1)	0	0	0
AR	Washington	Fayetteville-Springdale-Rogers, AR-MO	SPRINGDALE	051430005	0.072	25 (17)	9 (5)	4 (2)	0	0
CA	Inyo	Bishop, CA	DEATH VALLEY NATIONAL MONUMENT NEAR NEVARES SPRINGS ACCESS ROAD	060270101	0.072	35 (22)	8 (6)	0	0	0
CA	Butte	Chico, CA	TEMPORARY STATION FOR SPECIAL STUDY OF O3 TRANSPORT	060070007	0.075	55 (28)	19 (9)	5 (2)	1	1
CA	Tuolumne	Phoenix Lake-Cedar Ridge, CA	251 S BARRETTA, SONORA, CA 95370	061090005	0.073	39 (19)	6 (4)	1	0	0
CA	Tehama	Red Bluff, CA	Old Fire Lookout on top of Tucson Butte	061030004	0.074	42 (18)	13 (6)	3 (2)	0	0
CA	Tehama	Red Bluff, CA	RED BLUFF-TEHAMA COUNTY SHERIFF'S OFFICE	061030005	0.072	18 (8)	6 (4)	1	1	0
CA	Alameda	San Francisco-Oakland-Fremont, CA	Livermore – Rincon	060010007	0.071	17 (9)	8 (3)	3 (2)	1	1
CO	El Paso	Colorado Springs, CO	U.S. AIR FORCE ACADEMY	080410013	0.074	26 (12)	4 (2)	2 (1)	0	0
CO	El Paso	Colorado Springs, CO	MANITOU SPRINGS	080410016	0.074	27 (15)	5 (2)	1	0	0
CO	La Plata	Durango, CO		080671004	0.072	18 (15)	7 (7)	1	0	0

State	County	CBSA	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)	Total days 90+ ppb (max/yr)
DE	Kent	Dover, DE	PROPERTY OF KILLENS POND STATE PARK; BEHIND FARM BUILDINGS	100010002	0.074	30 (21)	19 (16)	12 (10)	9 (7)	2 (2)
FL	Sarasota	Bradenton-Sarasota-Venice, FL		121151005	0.071	14 (7)	6 (4)	1	0	0
FL	Orange	Orlando-Kissimmee, FL	WINTER PARK	120952002	0.071	14 (8)	7 (5)	1	0	0
FL	Hillsborough	Tampa-St. Petersburg-Clearwater, FL		120570081	0.071	15 (10)	6 (4)	1	0	0
GA	Bibb	Macon, GA	Macon SE	130210012	0.071	25 (17)	11 (8)	5 (3)	1	0
IL	McLean	Bloomington-Normal, IL	ISU HARRIS PHYSICAL PLANT	171132003	0.072	26 (22)	9 (7)	3 (2)	0	0
IL	Champaign	Champaign-Urbana, IL	BOOKER T. WASHINGTON ES	170190007	0.071	13 (7)	8 (4)	4 (3)	1	0
IL	Macon	Decatur, IL	IEPA TRAILER	171150013	0.071	16 (11)	5 (4)	0	0	0
IL	Hamilton	Mount Vernon, IL	TEN MILE CREEK DNR OFFICE	170650002	0.074	34 (27)	17 (14)	6 (6)	4 (4)	1
IL	Peoria	Peoria, IL	PEORIA HEIGHTS HS	171431001	0.071	25 (22)	11 (10)	3 (2)	1	0
IL	Sangamon	Springfield, IL	Illinois Building State Fairgrounds	171670014	0.072	21 (15)	11 (7)	3 (2)	1	0
IN	Shelby	Indianapolis-Carmel, IN	TRITON Middle SCHOOL, NORTH OF FAIRLAND	181450001	0.075	28 (19)	16 (12)	7 (5)	2 (2)	0
IN	Marion	Indianapolis-Carmel, IN	Indpls.- E. 16th St.	180970073	0.074	39 (25)	15 (10)	7 (6)	2 (2)	1
IN	Boone	Indianapolis-Carmel, IN	Perry Worth ELEMENTRY SCHOOL, WEST OF WHITESTOWN	180110001	0.073	23 (17)	11 (9)	5 (4)	2 (1)	1
IN	Marion	Indianapolis-Carmel, IN	Indpls- Washington Park/ in parking lot next to police station	180970078	0.072	24 (14)	10 (7)	3 (3)	1	0
IN	Knox	Vincennes, IN	Vincennes	180839991	0.073	35 (28)	13 (9)	6 (3)	2 (2)	0
KS	Shawnee	Topeka, KS	KNI	201770013	0.073	39 (25)	14 (9)	3 (3)	1	0
KY	Edmonson	Bowling Green, KY	Mammoth Cave National Park, Houchin Meadow	210610501	0.071	22 (17)	10 (10)	4 (4)	1	1
KY	Fayette	Lexington-Fayette, KY	LEXINGTON PRIMARY	210670012	0.071	26 (20)	13 (9)	3 (2)	1	0

State	County	CBSA	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)	Total days 90+ ppb (max/yr)
KY	Livingston	Paducah, KY-IL	SMITHLAND	211390003	0.074	31 (26)	15 (13)	7 (7)	5 (5)	2 (2)
KY	McCracken	Paducah, KY-IL	JACKSON PURCHASE (PADUCAH PRIMARY)	211451024	0.074	32 (26)	19 (16)	10 (9)	7 (6)	3 (3)
LA	East Baton Rouge	Baton Rouge, LA	LSU	220330003	0.075	35 (22)	11 (6)	7(4)	4 (2)	2 (1)
LA	Iberville	Baton Rouge, LA	Carville	220470012	0.075	25 (16)	10 (7)	7 (6)	2 (2)	0
LA	Pointe Coupee	Baton Rouge, LA	New Roads	220770001	0.074	26 (13)	14 (8)	3 (2)	2 (1)	0
LA	East Baton Rouge	Baton Rouge, LA	Capitol	220330009	0.072	23 (14)	8 (6)	5 (4)	4 (3)	1
LA	Livingston	Baton Rouge, LA	French Settlement	220630002	0.072	24 (15)	6 (4)	2 (1)	1	0
LA	Ascension	Baton Rouge, LA	Dutchtown	220050004	0.071	20 (13)	8 (7)	4 (4)	3 (3)	0
LA	Iberville	Baton Rouge, LA	Bayou Plaquemine	220470009	0.071	28 (14)	11 (7)	3 (3)	1	0
LA	Lafourche	Houma-Bayou Cane-Thibodaux, LA	Thibodaux	220570004	0.071	15 (8)	8 (5)	2 (1)	0	0
LA	St. John the Baptist	New Orleans-Metairie-Kenner, LA	Garyville	220950002	0.072	26 (14)	13 (7)	1	1	0
LA	St. Tammany	New Orleans-Metairie-Kenner, LA	Madisonville	221030002	0.072	23 (15)	11 (7)	3 (2)	1	0
LA	Bossier	Shreveport-Bossier City, LA	Shreveport / Airport	220150008	0.074	40 (33)	19 (17)	8 (7)	5 (5)	0
LA	Caddo	Shreveport-Bossier City, LA	Dixie	220170001	0.073	29 (23)	10 (7)	4 (4)	0	0
ME	York	Portland-South Portland-Biddeford, ME	KPW - Kennebunkport Parson'd Way	230312002	0.075	16 (7)	10 (4)	4 (2)	1	0
MD	Dorchester	Cambridge, MD	Blackwater NWR	240199991	0.075	31 (20)	14 (10)	6 (4)	2 (1)	0
MD	Washington	Hagerstown-	Hagerstown	240430009	0.071	22 (12)	7 (3)	3 (2)	1	0

State	County	CBSA	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)	Total days 90+ ppb (max/yr)
		Martinsburg, MD-WV								
MA	Barnstable	Barnstable Town, MA	TRURO NATIONAL SEASHORE	250010002	0.072	15 (8)	8 (6)	3 (3)	2 (2)	1
MA	Norfolk	Boston-Cambridge-Quincy, MA-NH	BLUE HILL OBSERVATORY	250213003	0.072	17 (8)	6 (3)	1	0	0
MA	Essex	Boston-Cambridge-Quincy, MA-NH	LYNN WATER TREATMENT PLANT	250092006	0.071	17 (7)	6 (2)	2 (1)	2 (1)	1
MA	Hampden	Springfield, MA	WESTOVER AFB	250130008	0.073	20 (11)	7 (3)	3 (1)	0	0
MI	Lenawee	Adrian, MI	6792 RAISIN CENTER HWY, LENAWEE CO.RD.COMM.OWNER, TECUMSEH	260910007	0.075	45 (31)	22 (16)	10 (8)	7 (6)	2 (2)
MI	Washtenaw	Ann Arbor, MI	TOWNER ST, SOUTH; 2 LANE RESIDENIAL – HOSPITAL	261610008	0.075	31 (21)	18 (13)	11 (10)	5 (4)	1
MI	Genesee	Flint, MI		260490021	0.074	26 (19)	17 (15)	10 (9)	4 (4)	2 (2)
MI	Genesee	Flint, MI	Otisville	260492001	0.074	26 (19)	14 (11)	6 (5)	4 (3)	1
MI	Kent	Grand Rapids-Wyoming, MI	GR-Monroe	260810020	0.074	27 (19)	17 (12)	8 (7)	1	0
MI	Kent	Grand Rapids-Wyoming, MI	APPROXIMATELY 1/4 MILE SOUTH OF 14 MILE RD	260810022	0.074	28 (21)	18 (13)	5 (3)	1	0
MI	Kalamazoo	Kalamazoo-Portage, MI	KALAMAZOO FAIRGROUNDS	260770008	0.075	30 (21)	19 (15)	8 (7)	3 (3)	2 (2)
MI	Ingham	Lansing-East Lansing, MI		260650012	0.072	21 (16)	10 (9)	5 (5)	0	0
MI	Clinton	Lansing-East Lansing, MI	ROSE LAKE, STOLL RD.(8562 E.)	260370001	0.071	19 (13)	11 (9)	3 (3)	3 (3)	1
MI	Benzie	Traverse City, MI		260190003	0.074	34 (25)	18 (15)	9 (7)	0	0
MS	Bolivar	Cleveland, MS	Cleveland	280110001	0.071	16 (9)	7 (4)	0	0	0
MO	Greene	Springfield, MO	Fellows Lake	290770042	0.072	31 (21)	13 (11)	4 (4)	0	0
MO	Andrew	St. Joseph, MO-KS	Savannah	290030001	0.073	32 (19)	14 (7)	6 (5)	1	0
NJ	Atlantic	Atlantic City, NJ	Brigantine	340010006	0.073	20 (11)	8 (4)	5 (3)	3 (2)	0

State	County	CBSA	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)	Total days 90+ ppb (max/yr)
NM	Bernalillo	Albuquerque, NM	Foothills	350011012	0.072	40 (21)	9 (5)	3 (2)	0	0
NM	Bernalillo	Albuquerque, NM	WESTSIDE TAYLOR RANCH	350010027	0.071	25 (12)	4 (2)	0	0	0
NM	Eddy	Carlsbad-Artesia, NM	5ZR ON BLM LAND BORDERING RESIDENTIAL AREA OUTSIDE CARLSBAD CITY LIM	350151005	0.071	15 (8)	4 (2)	0	0	0
NM	San Juan	Farmington, NM		350450018	0.071	24 (12)	3 (3)	1	0	0
NM	Dona Ana	Las Cruces, NM	6ZN US-MEXICO BORDER CROSSING. BOTH SIDES UNINHABITED AS OF 1996.	350130022	0.075	52 (35)	22 (16)	6 (4)	1	0
NM	Dona Ana	Las Cruces, NM	6ZM 2MI FROM MT CRISTO REY WHERE NM, TEX, AND MEXICO JOIN TOGETHER	350130021	0.072	22 (8)	7 (4)	2 (2)	0	0
NY	Niagara	Buffalo-Niagara Falls, NY	MIDDLEPORT	360631006	0.073	22 (16)	9 (7)	5 (4)	2 (1)	1
NY	Erie	Buffalo-Niagara Falls, NY	AMHERST	360290002	0.072	21 (14)	12 (8)	3 (3)	1	0
NY	Chautauqua	Jamestown-Dunkirk-Fredonia, NY	DUNKIRK	360130006	0.072	19 (14)	15 (11)	5 (4)	3 (2)	1
NC	Guilford	Greensboro-High Point, NC	Mendenhall School	370810013	0.072	26 (15)	11 (7)	5 (3)	1	0
NC	Lincoln	Lincolnton, NC	Crouse	371090004	0.072	25 (13)	10 (6)	1	1	0
NC	Wake	Raleigh-Cary, NC	Fuquay-Varina	371830016	0.071	27 (19)	11 (8)	3 (2)	2 (2)	1
NC	Rowan	Salisbury, NC	Rockwell	371590021	0.073	35 (21)	21 (13)	5 (4)	2 (1)	1
NC	Rowan	Salisbury, NC	Enochville School	371590022	0.072	28 (17)	16 (8)	5 (3)	3 (2)	1
NC	Forsyth	Winston-Salem, NC		370670022	0.073	28 (14)	13 (7)	5 (3)	2 (2)	0
OH	Ashtabula	Ashtabula, OH	CONNEAUT	390071001	0.075	26 (11)	12 (6)	5 (3)	4 (2)	2
OH	Allen	Lima, OH	LIMA BATH	390030009	0.073	38 (25)	17 (14)	3 (3)	1	1
OH	Knox	Mount Vernon, OH	CENTERBURG	390830002	0.073	22 (12)	6 (4)	4 (3)	1	1

State	County	CBSA	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)	Total days 90+ ppb (max/yr)
OH	Clark	Springfield, OH	SPRINGFIELD WELL FIELD	390230001	0.075	43 (23)	14 (7)	4 (3)	1	1
OH	Clark	Springfield, OH	MUD RUN	390230003	0.073	34 (20)	11 (6)	3 (2)	1	1
OH	Lucas	Toledo, OH	LOW_SER	390950034	0.073	22 (17)	15 (6)	5 (2)	2 (2)	1
OH	Wood	Toledo, OH	BOWLING GREEN	391730003	0.071	26 (15)	9 (7)	3 (3)	1	1
OH	Fayette	Washington Court House, OH, AR	Deer Creek	390479991	0.072	21 (10)	8 (4)	4 (2)	1	1
OH	Jefferson	Weirton-Steubenville, WV-OH	STEUBEN	390810017	0.071	22 (17)	9 (6)	3 (2)	0	0
OK	Sequoyah	Fort Smith, AR-OK		401359021	0.072	30 (19)	10 (8)	3 (3)	2 (2)	0
OK	Pittsburg	McAlester, OK	McALESTER MUNICIPAL AIRPORT	401210415	0.075	41 (25)	13 (8)	4 (2)	1	0
OK	Cherokee	Tahlequah, OK	TAHLEQUAH SHELTER	400219002	0.074	37 (18)	16 (8)	5 (4)	0	0
PA	Blair	Altoona, PA		420130801	0.073	22 (14)	12 (9)	5 (4)	4 (3)	2 (2)
PA	Clearfield	DuBois, PA	MOSHANNON STATE FOREST	420334000	0.071	13 (7)	2 (2)	2 (1)	1	1
PA	Erie	Erie, PA		420490003	0.074	26(18)	15(12)	8(6)	3(3)	1
PA	Dauphin	Harrisburg-Carlisle, PA	A420431100LAT/LON POINT IS AT CORNER OF TRAILER	420431100	0.074	21(11)	9(5)	3(3)	1	0
PA	Indiana	Indiana, PA		420630004	0.075	31(17)	19(14)	13(9)	3(2)	1
PA	Lancaster	Lancaster, PA	A420710007LAT/LON POINT AT CORNER OF TRAILER	420710007	0.075	29(16)	20(12)	9(6)	3(2)	1
PA	Lancaster	Lancaster, PA	Lancaster DW	420710012	0.075	27(14)	16(10)	8(5)	5(4)	1
PA	Lawrence	New Castle, PA		420730015	0.073	18(11)	8(5)	5(4)	2(2)	0
PA	Berks	Reading, PA	Reading Airport	420110011	0.073	24(12)	10(5)	3(2)	0	0
PA	Centre	State College, PA	Penn State	420279991	0.072	15(10)	10(6)	4(2)	2(2)	0
PA	York	York-Hanover, PA	York DW	421330011	0.074	28(16)	12(7)	6(3)	1	0
PA	York	York-Hanover, PA	A421330008LAT/LON POINT AT CORNER OF TRAILER	421330008	0.072	23(13)	9(6)	3(2)	1	0

State	County	CBSA	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)	Total days 90+ ppb (max/yr)
SC	Spartanburg	Spartanburg, SC	NORTH SPARTANBURG FIRE STATION #2 (Shady Grove)	450830009	0.072	19(14)	8(6)	5(4)	2(1)	0
TN	Sullivan	Kingsport-Bristol-Bristol, TN-VA	Blountville Ozone Monitor	471632002	0.071	18(11)	6(4)	3(3)	2(2)	1
TN	Blount	Knoxville, TN	Great Smoky Mountains National Park, Look Rock	470090101	0.074	45(36)	16(12)	7(7)	3(3)	0
TN	Jefferson	Morristown, TN	New Market ozone monitor	470890002	0.073	23(12)	8(5)	7(4)	3(3)	3(3)
TN	Sevier	Sevierville, TN		471550101	0.072	23(18)	11(9)	3(2)	1	0
TX	Travis	Austin-Round Rock, TX	Austin Audubon Society	484530020	0.073	17(8)	5(4)	1	0	0
TX	Travis	Austin-Round Rock, TX	Austin Northwest	484530014	0.072	25(17)	8(4)	4(3)	0	0
TX	Jefferson	Beaumont-Port Arthur, TX	SETRPC 40 Sabine Pass	482450101	0.075	33(20)	18(12)	9(7)	5(3)	5(3)
TX	Jefferson	Beaumont-Port Arthur, TX	Beaumont Downtown	482450009	0.072	19(12)	11(6)	2(1)	0	0
TX	Jefferson	Beaumont-Port Arthur, TX	Hamshire	482450022	0.071	23(15)	8(4)	2(1)	1	1
TX	Navarro	Corsicana, TX	Corsicana Airport	483491051	0.072	28(18)	8(3)	2(1)	1	0
TX	El Paso	El Paso, TX	El Paso UTEP	481410037	0.072	26(11)	7(3)	1	0	0
TX	Bell	Killeen-Temple-Fort Hood, TX	Killeen Skylark Field	480271047	0.074	30(15)	11(5)	2(1)	1	0
TX	Harrison	Marshall, TX	Karnack	482030002	0.072	30(19)	9(7)	2(1)	0	0
TX	Smith	Tyler, TX	Tyler Airport Relocated	484230007	0.075	36(21)	11(5)	4(3)	1	0
TX	McLennan	Waco, TX	Waco Mazanec	483091037	0.074	29(20)	11(7)	4(2)	1	0
UT	Weber	Ogden-Clearfield, UT	Harrisville	490571003	0.074	45(25)	11(7)	2(1)	1	0
UT	Weber	Ogden-Clearfield, UT	Ogden	490570002	0.072	18(11)	7(4)	2(1)	1	0
UT	Utah	Provo-Orem, UT	North Provo	490490002	0.073	21(11)	9(5)	2(1)	1	0

State	County	CBSA	Monitor Site Name	Monitor Site ID	Monitor Design Value (ppm)	Total days 70+ ppb (max/yr)	Total days 75+ ppb (max/yr)	Total days 80+ ppb (max/yr)	Total days 85+ ppb (max/yr)	Total days 90+ ppb (max/yr)
UT	Washington	St. George, UT	Zion National Park, Dalton's Wash	490530130	0.072	23(12)	5(5)	1	0	0
VA	Charles	Richmond, VA	Shirley Plantation	510360002	0.073	22(11)	13(6)	8(4)	5(3)	3(2)
VA	Henrico	Richmond, VA	MathScience Innovation Center	510870014	0.073	26(15)	13(8)	5(3)	4(3)	2(2)
VA	Hanover	Richmond, VA	Turner Property, Old Church	510850003	0.072	20(11)	9(4)	5(3)	2(2)	1
VA	Caroline	Richmond, VA	USGS Geomagnetic Center, Corbin	510330001	0.071	15(10)	6(4)	2(1)	0	0
VA	Hampton City	Virginia Beach-Norfolk-Newport News, VA-NC	NASA Langley Research Center	516500008	0.072	17(9)	7(4)	1	1	1
WV	Kanawha	Charleston, WV	CHARLESTON BAPTIST TEMPLE/SITE MOVED FROM OAQ AND FIRE STATION	540390010	0.073	30(17)	10(7)	5(4)	3(3)	3(3)
WV	Hancock	Weirton-Steubenville, WV-OH		540291004	0.072	26(16)	9(5)	5(3)	0	0
WI	Outagamie	Appleton, WI	APPLETON AAL	550870009	0.072	20(13)	8(6)	3(3)	1	1
WI	Dodge	Beaver Dam, WI	Horicon Wildlife Area	550270001	0.072	24(16)	10(8)	4(3)	1	1
WI	Fond du Lac	Fond du Lac, WI	FOND DU LAC	550390006	0.072	22(15)	9(7)	4(3)	1	1
WI	Kewaunee	Green Bay, WI	JUMBOS DRIVE-IN PROPERTY, SOUTH END OF KEWAUNEE, 250' EAST OF HWY 42	550610002	0.074	20(14)	13(11)	6(5)	5(4)	3(2)
WI	Walworth	Whitewater, WI	LAKE GENEVA	551270005	0.071	20(15)	8(7)	3(3)	2(2)	1

As discussed in above, given that it is clear that numerous days will have 8-hour average ozone concentrations at and well above the level of NAAQS in a single ozone season, EPA cannot rely on assuming that people, and especially children, will spend their time indoors to justify setting a higher ozone standard. EPA needs to ensure that the air is safe to breathe even—and especially—if people elect to spend time outdoors. This is what the Clean Air Act promises. And this is what EPA should be encouraging. Limiting children's opportunities to play outside by setting an unprotective NAAQS is unlawful and inconsistent with EPA's duty under the Clean Air Act.

d. EPA's Purported Justification for Its Preferred Form Is Arbitrary and Capricious

EPA's claim that its preferred form somehow provides "stability" that enhances implementation is irrational. See Proposed Rule at 75,294/3-95/1. EPA appears to be concerned that meteorological conditions vary from year to year and "could have the effect of reducing public health protection, to the extent they result in frequent shifts in and out of attainment." Id. at 75,294/2-3.

EPA fails to explain why the 4th highest level will be any more stable than the 3rd, 2nd, or 1st highest levels. Although EPA purports to justify its decision to use the 4th highest form of the standard on the need for administrative stability, this need can be almost fully accomplished through use of a 1st or 2nd highest 3-year average. To evaluate EPA's purported justification, we calculated "design values" for every CBSA for each 3-year period beginning with 2000-2002 and ending with 2012-2014. The "design value" was calculated in three different ways: (a) using the 3-year average of 4th highest 8-hour daily maximums; (b) using the 3-year average of 1st highest 8-hour daily maximums; and (c) using the 3-year average of 2nd highest 8-hour daily maximums. We then calculated the year-to-year absolute value difference between design values using each of these three forms. The data are provided in Exhibit 12. The results show that the stability benefit of using a 4th highest as opposed to a 1st or 2nd highest form is marginal at best. Based on the data from 2000 to 2014, the average year-to-year change in design value using a 1st highest design value form is 3.2 ppb, as compared with 2.8 ppb for a 2nd and 2.5 ppb for a 4th highest form. These very modest differences in year-to-year design value changes fail to support EPA's purported administrative stability rationale.

EPA also fails to explain how "frequent shifts in and out of attainment" would reduce public health protection. Provided a nonattainment area retains its nonattainment designation (and does not seek a "clean data determination" (which is itself unlawful and arbitrary)), all control requirements remain in place even if that area has unusually low ozone levels. Thus, there would be no reduction in public health protection. For the other circumstance—an attainment area that shifts into nonattainment—EPA suggests no basis for concluding that there would somehow be less public health protection in effect. To the contrary, there would be a greater need for public health protection.

Finally, the statute and EPA's past practice belie its explanation. EPA cannot redesignate a nonattainment area as attainment based on the area's happening to have an unusually good

year. 42 U.S.C. § 7407(d)(3)(E) (conditioning redesignation to attainment on, inter alia, "the improvement in air quality [being] due to permanent and enforceable reductions in emissions"). EPA's unlawful and arbitrary clean data determinations must themselves be revoked when an area's air quality deteriorates. E.g., 40 C.F.R. § 51.918. And, in the other direction, EPA arbitrarily and unlawfully virtually never redesignates attainment areas as nonattainment even when they consistently violate the ozone NAAQS. Thus, the form of the standard does not augment programmatic "stability."

C. The Proposal Fails to Fully and Rationally Explain Reasons for Differing from CASAC's Advice

Under the Act, EPA's proposal must "set forth or summarize and provide a reference to any pertinent findings, recommendations, and comments by [CASAC]," and "if the proposal differs in any important respect from any of these recommendations," must set forth "an explanation of the reasons for such differences." 42 U.S.C. § 7607(d)(3). Thus, EPA "must fully explain" its reasons for any departure from CASAC's recommendation. *Mississippi v. EPA*. 744 F.3d 1334, 1354 (D.C. Cir. 2013). EPA must also provide such an explanation in the final rule. *Id*. 1355.

Further, "EPA must be precise in describing the basis for its disagreement with CASAC. If EPA's quarrel is with CASAC's scientific analysis, then in order to preserve the integrity of CASAC's scientific role, EPA must give a sound scientific reason for its disagreement." *Id*.

Here, EPA's proposal fails to follow CASAC's recommendations in a number of important respects, as further detailed below. In each case, the proposal does not meet the above-described requirements for providing a full and rational explanation for such failure.

1. Primary Standard

a. Identification of Adverse Effects

CASAC expressly stated its view that "estimation of FEV1 decrements of ≥15% is appropriate as a scientifically relevant surrogate for adverse health outcomes in active healthy adults, whereas an FEV1 decrement of ≥10% is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease."[411] CASAC further found that lung function decrements greater than or equal to 15% in children "have been shown to result in significant adverse effects."[412]

EPA's proposal fails to follow the foregoing advice from CASAC regarding what constitutes effects that are adverse, and fails to rationally explain why. Although EPA asserts that it considers the occurrence of ozone-induced FEV1 decrements ≥ 10 and 15% as surrogates for

[411] CASAC Letter 2014a at 3.

[412] *Id.* at 7; *see also id.* at 4 (stating that "[a]t the level of the current standard, 11% to 22% of school age children are predicted to experience at least one day with an FEV1 decrement ≥10%, which is not protective of public health."); *id.* at 7 (citing "findings of adverse effects, including clinically significant lung function decrements and airway inflammation, after exposures to 60 ppb ozone in healthy adults").).

occurrence of adverse health outcomes,[413] its proposed decision arbitrarily refuses to treat such decrements as adverse. In actually deciding on options for the level of the standard, EPA repeatedly indicates that, as a practical matter, it does not consider adverse effects to be adequately shown unless: a) there is a finding of *both* lung function decrements and statistically significant increases in respiratory symptoms (compared to filtered air) in order to show adversity; or b) a showing of repeated occurrences of FEV decrements $\geq$ 10 and 15%.[414] Neither of these tests is consistent with CASAC's recommendations. Nowhere did CASAC state that such lung function decrements needed to be accompanied by statistically significant respiratory symptoms in order for adverse effects to be shown. Further, as shown above, CASAC made clear that individual instances of lung function decrements $\geq$ 10 and 15% are adequate surrogates by themselves for adverse effects: CASAC did not advise that multiple occurrences of such decrements were necessary to show adversity.[415]

EPA fails to explain or rationally justify its employment of more demanding tests for adverse effects than recommended or employed by CASAC. The agency asserts that the ATS has identified the combination of lung function decrements and respiratory symptoms as adverse, but that is only one of a number of grounds established by ATS for identifying effects as adverse, and ATS has also identified adverse effects criteria that are clearly met in this case at levels of 70 ppb and lower. For example, ATS has also identified as adverse "medically significant physiologic changes generally evidenced by" (among other things), use of medicine and "[i]nterference with the normal activity of the affected person or persons,"[416] while EPA's 2006 Criteria Document (Table 8-3, p. 8-68) makes clear that for people with lung FEV_1 decrements $\geq$ 10% but < 20% would likely interfere with normal activities for many individuals, and would likely result in more frequent medication use. Likewise, in the 2008 ozone NAAQS, EPA itself said that a lung function decrement of $\geq$ 10% "represent[s] a level that should be considered

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[413] Proposed Rule at at 75,306/1.

[414] *E.g. id*. at 75,304/3, 75,305/1, 75,306/2. EPA's proposal is at best equivocal even as to whether such repeated decrements qualify as adverse. The agency vaguely asserts that it "considers the extent to which a standard …would be expected to protect the population from experiencing O3-induced FEV1 decrements $\geq$10% and $\geq$15% …", and that multiple such exposures "may" be considered to be adverse. *Id*. 75,306/2. Moreover, in describing the grounds for its proposed decisions (e.g., selection of the range of 65-70 ppb), EPA focuses on the combination of lung decrements and statistically significant respiratory symptoms as the benchmark for adverse effects, without treating lung decrements $\geq$10% or $\geq$15% as also determinative of adverse effects. *E.g.*, *id*. at 75,304/2-3, 75,308/3-75,309/1, 75,309/3. That is not consistent with CASAC's advice. Rather, CASAC made clear its view that FEV1 decrements $\geq$10% were scientifically relevant surrogates for adverse effects in persons with asthma and lung disease and decrements $\geq$15% were surrogates for adverse effects in healthy individuals. It did not state or suggest that such decrements merely "may" be considered adverse. CASAC further expressly described such effects as adverse or the equivalent. CASAC Letter 2014a at 4 (ozone producing an FEV1 decrement $\geq$10% in children is not protective of public health."); *id*. at 7 (describing as "adverse effects" clinically significant lung function decrements and airway inflammation); *id*. (finding that lung function decrements greater than or equal to 15% in children "have been shown to result in significant adverse effects"). EPA makes no attempt to explain why CASAC's position on this score is wrong.

[415] CASAC Letter 2014a at 3, 4, 7. To the extent EPA is suggesting that in determining adversity it is merely giving less weight to effects that do not meet its more demanding tests, that approach is also contrary to CASAC's advice. CASAC nowhere qualified its advice that FEV1 decrements $\geq$10% and $\geq$15% are adequate surrogates for adversity, nor did CASAC suggest that determination of adversity involved some sort of balancing or weighing of impacts. To the contrary, as noted above, CASAC expressly found that single occurrences of such decrements showed adverse effects.

[416] Proposed Rule at 75,263/1.

adverse for asthmatic individuals."[417] And in 2011, the CASAC Ozone Panel stated that "'[c]linically relevant effects are decrements >10%, a decrease in lung function considered clinically relevant by the American Thoracic Society."[418] The CASAC Ozone Panel also stated that: [A] 10% decrement in FEV_1 can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (i.e., decreased baseline FEV_1) such that a ≥ 10% decrement could lead to moderate to severe respiratory symptoms.[419] Yet EPA is now abandoning the FEV_1>10% benchmark that CASAC, ATS and even EPA itself have all adopted without even an explanation as to how this sudden shift is even justified by the science.

EPA also fails to rationally justify its view that single occurrences of FEV1 decrements ≥ 10 and 15% are somehow of doubtful adversity. CASAC found that such single occurrences of such decrements do qualify as adverse and can endanger public health. ATS has further specifically identified exposure to air pollution that increases the risk of an adverse effect to the entire population as adverse, even though it may not increase the risk of any individual to an unacceptable level.[420] EPA asserts that it "does not believe it would be appropriate to set a standard that is intended to eliminate all ozone-induced FEV_1 decrements," but that is different than FEV_1 decrements ≥10%, and EPA asserts that this is consistent with CASAC's advice, which did not include a recommendation to set the standard level low enough to eliminate all ozone-induced FEV_1 decrements ≥10% or ≥15%.[421] That bare assertion, however, simply does not address whether the referenced decrement levels are sufficient to connote adverse effects, and if not, why not.

CASAC's advice on this score is scientific in nature and quite clear: It refers to the 10% and 15% FEV1 decrement thresholds as "scientifically relevant" surrogates for adverse effects. Therefore, EPA would need to articulate sound scientific grounds for rejecting that advice. EPA has failed to do so. Even if CASAC's advice were policy advice, EPA has offered no rational reason for departing from it.

b. Failure to Propose a Range of the Primary Standard that Includes Levels Down to 60 ppb

CASAC stated as one of its "scientific conclusions" the following:

> The CASAC concurs that 60 ppb is an appropriate and justifiable scientifically based lower bound for a revised primary standard. This is based upon findings of adverse effects, including clinically significant lung function decrements and airway inflammation, after exposures to 60 ppb ozone in healthy adults with moderate exertion (Adams 2006; Schelegle et al., 2009; Brown et al. 2008; Kim et al., 2011), with limited evidence of adverse effects below 60 ppb. The CASAC further notes that clinical studies

[417] 73 Fed. Reg. at 16,454/3-55/1.
[418] Letter from CASAC Chair Dr. Jonathan M. Samet to U.S. EPA Administrator Lisa P. Jackson on CASAC Response to Charge Questions on the Reconsideration of the 2008 Ozone National Ambient Air Quality Standards, (EPA-CASAC-11-004), 2, Mar. 30, 2011
[419] *Id*. at 7.
[420] Proposed Rule at 75,263/1.
[421] *Id*.

> do not address sensitive subgroups, such as children with asthma, and that there is a scientific basis to anticipate that the adverse effects for such subgroups are likely to be more significant at 60 ppb than for healthy adults.[422]

The above statement appeared in a portion of the CASAC letter specifically identified by CASAC as providing scientific conclusions.[423]

Contrary to the above-quoted scientific advice from CASAC, EPA did not set 60 ppb as the lower bound for its proposed range for the primary standard. EPA fails to offer sound scientific reasons for rejection of CASAC's advice on this score. Instead, EPA relies on various assertions of alleged uncertainties and policy concerns.[424] Indeed, in a key paragraphs that seeks to justify rejection of CASAC's recommendation of 60 ppb as the lower bound, EPA recites claims of "uncertainty" no less than seven times. Yet the D.C. Circuit has ruled that assertions of uncertainty do not suffice to explain rejection of CASAC's scientific conclusions. *Mississippi*, 744 F.3d at 1357. EPA must explain why the evidence on which CASAC relied cannot support the degree of confidence CASAC placed in it. Id. EPA fails to do so here. The only explanations EPA offers on this score lack sound scientific support, are arbitrary, and/or are based on policy concerns.

For example, EPA asserts that a decision to set the primary standard at 60 ppb "would place a large amount of weight on the potential public health importance of virtually eliminating even single occurrences of exposures of concern at and above 60 ppb, though controlled human exposure studies have not reported the adverse combination of respiratory symptoms and decrements in lung function following exposures to 60 ppb."[425] This explanation:

- Fails to confront CASAC's stated rationale: namely, the "findings of adverse effects, including clinically significant lung function decrements and airway inflammation, after exposures to 60 ppb ozone in healthy adults." CASAC did not find that its recommendation of 60 as the low end of the range required placing a large amount weight on virtually eliminating single exposures above 60 (although such single exposures would plainly meet CASAC's definition of adverse effects as discussed above). To the contrary, CASAC noted that a 60 ppb standard would "reduce" such single exposures, and allow virtually no children to experience "*two or more* exposures in a year." [426]

- Supplants CASAC's, ATS's, and even EPA's assessment of what constitutes an adverse health effect—FEV1 10% or greater decrements--with EPA's new requirement to that there be a combination of respiratory symptoms and lung decrements. Indeed, EPA takes this a step further by attempting to ascribe significance to the alleged absence of exposure studies finding the combination of

[422] CASAC Letter 2014a at 7.
[423] *Id*. at 6 ("While uncertainty is inherent in assessments of this type, CASAC finds that there is sufficient weight of evidence and degree of confidence to reach the following scientific conclusions").
[424] Proposed Rule at 75,309/3.
[425] *Id*.
[426] CASAC Letter 2014a at 7 (emphasis added).

respiratory symptoms and lung decrements following 60 ppb exposures, but as the discussion above shows, EPA has failed to rationally explain why such a combination is necessary to show adversity of effects.

- Ignores whether the air is in fact save to breath by substituting an analysis of exposures to ozone with the required analysis of what level of ozone is in fact safe to breathe.

EPA likewise asserts that setting the primary standard at 60 ppb would place a large amount of weight on the potential public health importance of further reducing the occurrence of ozone-induced lung function decrements $\geq$ 10 and 15%.[427] Again, this rationale is not responsive to CASAC's reasoning, which: a) found the presence of adverse effects in healthy adults at 60 ppb and identified not only lung function decrements, but lung inflammation as the bases for such finding, and b) found a scientific basis to anticipate that the adverse effects for sensitive subgroups are likely to be more significant at 60 ppb than for healthy adults. EPA offers no scientific bases for questioning any of these findings. The "potential public health importance" of further reducing lung function decrements is a policy consideration, not a refutation of CASAC's scientific findings. In any event, EPA fails to rationally explain why further reducing the occurrence of ozone-induced lung function decrements $\geq$ 10 and 15% is not of public health importance. EPA agrees that such decrements are surrogates for the occurrence of adverse health outcomes.[428] As CASAC has found, and EPA does not dispute, lung function decrements greater than or equal to 15% in children "have been shown to result in significant adverse effects." It is therefore irrational to suggest that reducing the occurrence of such decrements is somehow not of public health importance and direct legal relevance under the Clean Air Act. EPA asserts that not every occurrence of an ozone-induced FEV1 decrement will be adverse, but the agency itself concedes that single occurrences of such decrements can potentially result in adverse effects, and repeated occurrences may be considered adverse even in healthy adults.[429] And the agency's own risk assessment shows that the number of children expected to suffer such decrements is hardly trivial. For example, in just the 15 cities studied, the HREA estimates that 122,000 more asthmatic children would suffer at least one decrement $\geq$ 10% at 70 ppb ozone than at 60 ppb.[430] And 337,000 more children would suffer at least one decrement $\geq$ 15% at 70 ppb than at 60.[431] Likewise, the HREA estimates that in the case study cities alone, 72,000 more asthmatic children will suffer two or more decrements $\geq$ 10% at 70 ppb ozone than at 60 ppb.[432] And 175,000 more children would suffer two or more decrements $\geq$ 15% at 70 ppb ozone than at 60 ppb.[433] The numbers suffering such exposures would obviously be much greater nationally. EPA does not and cannot explain why impacts of such magnitude are not a public health concern in the setting the NAAQS. Indeed, EPA says that it agrees with CASAC on the importance of limiting exposures to O3 concentrations as low as 60 ppb.[434]

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[427] Proposed Rule at 75,309/3.
[428] *Id*. 75,306/1.
[429] *Id*. 75,306/2.
[430] *Id*. at 75,275.
[431] *Id*.
[432] *Id*.
[433] *Id*.
[434] *Id*. at 75,310.

EPA also asserts that setting the primary standard at 60 ppb would place a large amount of weight: a) on analyses of ambient ozone concentrations in locations of multicity epidemiologic studies, despite alleged uncertainties in linking multicity effect estimates for short-term ozone with air quality in individual study cities; and b) on epidemiology-based risk estimates, despite allegedly important uncertainties in those estimates. EPA's refusal to give serious weight to the multicity studies and epidemiology-based risk estimates in determining the level of the standard is arbitrary for reasons discussed elsewhere in these comments, incorporated by reference here.[435] In any event, CASAC did not say it was placing "a large amount of weight" on such studies and estimates in recommending a lower bound of 60 ppb. It said only that a 60 ppb standard 'would be expected to reduce epidemiology-based mortality and morbidity risk for short-term exposures to ozone," and cited that fact as only one among a number of grounds supporting such a level as a lower bound.[436] Further, it is arbitrary for EPA to contend that alleged uncertainties in the epidemiology-based risk estimates are so great that it is not appropriate to use them at all to support the appropriateness of standard levels below 65 ppb.[437] Neither the ISA nor the PA take such an extreme position, nor does the record support it. Nor did CASAC, and EPA has failed to justify its departure from CASAC on this point. Moreover, EPA fails to provide a reasoned basis for using these risk estimates to support consideration of standard levels of 70 and 65, but not 60: There is no reasoned explanation for concluding that uncertainties in those estimates become so much greater at 60 ppb than at 65 ppb that EPA is justified in ignoring them entirely in considering a 60 ppb standard.

Similarly, EPA's assertions that CASAC's recommendation of a lower bound of 60 ppb placed "a large amount of weight" on specific factors as noted above is further belied by CASAC's explicit statement that its recommendation of a range of 60-70 ppb was based on the entire body of scientific evidence:

> The CASAC further concludes that there is adequate scientific evidence to recommend a range of levels for a revised primary ozone standard from 70 ppb to 60 ppb. The CASAC reached this conclusion based on the scientific evidence from clinical studies, epidemiologic studies, and animal toxicology studies, as summarized in the Integrated Science Assessment (ISA), the findings from the exposure and risk assessments as summarized in the HREA, and the interpretation of the implications of these sources of information as given in the Second Draft PA.[438]

Thus, EPA's stated reasons for rejecting CASAC's recommendations are based on the false premise that CASAC placed a large amount of weight on the individual factors EPA cites, when in reality CASAC relied on the evidence collectively.

EPA further asserts that because not all exposures of concern lead to adverse effects and the NAAQS are not meant to be zero-risk or background standards, "alternative standard levels

[435] EPA cites "particularly uncertainties in the shape of the concentration-response functions at lower O3 concentrations," *Id.* at 75,309/3, but in so finding, the ISA and Policy Assessment were referring to ozone levels below 60 ppb. *See, e.g.*, 3-64, 3-115-16.
[436] CASAC Letter 2014a at 7.
[437] Proposed Rule at 75,309/3.
[438] CASAC Letter 2014a at ii (emphasis added); *see also id*. at 8.

below 65 ppb are not needed to further reduce such exposures."[439] That claim misstates CASAC's rationale and is arbitrary. CASAC did not claim that all exposures of concern lead to adverse effects, nor did it advocate zero-risk or background standards: Instead, it cited the range of evidence showing the likelihood of adverse effects at 60 ppb, and EPA's own risk assessment that a substantial number of children suffer such effects at levels at and above 60 ppb. Moreover, it is a non-sequitur for EPA to equate 60 ppb with a "zero-risk" or "background" standard. As EPA's own findings show, even at 60 ppb, substantial numbers of children would suffer lung decrements that EPA agrees are surrogates for adverse effects, so 60 is plainly not zero risk. And EPA's analysis in this rulemaking shows that 60 ppb is far above average (and most maximum) U.S. background levels.

* Finally, EPA's uncertainty claims simply fail to address the strength of the exposure studies that show adverse lung decrements as well as inflammation at 60 ppb. See part V.B.1.b above. CASAC justifiably relied on these studies in recommending 60 ppb as the low end of the range, and EPA fails to offer any reasoned justification for questioning that reliance. That failure is especially telling given that EPA purports to give the greatest weight to the exposure studies in determining the level of the NAAQS.

For all of the above reasons, EPA acted arbitrarily and failed to articulate a sound scientific basis for rejecting CASAC's recommendation that 60 ppb be considered as a level of the standard.

c. EPA's Inclusion of 70 ppb in the Proposed Range

Although CASAC recommended a range of 60 to 70 ppb for the primary standard, it also made scientific findings that adverse effects are likely at, and below,70 ppb. Specifically, CASAC found that "[a]t 70 ppb, there is substantial scientific certainty of a variety of adverse effects, including decrease in lung function, increase in respiratory symptoms, and increase in airway inflammation." CASAC Ltr. 8 (emphasis added). CASAC reiterated and supported that finding elsewhere in its letter to the Administrator. *See id*. at 6 ("The 70 ppb-8hr benchmark level reflects the fact that in healthy subjects, decreases in lung function and respiratory symptoms occur at concentrations as low as 72 ppb and that these effects almost certainly occur in some people, including asthmatics and others with low lung function who are less tolerant of such effects, at levels of 70 ppb and below)(emphasis added); *id*. at 7 ("At a level of 70 ppb for the averaging time and form of the current standard, clinical and epidemiological studies show adverse effects to human health); id (HREA findings "indicate that ozone exposures of 70 ppb …are of significant concern, especially for children, asthmatics, the elderly and other at risk populations").

EPA's proposal of a range for the primary standard that includes 70 ppb cannot be squared with the above-quoted CASAC findings. The primary standard must "be set at a level at which there is 'an absence of adverse effect' on [] sensitive individuals" such as children, the elderly, and people with respiratory illnesses. *Lead Indus. Ass'n v. EPA*, 647 F.2d 1130, 1153 (D.C. Cir. 1980). Because CASAC found substantial scientific certainty of adverse effects at 70 ppb, and found the adverse combination of decrements and respiratory symptoms is almost

[439] Proposed Rule at 75,301/1.

certain to occur at and below 70 ppb, EPA must set the standard below 70 ppb to assure the absence of adverse effects, unless EPA articulates sound scientific reasons for rejecting CASAC's findings on this score. *Mississippi*, 744 F.3d at 1355 ("If EPA's quarrel is with CASAC's scientific analysis, then …EPA must give a sound scientific reason for its disagreement"); *Am. Trucking Ass'ns*, 283 F.3d at 378-79 (The Act "require[s] that EPA must either follow CASAC"s advice or explain why the proposed rule „differs . . . from . . . [CASAC"s] recommendations"")(citations omitted, alterations in original).

EPA's proposal does not even attempt to articulate scientific grounds for rejecting the above-quoted CASAC findings. EPA notes CASAC's statement that EPA's choice of a level within the 60-70 ppb range is a policy judgment, but that hardly converts the above-quoted scientific findings into policy judgments. The D.C. Circuit has made clear that where CASAC makes a scientific finding that adverse effects are likely at a given ozone level, EPA must either act in accord with that finding or articulate sound scientific reasons for disagreeing therewith. *Mississippi*, 744 F.3d at 1355, 1357-58; *Am. Trucking Ass'ns*, 283 F.3d at 378-79. Further, even if CASAC's advice on this score were purely a policy recommendation, EPA must still provide a rational, non-arbitary basis for rejecting it. *Id.*1355. As discussed above and elsewhere in these comments, EPA has failed to provide such a basis. Indeed, EPA's own staff found that effects EPA agrees are adverse are likely at levels of 70 ppb and below ("Thus, respiratory symptoms combined with lung function decrements are likely to occur to some degree in healthy adults with 6.6-hour exposures to concentrations below 70 ppb, and are more likely to occur with 8-hour exposures to 70 ppb and below").[440]

EPA does note a CASAC statement, in the context of the adequacy of the current NAAQS, that the adverse combination of lung function decrements and respiratory symptoms almost certainly occurs in some people following exposures to ozone concentrations lower than 72 ppb – the level at which such adverse combination was shown in the exposure study by Schelegle et al. (2009).[441] EPA goes on to state: "Though CASAC did not provide advice as to how far below 72 ppb adverse effects would likely occur, the Administrator agrees that such effects could occur following exposures at least somewhat below 72 ppb."[442] EPA is simply incorrect in claiming that CASAC "did not provide advice as to how far below 72 ppb adverse effects would likely occur." As noted above, CASAC expressly found that there is substantial scientific certainty of adverse effects at 70 ppb, and that adverse effects are almost certain to occur at levels below 70 ppb as well.[443] CASAC makes clear throughout its comments that concentrations down to 60 ppb "result in lung function decrements large enough to be judged an abnormal response by ATS and that could be adverse in individuals with lung disease" and that a standard set at a level of 70 ppb is still of "of significant concern."[444]

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[440] Policy Assessment at 4-11.
[441] Proposed Rule at 75305/1, *citing* CASAC Letter 2014a at 5.
[442] *Id*.
[443] Although not directed at CASAC's findings, EPA does assert at one point that the Administrator "has decreasing confidence that adverse effects will occur following exposures to O3 concentrations below 72 ppb." *Id*. 75,305/2. But in the next sentence EPA goes on to say: "In particular, compared to O3 exposure concentrations at or above 72 ppb, she has less confidence that adverse effects will occur following exposures to O3 concentrations as low as 60 ppb." *id.* at75305/2-75306/1. EPA thus says nothing there to dispute CASAC's finding at 70 ppb.
[444] CASAC Letter 2014a at 7.

D. EPA Has Failed To Explain How It Has Accounted For A Margin Of Safety and Has Failed To Rationally Explain How The Standards It Has Proposed Incorporate A Margin Of Safety That Is Adequate

1. EPA Must Set Standards Requisite To Protect The Public Health And Provide For An Adequate Margin Of Safety

As discussed above, EPA is required to set primary standards that protect public health with an adequate margin of safety. 42 U.S.C. § 7409. This requirement is intended to address uncertainties associated with inconclusive scientific and technical information. It is also intended to provide a reasonable degree of protection against hazards that research has not yet identified. *State of Mississippi v. EPA*, 744 F.3d 1334, 1353 (D.C. Cir. 2012).

The uncertainties to be addressed by the margin of safety are "components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty."[445] By requiring an adequate margin of safety, "Congress was directing EPA to build a buffer to protect against uncertain and unknown dangers to human health." *State of Mississippi*, 744 F.3d at 1353.

A public health standard must be set at "the maximum permissible ambient air level . . . which will protect the health of any [sensitive] group of the population."[446] Thus, the primary standards must be set at a level that protects against adverse health effects in sensitive persons. *See generally Coal. Of Battery Recyclers Ass'n v. EPA*, 604 F.3d 613, 618 (D.C. Cir. 2010). Importantly, the standards must also provide an adequate margin of safety for vulnerable subpopulations. *Am. Farm Bureau Fed'n*, 559 F.3d at 525-26.

2. EPA's Claim that Its Proposal Provides for an Adequate Margin of Safety Is Arbitrary and Capricious

EPA has arbitrarily and capriciously failed to show how its proposed range of 65-70 ppb provides an adequate margin of safety, for EPA fails to give appropriate consideration to key aspects of the margin of safety inquiry, such as how pollution thresholds might impact sensitive sub-populations. *Am. Farm Bureau Fed'n*, 559 F.3d.

As discussed above, CASAC has unequivocally concluded that there are adverse impacts at a 70 ppb level, using CASAC's, ATS's, and even EPA's own definitions of what constitutes an adverse impact.[447] Again, CASAC advised the following:

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[445] Proposed Rule at 75,238

[446] *Id*. at 75,237 (referencing S. Rep. No. 91-1196, 91st Cong. 2d Sess. 10 (1970)).

[447] *See* CASAC Letter 2014a; Policy Assessment at ES-5 (stating a "standard set within this range would result in important improvements in public protection, compared to the current standard, and could reasonably be judged to provide an appropriate degree of public health protection, including for at-risk populations and lifestages.")

> At 70 ppb, there is substantial scientific evidence of adverse effects as detailed in the charge question responses, including decrease in lung function, increase in respiratory symptoms, and increase in airway inflammation…[448]

CASAC reiterated its scientific conclusion that there are adverse impacts at 70 ppb.

> The 70 ppb-8hr benchmark level reflects the fact that in healthy subjects, decreases in lung function and respiratory symptoms occur at concentrations as low as 72 ppb and that these effects almost certainly occur in some people, including asthmatics and others with low lung function who are less tolerant of such effects, at levels of 70 ppb and below)…[449]

> At a level of 70 ppb for the averaging time and form of the current standard, clinical and epidemiological studies show adverse effects to human health ozone exposures of 70 ppb…are of significant concern, especially for children, asthmatics, the elderly and other at risk populations. [450]

And EPA staff conclusions are consistent with CASAC's determination thatadverse effects *are likely* at even lower concentrations of ozone if the 8-hour averaging period used in the standard is applied.[451]

Nonetheless, EPA's proposal includes consideration of a standard of 70 ppb. Even if this could be reconciled with the health studies of where significant adverse health impacts will occur—which it cannot—it makes it absolutely clear that at 70 ppb there is no margin of safety built into a 70ppb standard. With its proposal, EPA cannot reasonably assert that it has built a buffer that protects against uncertain and unknown danger posed to human health by ozone exposures.

Indeed, EPA agrees with CASAC "on the importance of limiting exposures to O3 concentrations as low as 60 ppb," and "recognizes that levels as low as 60 ppb could potentially be supported."[452] Setting a standard of 70 and asserting that it incorporates an adequate margin of safety is irreconcilable with EPA's conclusion that it is important to limit exposures to 60 ppb and that the evidence in the record could justify a level of 60 ppb given the adverse impacts observed at levels above 60 ppb.

Further, contrary to EPA assertions, CASAC did not conclude that setting the health standard at 60 ppb would equate to the establishment of a zero-risk primary NAAQS. CASAC concluded the exact opposite—adverse health impacts were seen at 60 ppb in controlled human studies, just not below:

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[448] CASAC Letter 2014a at 8 (emphasis added).
[449] *Id*. at 6.
[450] *Id*. at 7.
[451] Policy Assessment at 4-56; *see also* CASAC Letter 2014a at 5, ("It is the judgment of CASAC that if subjects had been exposed to ozone using the 8-hour averaging period used in the standard, adverse effects could have occurred at [a] lower concentration. Further, in our judgment, the level at which adverse effects might be observed would likely be lower for more sensitive subgroups, such as those with asthma.")
[452] Proposed Rule at 75,310.

> The CASAC concurs that 60 ppb is an appropriate and justifiable scientifically based lower bound for a revised primary standard. This is based upon findings of adverse effects, including clinically significant lung function decrements and airway inflammation, after exposures *to 60 ppb* ozone in health adults with moderate exertion…with limited evidence of adverse effects *below 60 ppb*.[453]

Again, this indicates that a standard of 70 ppb cannot be reconciled with protecting the public health, and certainly not with providing an adequate margin of safety.

EPA's own staff has found substantial evidence supporting ozone standard levels lower than the 65-70 ppb that the agency has proposed:

> [T]he evidence from controlled human exposure studies supports considering alternative O_3 standards levels *at least as low as 60 ppb*. Potentially adverse lung function decrements and pulmonary inflammation have been demonstrated to occur in healthy adults at 60 ppb. Thus, 60 ppb is a short-term exposure concentration that may be reasonably concluded to elicit adverse effect in at-risk groups. Pulmonary inflammation, particularly if experienced repeatedly, provides a mechanism by which O3 may cause other more serious respiratory morbidity effects (e.g. asthma exacerbations) and possibly extrapulmonary effects . . . [T]he physiological effects reported in controlled human exposure studies down to 60 ppb O_3 have been linked to aggravation of asthma and increased susceptibility to respiratory infection, potentially leading to increased medication use, increased school and work absences, increased visits to doctors' offices and emergency departments, and increased hospital admissions.[454]

EPA has failed to rationally explain why an ozone standard level in the range of 65 ppb to 70 ppb would provide an adequate margin of safety (much less protect the public outright) when EPA's staff has found that 60 ppb "may be reasonably concluded to elicit adverse effect in at-risk groups," and when there is substantial evidence supporting that finding. EPA must reconsider its decision to disregard the recommendations given to it by CASAC and EPA staff.

3. EPA Has Failed to Rationally Explain How the Standards It Has Proposed Incorporate an Adequate Margin of Safety

EPA can choose reasonable means to provide an adequate margin of safety, but it must also fully and rationally explain how it did so, why it believes the proposed standard will provide an adequate margin of safety, and "why [the agency] chose one method rather than another" for ensuring the margin of safety. *Am. Farm Bureau*, 559 F.3d at 526; *Am. Trucking Ass'ns v. EPA*, 283 F.3d 355, 368 (D.C. Cir. 2002); *Lead Indus. Assn'n v. EPA*, 647 F.3d 1130, 1161-1162 (D.C. Cir. 1980). EPA has failed to provide such an explanation here. EPA asserts that "it takes the need for an adequate margin of safety into account as an integral part of [its] decisionmaking on

[453] CASAC Letter 2014a at 7 (citations omitted) (emphasis added).
[454] Policy Assessment at 4-57.

the appropriate level, averaging time, form, and indicator of the standard."[455] But nowhere does the agency explain: a) how and where the margin of safety requirement was factored into EPA's decisionmaking on these matters in the proposal at issue here; b) why specifically EPA believes its decisions on these matters will ensure an adequate margin of safety; and c) why EPA chose the described method for meeting the margin of safety requirement as opposed to another approach (e.g., an approach whereby EPA adds the margin of safety after determining a level of likely adverse effects). EPA offers only bare assertions that its proposal provides for a margin a safety – assertions that fall far short of providing the above-described explanations as required by the statute, case law, and requirements for reasoned decisionmaking.

EPA's cursory background discussion[456] of factors relevant to the margin of safety plainly does not suffice as an explanation of how the agency provided for a margin of safety in this specific case, and why it was sufficient. *See Missouri Public Serv. Comm'n v. FERC*, 601 F.3d 581, 586 (D.C. Cir. 2010) ("a passing reference to relevant factors…is not sufficient to satisfy the [agency's] obligation to carry out reasoned and principled decisionmaking." (internal quotation marks omitted; second alteration in original)). Merely stating the governing legal or factual test is no substitute for applying that test to the record before the agency. *Douglas Foods Corp. v. NLRB*, 251 F.3d 1056, 1066 (D.C. Cir. 2001) ("The NLRB cannot discharge its obligation [to carry out an analysis of three factors and to weigh them] merely by citing the appropriate authority and averring that it gave proper consideration. It actually must consider the factors as they apply to the instant case, and explain the basis for its conclusions."). In context of the Act's health standards, EPA has the "heaviest of obligations to explain and expose every step of its reasoning." *American Lung Ass'n v. EPA*, 134 F.3d 388, 392 (D.C. Cir. 1998). Deference to EPA's expert judgment requires that the agency carefully and clearly explain exactly how it reached the result it did. *Id*. EPA has failed to provide such a clear explanation here.

The mere fact that EPA has considered ozone's effects on sensitive subpopulations does not by itself adequately explain how EPA's proposal will actually assure an adequate margin of safety. Indeed, nowhere does EPA state that its consideration of ozone's effects on sensitive populations is meant to address the margin of safety requirement. Nor does EPA explain how it is establishing a margin of safety for sensitive subpopulations. Acknowledging that these sensitive subpopulations exist and are more likely to suffer adverse effects at various ozone levels does not equate to explaining how or why the proposed standard will provide them with an adequate margin of safety.

Nor is it sufficient for EPA to simply assert that it takes the need for an adequate margin of safety into account as "an integral part of [its] decisionmaking on the appropriate level, averaging time, form, and indicator of the standard."[457] Such an assertion does not explain how or why such decisionmaking adequately addresses the margin of safety requirement. For example, nowhere does EPA explain how margin of safety considerations were integrated into its proposal on the form of the standard: Such integration is hardly apparent from the face of the proposed form, which – as discussed in part V.B.3. above – allows multiple exposures far in excess of levels in the 60-70 ppb range and an unlimited number of 8-hour periods in a given

[455] Proposed Rule at 75,309/1.
[456] *E.g., id.* at 75,237-38, 75,304, n.157, 75,309.
[457] *Id*. at 75,309

year in which people can be exposed to ozone levels higher than those at which EPA says adverse effects are likely to occur. Nor does EPA explain how margin of safety considerations were integrated into its proposal on the level of the standard. Among other things, as further discussed below and elsewhere in these comments, EPA fails to explain how its proposed range assures the absence of adverse effects to sensitive populations, much less provides such populations with an adequate margin of safety. Indeed, in determining the level of the proposed standard, EPA's proposal repeatedly opts for a direction contrary to a key purpose of the margin of safety – namely, to build a buffer to protect against uncertain and unknown dangers to human health. For example, faced with evidence of adverse effects from ozone exposures in the 60-65 ppb range, EPA – citing alleged uncertainties - refuses to even consider preventing such exposures.[458] Likewise, in proposing 70 ppb as the upper end of its range, EPA provides no buffer at all against adverse effects that CASAC found to be certain at 70 ppb with substantial scientific certainty. Nor does EPA respond at all to CASAC's specific advice to set the standard below 70 ppb to provide an adequate margin of safety -- other than to assert that the choice involves a policy judgment.

EPA's failure to provide an explanation of how it is providing for the margin of safety here contrasts sharply with the agency's actions in other NAAQS decisions, where the agency did provide such explanations. For example, in its 1979 ozone NAAQS decision, EPA "determined…the 'probable level for adverse effects in sensitive persons,'" and then provided a margin of safety below that level based on a detailed consideration of multiple factors. *Am. Petroleum Inst. v. Costle*, 665 F.2d 1176, 1187 (D.C. Cir.1981) (citing 44 Fed. Reg. 8202, 8216-17 (1979)). Here, in contrast, EPA does not identify a probable level for adverse effects in sensitive persons, much less explain how its proposed standard range provides a margin of safety below that level. In *Lead Industries*, rather than find an adverse effect level, then add a margin of safety, EPA incorporated the margin of safety at two places in the standard-setting process. 647 F.2d at 1161-62 & n.80. EPA explained "why [it] chose one method rather than another" of providing a margin of safety, *id.* 1162, and explained in the rule itself the points at which it was accounting for margin of safety and how it was doing so, *id.* 1144. Thus, there are plainly ways for EPA to lay out how it is adequately addressing the margin of safety, including for sensitive subpopulations.

EPA's failure to explain itself on these issues is crucial because it goes to the heart of the agency's duty in setting the NAAQS. Without a reasoned explanation, the public and reviewing courts cannot know whether EPA designed the standard to protect against not only known adverse effects on health, but those of scientific uncertainty.

For all the foregoing reasons, EPA has failed to adequately explain how its proposal assures an adequate margin of safety as required by the Act.

E. Even if EPA Declines to Deviate from Its Proposed Range, EPA Must Set the Standard No Higher than 65 ppb, at the Bottom of Its Proposed Range

As explained above, to fulfill its obligation under the Clean Air Act and to act rationally, EPA must set the NAAQS at 60 ppb. If EPA refuses to adopt a standard below its proposed

[458] *Id*. at 75,309-10.

range of 65-70 ppb, however, it must set the standard at 65 ppb, for that will be closest to the requisite level. In no event should EPA set the standard as high as 70 ppb. We do not accept EPA's reasoning for rejecting a level of 60 ppb, but on this basis, a standard of 65 ppb would better meet EPA's stated criteria and provide significantly more protection.

The information before EPA shows that a standard of 65 ppb would prevent substantial incidents of adverse effects that are likely to occur at 70 ppb and would be significantly more protective than a standard of 70 ppb. EPA rejects a standard of 60 ppb, saying it "focuses on" and "emphasizes" limiting multiple exposures of concern to ozone and "balances" that focus against three factors: (1) the fact that "not all exposures of concern will result in adverse effects"; (2) a decreased confidence that adverse effects will occur at 60 ppb as opposed to at 70 or 80 ppb; and (3) the belief that "NAAQS are not meant to be zero-risk standards."[459]

As an initial matter, the balance EPA proposes is itself without foundation. As discussed elsewhere in these comments, a focus or emphasis on multiple exposures fails to assure the absence of adverse effects, as the evidence shows (and EPA concedes) that single exposures can and do produce adverse effects, both at the individual and population levels. For the same reasons, it is irrelevant that not all exposures of concern will result in adverse effects: The Act requires the standard to assure the absence of adverse effects in all groups, including sensitive populations, a mandate that is not met merely because some individuals or portions of the population do not suffer adverse effects from single or even multiple exposures to a given ozone level. Further, for the reasons given above, it is very likely that exposure to ozone at 60 ppb causes adverse effects in sensitive populations; EPA's second countervailing factor is thus incorrect. EPA's third countervailing factor rests on false premises. EPA cites *Mississippi*, 744 F.3d at 1343, for the proposition that "NAAQS are not meant to be zero-risk standards." Proposed Rule at 75,305/3 & n.163. That case does not establish the proposition. *Mississippi* says only, "Determining what is 'requisite' to protect the 'public health' with an 'adequate' margin of safety may indeed require a contextual assessment of acceptable risk." 744 F.3d at 1343 (quoting *Whitman*, 531 U.S. at 494-95 (Breyer, J., concurring in part and concurring in the judgment)). The "may" indicates that the statement is just dicta. The underlying Supreme Court concurring opinion does not have the force of law, either: it is one Justice's opinion that was not joined by any other member of the Court. In any event, the concern over "zero-risk standards" is misguided, for the issue here is whether EPA may set standards that allow large-scale, known adverse effects to persist. It may not.

Even if EPA's "focus," "emphasis," and countervailing factors were valid (which we do not accept), they support a standard at least as protective as 65 ppb. Again, as discussed above, EPA cannot rely on assertions that exposures will not happen because people will stay inside and will not experience the unsafe ozone events that empirical evidence makes clear will in fact occur all around them. Nor in any event is the exposure study defensible. But even beyond these points, EPA's own analysis makes clear that setting the standard at levels below 70 ppb reduces what even EPA acknowledges are multiple exposures to levels of pollution that CASAC has judged to cause adverse health impacts.

[459] *Id*. at 75,305/3.

For example, EPA finds that a level of 70 ppb would "virtually eliminate the occurrence of two or more O_3 exposures of concern for the 70 and 80 ppb benchmarks, and … substantially reduce the occurrence of two or more O_3 exposures of concern for the 60 ppb benchmark, compared to the current standard" approximately a 60% reduction from the current standard.[460] Again, as noted above, CASAC has concluded that the health science reflects adverse impacts at 60 ppb and above. In contrast, a level of 65 ppb would be much more effective at eliminating these exposures, in fact it would "eliminate[ed] almost all exposures of concern" to the benchmarks of 70 and 80 ppb and "protect[ing] more than 99% of children in urban study areas" from multiple exposures to the benchmark of 60 ppb. For the 60 ppb benchmark, that protection would be approximately a 95% reduction from the current standard.[461] For the 15 areas studied, a 70 ppb standard would likely force 253,000 more kids to endure multiple exposures to at least 60 ppb of ozone than a 65 ppb standard would, and 5,100 more kids to endure multiple exposures to at least 70 ppb of ozone.[462]

Other factors also indicate that a 65 ppb standard would be significantly more protective than a 70 ppb standard. The level 70 ppb would "virtually eliminate" even single exposures to the benchmark 80 ppb and would yield "important reductions, compared to the current standard," for the benchmarks 70 and 60 ppb (about 70% for 70 ppb and about 50% for 60 ppb)—meaning that exposures to 70 and above would persist.[463] By contrast, the level 65 ppb would eliminate "almost all" exposures to the benchmarks of 80 and 70 ppb, and would reduce exposures to the 60 ppb benchmark by about 80% from the current standard.[464] In just the 15 areas studied, that is 80,000 fewer kids exposed to the 70 ppb benchmark and 784,000 fewer kids exposed to the 60 ppb benchmark.[465] Those differences are substantial: EPA offers no reasoned explanation to conclude otherwise.

A 65 ppb level would be significantly more effective: even in the worst case, 98% to over 99% of kids would be protected from decrements of 15% (on average, a 54% reduction from the current standard), and 89-99% would be from decrements of 10% (a 37% reduction).[466] As noted above, EPA, CASAC, and the PA all have said that a 10% decrement is adverse for people with asthma. The contrast between the 70 and 65 ppb levels is even more striking when comparing the level of protection against single instances of such decrements: the 70 ppb level would be only a 26% reduction from the current standard for 15% decrement and a 15% reduction for 10% decrement;[467] the 65 ppb level would be about twice as effective, making a 50% reduction from the current standard for 15% decrement and a 31% reduction for 10% decrement.[468]

As the tables below make clear, a standard of 70 ppb would allow from tens of thousands to millions of sensitive individuals to experience exposures of concern, and from hundreds of thousands to millions of kids to endure significant, adverse lung function decrements. EPA offers

[460] *Id*. at 75,305/3-06/1.
[461] *Id*. at 75,306/3.
[462] *See id.* at 75,298 tbl.4.
[463] *Id*. at 75,306/1.
[464] *Id*. at 75,306/3.
[465] *See id.* at 75,297 tbl.4.
[466] *Id*. at 75,306/3-07/1.
[467] *Id*. at 75,306/3
[468] *Id*. at 75,307/1.

no explanation for why it is lawful or rational under the Clean Air Act to allow these exposures and effects to persist with a 70 ppb standard. Though it is itself inadequate, a 65 ppb standard would be about twice as protective compared to the current standard.

TABLE 23 Mean number of people with at least one daily maximum 8-hr average O_3 Exposure at or above 60ppb while at moderate or greater exertion (includes 15 urban area studies)
(Data derived from EPA, HEALTH RISK AND EXPOSURE ASSESSMENT FOR OZONE FINAL REPORT, EPA-452/R-14-004A, 5-87, TABLE 5-13 (AUGUST 2014).

Standard 8-hr level	School age children	Asthmatic school-age children	Asthmatic adults	Seniors (aged 65-95)
65 ppb	392,000	42,000	25,000	38,000
70 ppb	1,176,000	126,000	83,000	129,000
75 ppb	2,316,000	246,000	180,000	282,000

TABLE 24 Lung Function Decrements for Alternate Ozone Standards for Children (ages 5-18) Experiencing One or More Decrements Per Season
(Data derived from 79 Fed. Reg. at 75,279 tbl.5)

Decreased Lung Function	Alternate Ozone Standard	Number of children experiencing decrement	Average % reduction from current standard
≥10%	65 ppb	1,896,000	31%
	70 ppb	2,527,000	15%
≥15%	65 ppb	356,000	50%
	70 ppb	562,000	26%
≥20%	65 ppb	106,000	59%
	70 ppb	189,000	32%

As for other health endpoints, epidemiological studies and related risk assessments also support the 65 ppb level over a standard of 70 ppb. EPA contends that the single-city studies before it show that any level between 65 and 70 ppb "would result in improvements in public health, beyond the protection provided by the current standard,"[469] but suggests that they provide no basis for distinguishing between 65 and 70 ppb.[470] That analysis ignores studies like Delfino (1997), Burnett (1997), Burnett (1999), Koken (2003), Yang (2003), and Vedal (2003), all of which were cited in the Staff Paper in the last ozone NAAQS review.[471] For multi-city studies, EPA itself notes that at least one reported adverse effects where a majority of the cities would have met a standard of 70 ppb but not a standard of 65 ppb, and "several" reported health effects even though a majority of the cities studied would have met a NAAQS set at 65 ppb.[472] Setting

[469] *Id.* at 75,307/3,
[470] *See id.*
[471] *See* U.S. EPA (2007). Review of National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper (EPA-452/R-07-07), app.3B. (See Exhibit 13).
[472] Proposed Rule at 75,307/3-08/1.

the standard at 65 ppb would thus accord with the underlying city data in those studies. The risk assessment also predicts that 65 ppb will prevent significant numbers of adverse effects that 70 ppb will not: For deaths tied to short-term exposure, when using 40 ppb as the cutoff for deaths caused by ozone, a 70 ppb standard would reduce deaths by 10%, while a 65 ppb standard would reduce deaths by 50% (both over the current standard); with 60 ppb as the cutoff, a 70 ppb standard would reduce deaths by 50-70%, and 65 ppb would reduce them by over 80%.[473]

EPA suggests that setting the standard at 65 ppb would require it to weigh various "uncertainties" less and the importance of reducing various "exposures of concern" and lung function decrements more.[474] But EPA cannot rely on assertions of uncertainty when faced with scientific findings by CASAC and scientific evidence that adverse effects are likely at 65 ppb. As noted elsewhere in these comments, CASAC and EPA's own staff have found, based on the scientific record, that adverse effects are likely at ozone levels above 60 ppb. Even if there were some doubt, the Clean Air Act's "precautionary and preventative orientation" reflects that "Congress directed the Administrator to err on the side of caution in making the necessary decisions." *Lead Indus. Ass'n*, 647 F.2d at 1155.

For similar reasons, EPA could not lawfully or rationally find that a standard of 70 ppb would be sufficiently protective to satisfy the Administrator's statutory obligations to set the standard at a level that avoids adverse effects and provide an adequate margin of safety. CASAC has found that "[a]t 70 ppb, there is substantial scientific certainty of a variety of adverse effects."[475] Indeed, CASAC repeatedly made clear that adverse effects are virtually certain at 70 ppb: it said that the adverse combination of decrements and symptoms occur in healthy individuals at 72 ppb and "these effects almost certainly occur in some people, including asthmatics and others with low lung function who are less tolerant of such effects, at levels of 70 ppb and below";[476] and it also said that "[a]t a level of 70 ppb for the averaging time and form of the current standard, clinical and epidemiological studies show adverse effects to human health."[477] To establish a standard at 70 ppb, EPA would have to explain why, as a scientific matter, it disagrees with CASAC's finding. *Mississippi*, 744 F.3d at 1357-58. As further discussed elsewhere in these comments, it has not done so. Thus, again, EPA has also failed to explain how a 70 ppb standard would be set at a level where ozone does not cause adverse effects. *See Coal. of Battery Recyclers Ass'n*, 618 F.3d at 618.

EPA has also not explained how the 70 ppb level provides an adequate margin of safety. As explained elsewhere in these comments, EPA has arbitrarily failed to explain what its approach is for accounting for the margin of safety. To the extent EPA appears to consider relevant factors, its purported concerns about "uncertainty" seem only to drive it toward setting a less protective standard.[478] That is precisely the opposite of the orientation of the Act and is thus unlawful and irrational. *E.g.*, *Lead Indus. Ass'n*, 647 F.2d at 1155 ("orientation" of the Act is

[473] *Id.* at 75,308/3.
[474] *See id*. at 75,309/1-2.
[475] CASAC Letter 2014a at 8.
[476] *Id.* at 6; *see also id.* at 5 (explaining that the combination of decrements and statistically significant increase in respiratory symptoms is adverse)
[477] *Id.* at 7
[478] *See, e.g.*, Proposed Rule at 75,309/2 (suggesting that a 70 ppb standard would more heavily weigh various uncertainties).

toward protection and precaution); *see, e.g.*, *NRDC v. EPA*, 2014 WL 7269521, at *13 (D.C. Cir. Dec. 23, 2014) (where EPA action is "is 'untethered to Congress's approach,'" it is unlawful); *Ass'n of Private Sector Colleges v. Duncan*, 681 F.3d 427, 448 (D.C. Cir. 2012) (agency failed to explain elimination of "safe harbor" that was consistent with statutory "goal").

Further, as CASAC suggested, [479] a 70 ppb level (and nearby levels) does not provide a margin of safety, and EPA has not explained how it does. EPA acknowledges that (1) healthy young adults experience adverse effects when exposed to 72 ppb ozone over 6.6 hours;[480] (2) CASAC found that if "subjects had been exposed to ozone using the 8-hour averaging period used in the standard…, adverse effects could have occurred at lower concentration";[481] and (3) the PA found that "significant increases in respiratory symptoms combined with lung function decrements" are a combination that "is likely to occur to some degree in healthy adults with 6.6-hour exposures to concentrations below 72 ppb, and also are more likely to occur with longer (*i.e.*, 8-hour exposures)."[482] Thus, even if a focus on the upper end of EPA's proposed standard range were appropriate, to apply an adequate margin of safety for healthy young adults, EPA would have to establish a standard below 72 ppb. Nowhere does EPA explain why 2 ppb would be adequate both to prevent adverse effects and assure an adequate margin of safety. The lack of explanation is particularly problematic given that EPA mischaracterizes what the PA said. Rather than warning of adverse effects below "72 ppb," as EPA intimates, the PA said that "respiratory symptoms combined with lung function decrements are likely to occur to some degree in healthy adults with 6.6-hour exposures to concentrations below 70 ppb, and are more likely to occur with 8-hour exposures to 70 ppb and below."[483]

As was the case in the 2006 PM NAAQS, *see Am. Farm Bureau Fed'n*, 559 F.3d at 525-26, EPA also fails to explain how a 70 ppb (and nearby levels) standard could provide an adequate margin of safety for sensitive populations. Nothing in EPA's discussion of how such a standard might provide an adequate margin of safety even mentions protecting sensitive populations.[484] Yet EPA acknowledges that (1) "at-risk groups…could experience larger and/or more serious effects" than these healthy young adults;[485] (2) "subjects with asthma appeared to be more sensitive to acute effects of O_3 in terms of FEV_1 and inflammatory responses than healthy non-asthmatic subjects";[486] and (3) CASAC made the scientific finding that "the level at which adverse effects might be observed would likely be lower for more sensitive subgroups, such as those with asthma."[487] Moreover, CASAC made an even stronger scientific finding than EPA gives it credit for: that the adverse combination of decrements and respiratory symptoms "almost certainly occur in some people, including asthmatics and others with low lung function who are less tolerant of such effects, at levels of 70 ppb and below."[488] Thus, EPA has not explained how a 70 ppb level (and nearby levels) could provide an adequate margin of safety for

[479] CASAC Letter 2014a at 8 ("a level of 70 ppb…may not meet the statutory requirement to protect public health with an adequate margin of safety.")
[480] *E.g.*, Proposed Rule at 75,304/3
[481] *Id.* at 75,305/1 (agreeing that adverse effects "could occur following exposures at least somewhat below 72 ppb")
[482] *Id.* at 75,296/2.
[483] Policy Assessment at 4-11 (emphasis added).
[484] Proposed Rule at 75,309/1-2.
[485] *Id.* at 75,288/3
[486] *Id.* at 75,265/3
[487] *Id.* at 75,287/1, 75,296/2 n.127 (quoting CASAC 6-26-14 Letter at 5)
[488] CASAC Letter 2014a at 6 (emphasis added).

both the healthy young adults who experience adverse effects at 72 ppb and the sensitive subpopulations who experience adverse effects at levels below 72 ppb. Nor has EPA explained how a standard at 70 ppb (and nearby levels) would provide any margin of safety for sensitive groups, and, as further discussed elsewhere in these comments, EPA has not explained any departure from CASAC's scientific conclusion that adverse effects in sensitive groups "almost certainly occur…at levels of 70 ppb and below."

Finally, we note that in the previous ozone NAAQS review (concluding in 2008), EPA took the position that to provide requisite protection for public health and an adequate margin of safety, the standard "must be set at a level appreciably below 0.080 ppm, the level at which there is considerable evidence of effects in healthy people."[489] EPA went on to find that "appreciably below" meant 0.005 ppm (5 ppb) below, and set the standard at 0.075 ppm (75 ppb). In the current review, EPA's staff has found that adverse effects in healthy people are likely to occur at 70 ppb and below (finding that the evidence supporting the occurrence of adverse respiratory effects is strongest for exposures at or above the 70 and 80 ppb benchmarks).[490] Thus, to set the standard at a level "appreciably below" the level at which adverse effects are likely in healthy people in a manner consistent with EPA's 2008 NAAQS decision, EPA would need to set the standard no higher than 65 ppb. EPA offers no reasoned basis for adopting a less protective approach (or less protective definition of "appreciably below") in the current review than it did in 2008.

F. Public Welfare Secondary National Ambient Air Quality Standard for Ozone

1. Introduction

Secondary National Ambient Air Quality Standards (NAAQS) must protect the public welfare from any known or anticipated adverse effects of air pollution. This standard is distinguishable from the primary human health-based NAAQS as it encompasses safeguards for terrestrial and aquatic ecosystems including wildlife and vegetation. In the context of ozone pollution, the standards must protect against any known, or anticipated adverse effects from ozone present in the air. In particular, plants are at increased risk. Adverse effects include disruption of normal storage of nutrients and carbon, direct visible damage to foliage, and climate change. These impacts directly translate to public welfare harm due to the effects on crop and forest productivity, resilience, scenic beauty, ecosystem functioning, and climate change.

The EPA is charged with reviewing the latest science and evaluating the protectiveness of both the primary and secondary standards every five years under the Clean Air Act NAAQS process. To date the secondary standard for ozone has been set at the same level and form as the primary health standard, even in the face of the past three reviews that provided significant science-based evidence that this was not protective of the public welfare.

[489] 73 Fed. Reg. at 16,480 (Mar. 27, 2008); *see also id.* at 16,483 (standard should be set at level that "is appreciably below 0.080 ppm, the level in controlled human exposure studies at which adverse effects have been demonstrated").

[490] Policy Assessment at 4-11; *see also* Proposed Rule at 75,305/2 (finding that the evidence supporting the occurrence of adverse respiratory effects is strongest for exposures at or above the 70 and 80 ppb benchmarks).

Our organizations believe that EPA must set a W126 standard of 7 ppm-hrs to be protective of tree growth, foliar health, and to mitigate anthropogenic climate change. This form and level reflects the best protection promoted by the CASAC and by the National Park Service.[491] We are dismayed that EPA has left the secondary standard in the shadows in its 2014 proposal. Of particular concern is EPA's unwillingness to propose a W126 standard despite the Agency's clear recognition that this is the correct metric to characterize impacts to vegetation. Such an approach is unlawful and arbitrary. Further, in selecting the form of the standard and level of protection, the Agency has arbitrarily rejected or ignored advice input from CASAC and the National Park Service. EPA appears to have selected a range of 13-17 ppm-hrs based on the Agency's estimated equivalency with the primary range of 65-70 ppb rather than as a matter of public welfare protection. EPA has failed to rationally justify advancing this deficient standard for public welfare and as such it must be corrected to reflect the recommendation of its scientific advisors and federal land managers.

The EPA Administrator is taking comment on the W126 as the alternative form of the secondary NAAQS, as well as a level as low as 7 ppm-hrs as recommended by CASAC and which we support. We urge Administrator McCarthy to set the standard using the W126 form which has been clearly demonstrated as the most scientifically relevant form to protect vegetation. The standard must reflect the fact that ecosystems and species respond differently to ozone pollution than humans. Protection of the above-described welfare values is at the core of this decision.

In consideration of the advice of CASAC, the position of the National Park Service, and the clear science that demonstrates impacts on tree growth and foliar damage at levels greater than 7 ppm-hrs, we urge the Administrator to select this level of protection. Finally, consideration of the conservative approach of calculating the W126, which limits the summation window to only 12 daylight hours, only 3 months of the growing season, and the averaging across a 3 year window, further supports selection of the level of 7 ppm-hrs because it would afford the level of protection requisite to protect public welfare.

2. Secondary NAAQS must protect public welfare

Section 109(b)(2) of the Clean Air Act states that the secondary standard must "specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air."

EPA must base its decision on the significant body of science brought forth in the Integrated Science Assessment (ISA), the Welfare Risk and Exposure Assessment (WREA), as well as the advice of CASAC and the National Park Service. These authorities all strongly support a separate secondary standard, and one far more protective than EPA has proposed.

In selecting a form to protect vegetation EPA must use the best biologically relevant metric. The W126 metric has been recommended to EPA (and found by the EPA Administrator

[491] NPS Letter N3615 (2350) Mar. 20, 2014.

herself) as being the most biologically relevant form in this current review, as well as in the 2006 review and the 2010 reconsideration.

a. Adverse effects to vegetation and ecosystems

Adverse effects from ozone include disruption of normal storage of nutrients and carbon and direct visible damage to foliage. These impacts directly translate to public welfare harm due to the effects on crop and forest productivity, resilience, scenic beauty, and ecosystem functioning. In terms of ecosystem services impacts include, but are not limited to, cultural (e.g. recreation) and product (e.g. agriculture) related services.

In this review EPA is clear that ozone exposures on vegetation and ecosystems are wide-spread and pervasive. The final 2013 ozone ISA documents the ecosystem effects that the Agency considers causal and likely casual, including:[492]

- Visible injury to plants and tree foliage effects
- Reduced vegetation growth
- Reduced productivity in terrestrial ecosystems
- Reduced yield and quality of agricultural crops
- Alteration of below ground biogeochemical cycles
- Alteration of terrestrial ecosystem water cycling
- Reduced carbon sequestration in terrestrial ecosystems
- Alteration of terrestrial community composition

As outlined in the most recent review the latest science has advanced our understanding around ozone's role in disrupting below ground processes including carbon storage. This has important ramifications related to carbon sequestration and mitigation of greenhouse gas emissions.

The causal effects defined above clearly show that when ozone is present in the ambient air there are significant and multiple costs to vegetation, and while some species are more sensitive than others it is also recognized that there is a cumulative impact for the ecosystem, wildlife habitat, and larger landscapes. This ubiquitous effect of ozone pollution must be addressed in setting the public welfare standard.

b. Adverse effects to climate

Our groups are concerned that the proposed rule does not properly characterize the welfare impacts of radiative forcing from ozone. Ozone not only harms vegetation, but also is a potent greenhouse gas. The ISA states that there is a "relationship between the changes in tropospheric O3 concentrations and effects on climate."[493] The proposed rule also states that "the evidence supports a causal relationship between changes in tropospheric O3 concentrations and radiative forcing."[494] While it is true, as outlined in the ISA, that there are a number of details related to ozone's climate impacts that have not been resolved, the important facts are known.

[492] ISA, Table 1–2.
[493] *See* ISA at 2–47.
[494] Proposed Rule at 75234, 75315.

First, it is clear that ozone has a strong warming impact, especially in Northern mid-latitudes (where the United States is) and in the Arctic. Second, whatever its exact radiative forcing, ozone is the third strongest greenhouse gas. Third, it is well-established that ozone can be reduced through decreases in methane, carbon monoxide and VOCs. As EPA acknowledges, reducing these precursors would significantly benefit public health as well as climate. Consequently, we urge Administrator McCarthy to consider the direct as well as indirect climate impacts of ozone as she makes her judgment about setting the level of the secondary standard.

i. Direct Climate Impacts in the Arctic

The Arctic region deserves special consideration both due to the Class I areas located within Alaska[495] near the Arctic Circle and because ozone-mediated climate impacts in the Arctic have implications for the contiguous United States.

Ozone has a larger impact in the Arctic than in other regions.[496] This is so both because ozone remains in the atmosphere longer than average in the Arctic Winter and Spring[497] and because ozone is most effective at absorbing shortwave radiation, such as that reflected from snow and ice, in the Arctic.[498] In fact, ozone is estimated to exert a radiative forcing of approximately 1 W/m^2 during the Arctic summer[499] and to have increased temperatures as much as 0.5 °C in the winter and spring in the Arctic in the last century.[500]

The Arctic is a unique region that hosts an intricate and highly specialized ecosystem. Many of the species in the region are the last survivors from the previous ice age. Because the environment is harsh, species in the Arctic are highly adapted to survive in a narrow range of conditions. Furthermore, the inhabitants of the Arctic have no options for migration in the face of a warming environment: they are already as far north as possible and there is no higher elevation to which these species can climb. Arctic species rely upon fundamental features of the Arctic landscape, such as sea ice, permafrost, and seasonal snowpack. Yet, these features are currently teetering on the brink of various climatic "tipping points."

The Arctic is currently experiencing climate changes of a magnitude not experienced anywhere else on Earth. For instance, the Arctic is warming at twice the rate of the rest of the world.[501] Furthermore, sea ice is melting at precipitous and unprecedented rates.[502] Greenland ice sheets are contracting at accelerating rates and permafrost is experiencing deeper and more frequent freeze/thaw cycles.

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[495] The Class I areas within Alaska are: Denali National Park, Bering Sea Wilderness Area, Simeonof Wilderness Area, and Tuxedni Wilderness Area. 40 CFR Part 81, Subpart D.
[496] ISA sec. 10-13,14,18.
[497] ISA at 10-18.
[498] ISA at 10-13.
[499] ISA at 10-13.
[500] ISA at 10-18.
[501] IPCC (2007). CLIMATE CHANGE 2007: SYNTHESIS REPORT at 30.
[502] *See, e.g.,* J. Stroeve et al. (2008). Arctic Sea Ice Extent Plummets in 2007. *Eos, Transactions, American Geophysical Union* 89(2): 13-14; R. Kwok & D.A. Rothrock, Decline in Arctic sea ice thickness from submarine and ICESat records: 1958-2008, *Geophys. Research Letters*, 36, 15501.

Adverse impacts resulting from the accelerated loss of Arctic sea ice extend well beyond the Arctic Ocean and its coast. By reflecting the sun's energy back into space, sea ice is an effective insulator, preventing heat in the Arctic Ocean from escaping upward and warming the lower atmosphere.[503] The decline of sea ice amplifies warming in the Arctic, which in turn has major implications for temperature patterns over adjacent, permafrost-dominated land areas and for weather patterns across the Northern Hemisphere.[504] Higher temperatures will thaw out extensive expanses of permafrost, resulting in the potential release of methane and carbon dioxide currently frozen in Arctic soils, thereby accelerating further warming.[505] Additional warming in the Arctic resulting from the loss of sea ice will also affect weather patterns by altering atmospheric circulation patterns, leading to more extreme weather events and affecting transportation, agriculture, forestry and water supplies.[506] Loss of sea ice in the Arctic Ocean will therefore have serious repercussions as climactic feedbacks resulting from higher temperature increases accelerate, the timing of the seasons is altered, and shifting circulation patterns cascade through the Arctic and beyond.

These climate impacts will directly affect proximate Class I areas within Alaska. In addition, a strong secondary standard for ozone is a critical part of the overall effort to avoid cascading catastrophic consequences for the lower 48 states.

c. Ecosystem Services

In this review EPA clearly delineates Ecosystem Services as a construct to assess the relationships between ozone impacts to vegetation and resultant impacts on public welfare. Ozone pollution has both direct ecological impacts on ecosystem services, such as the damage that happens to plant and tree foliage when exposed to ozone, as well as the resulting impacts to cultural (e.g., recreation) and product (e.g., agriculture) related services. Ecosystem Services establish the benefits received from specific species or ecosystems. In characterizing the impact to public welfare in this way, EPA has access to a construct through which the agency may develop methods to assess what might be expected to change under air quality scenarios representing varying alternatives for a secondary standard. We strongly agree with EPA's recognition that some benefits cannot be marketed and are difficult to quantify but are highly valued in the context of the public welfare nonetheless.

d. Protecting Public Lands

We agree with EPA Class I areas (National Parks, Wildernesses, Forests, and Refuges) hold special value and context to the public and therefore warrant strong protection. Air pollution that harms ecosystems and scenic beauty in these national public lands adversely affects public welfare because, among other things, these special places were set aside for conservation of their natural values, for use and enjoyment by the public, and under the Clean

[503] World Wildlife Fund Int'l (2009). ARCTIC CLIMATE FEEDBACKS: GLOBAL IMPLICATIONS at 8 (M. Sommerkorn & S. Hassol eds.) at 19-20.
[504] *Id.* at 18.
[505] *Id.*
[506] J. Francis & S. Vavrus (2012). Evidence Linking Arctic Amplification to Extreme Weather in Mid Latitudes, 39 *Geophys. Research Letters*, L06801.

Air Act, as places to have the most pristine air quality. 42 U.S.C. 7472. EPA must limit the impacts from ozone pollution to Class I areas to fulfill, and act consistently with, the Congressional mandates to protect and preserve these places for the foregoing purposes. Advancing protections to safeguard the air and resources in these areas is critical to the meeting the Clean Air Act objective.

In giving priority to these areas, the Administrator must consider that Class I areas include many mountain systems that can have high background ozone with little change in diurnal concentrations, even during the daylight hours. Consequently, when ozone pollution events occur they build upon these high background levels and therefore exacerbate overall cumulative impacts. Class I areas also include many wetland ecosystems, that support significant diverse wildlife, and where foliar injury from ozone can be more severe.

In addition to the ozone impacts to vegetation, EPA must consider the climate change impacts of ozone in Class I areas. Ozone increases radiative forcing, which in turn exacerbates climate harms in national parks and other Class I areas. National parks are significantly threatened by a rapidly warming planet. Impacts range in degree and breadth and include coastal areas affected by rising oceans, deserts experiencing extreme heat events, and alpine regions beleaguered by extended drought.

For example, rising sea levels in Florida's Everglades National Park threaten the mangrove ecosystem that filters saltwater and thereby preserves freshwater wetlands. Rising temperatures and drought in New Mexico's Bandelier National Monument have driven bark beetles to higher elevations, causing high mortality rates to the piñon pines. Rising temperatures in Yellowstone National Park are also killing whitebark pine trees, which translates to reduced chances of grizzly bear survival in Yellowstone because grizzlies rely heavily on whitebark pine seeds as a critical source of nutrition. Warmer temperatures in Great Smoky Mountains National Park could increase ozone levels, further damaging critical tree and plant species. Scientists have linked these and other changes occurring in our national parks directly to climate change.

In 2014, NPS published a study that examined the extent to which 289 parks are experiencing extreme climate changes when compared to the historical records from 1901–2012.[507] Results show that parks are overwhelmingly at the extreme warm end of historical temperatures. The 2014 Parks Study also points to changes in precipitation patterns since 1901. These findings are supported by previous scientific research. Parks that have been experiencing extremely warm and dry climates include Kalaupapa National Historical Park in Hawaii, Mojave National Preserve in southern California, and Lake Mead National Recreation Area in Nevada and Arizona. Parks that have become extremely warm and wet include Cape Lookout National Seashore in North Carolina, Florissant Fossil Beds National Monument in Colorado, and Delaware Water Gap National Recreation Area in New Jersey and Pennsylvania.

The Appalachian National Scenic Trail (ANST) was designated as a unit of the National Park System and the first National Scenic Trail in 1968. The Appalachian Trail follows the hills and valleys of the Appalachian mountain range in the eastern United States. The 2014 Parks

[507] Monahan & Fisichelli (2014) Climate Exposure of US National Parks in a New Era of Change. [hereafter 2014 Parks Study]

Study found the recent mean temperatures on the ANST were ranked as "extreme warm" compared to the historical data set. Further, climate data collected at a northern ANST mid-elevation site in the White Mountain National Forest, where winter recreation is very important to local economies, show that snowpack is disappearing 15 days earlier in the spring and annual snowfall has declined by 69 inches over the time period of 1935-2012.[508]

According to the 2014 Parks Study, species within national parks are experiencing extreme climates, causing changes to plant and animal behavior. For example, temperate tree species in the Great Lakes region appear most sensitive to higher summer temperatures, while white-tailed deer are more sensitive to winter conditions.

Taken together, these data show that welfare effects from climate change—including at least some contributing effect from tropospheric ozone—not only can be "anticipated," but in fact are already occurring in the nation's National Parks and other Class I areas. EPA must address the dire need to reduce direct climate impacts from ozone, in addition to addressing vegetation effects, when setting the standard.

All publicly protected lands are visited for recreation and rejuvenation and are often important wildlife habitat areas. This nexus of sensitive ecological systems with the significant ecosystem services must be weighted in the context of the public welfare.

3. Scientific Consensus Supports a Different Form

a. Support for a Cumulative Seasonal W126 Standard

The scientific foundation supporting the use of a cumulative standard to protect vegetation began in the 1996 review. At that time EPA had a significant amount of science recognizing that the form of the standard used to protect human health was not appropriate for protecting vegetation. In the 2006 review, again in the reconsideration in 2010, and in the current review EPA clearly supports the main assertions that ozone's impact on vegetation is cumulative and that higher ozone concentrations are more important in causing measureable impacts to plants than lower concentrations. This is the foundation for the W126 metric which sums the seasonal ozone exposure and uses a sigmoidal weighting function to weight higher concentrations more than lower levels. EPA has continued to focus on this type of metric as the "most biologically relevant metrics for consideration of O3 exposures eliciting vegetation-related effects. Such a metric has an ''explanatory power'' that is improved ''over using indices based on mean and peak exposure values.''[509] As shown in the record for the current rulemaking, CASAC and EPA's staff have repeatedly and unequivocally recommended adoption of this metric for the secondary ozone standard.

Considering this long standing support of the W126 metric, vetted in two reviews (and one re-consideration) over a nearly 20 year period it is clear that the implementation of this metric is too long delayed. EPA must adopt this metric as the standard instead of the health based metric that is used for the primary health standard.

[508] AMC unpublished data.
[509] ISA sec. 2.6.6.1, at 2–44.

b. 8-hr Form Is Not Protective of the Public Welfare

EPA cannot lawfully or rationally set a national welfare standard based on a metric designed to protect public health. It is arbitrary and unlawful for EPA to propose a welfare standard in the 8-hour form in direct contradiction with its own conclusion that public welfare protection is – as a matter of science - appropriately judged through the use of the cumulative seasonal W126-based metric. EPA has again disregarded CASAC's advice that is founded on a plethora of scientific information and context built in this review and past reviews. This is the very definition of an arbitrary and capricious decision.

Moreover, EPA fails to offer a full scientific (or indeed any) basis for rejecting CASAC's advice on this issue, as the law requires. See part V.C. above. The agency makes no claim that reliance on the 8-hour form will do a better job of protecting welfare than the W126 form, and indeed the record provides no support for such a claim. Nor is there any claim by the agency that some statutorily relevant purpose is served by foregoing the W126 metric – which the agency agrees is more biologically relevant and appropriate. And even if implementation concerns were relevant at the standard-setting stage (and they are not), EPA cites no potential implementation problems as a justification for foregoing the W126 metric

Further, EPA is simply wrong in suggesting that the 8-hour primary form (3-year average of the 4th highest daily 8-hour average) for the ozone secondary welfare standard can adequately substitute for the W126 standard to protect vegetation based on alleged relationships between three year averaged 8-hour values and three year averaged W126 values. CASAC expressly rejected reliance on such comparisons, and EPA fails to fully or rationally explain why CASAC was error on this point. As CASAC stated:

> However, as noted in the CASAC's review of the Second Draft WREA (EPA-CASAC-14-003), the CASAC finds that a W126 level of 15 ppm-hrs may not be similar to the current standard, since the actual approaches that would be used to achieve such a level are likely to be different than those assumed in the HREA air quality scenarios for just meeting the current standard. Specifically, and quoting from our review of the Second Draft WREA:
>
> "The currently reported finding of only small differences in risk between just meeting the current standard and a W126-based level of 15 ppm-hrs must not be interpreted to mean that just meeting the current standard will be as protective as meeting a W126-based standard at 15 ppm-hrs. There are two key factors that must be considered when making this comparison. First, air quality was simulated in the Second Draft WREA based on the magnitude of across-the-board reductions in NOx emissions required to bring the highest monitor down to the target level. Meeting a target level at the highest monitor requires substantial reductions below the targeted level through the rest of the region. This artificial simulation does not represent an actual control strategy and may conflate differences in control strategies required to meet different standards and different targets. As a result, there may be a number of monitors that meet the current

> standard but would not meet an alternative W126 standard. Second, and equally important, the current form of the standard is much less biologically relevant for protecting vegetation than is a seasonal, peak weighted index such as the W126, which was designed to measure the cumulative effects of ozone exposure." [510]

EPA offers no rational response to the above CASAC rationale.

The Policy Assessment likewise found that there was no consistent relationship between 8-hour and W126 levels:

> [W]hile the western sites that are influenced by upwind urban plumes may have increased cumulative seasonal values coincident with increased daily 8-hourpeak O3 concentrations, this analysis indicates that, in sites without such an urban influence (the eastern sites in this analysis), such a relationship does not occur (U.S. EPA, 2013a, section 3.6.3.2). Thus, the lack of such a relationship indicates that **i**n some locations, O3 air quality patterns can lead to elevated cumulative, seasonal O3 exposures without the occurrence of elevated daily maximum 8-hour average O3 concentrations (U.S. EPA, 2013a, section 3.6.3.2). Further, staff notes that the prevalence and geographic extent of such locations is unclear, since as in the last review, there continue to be relatively fewer monitors in the western U.S., including in high elevation remote sites. . . . . …O3 concentrations measured in some rural sites can be higher than those measured in nearby urban areas (U.S. EPA, 2013a,section 3.6.2.2) and the ISA concludes that ''cumulative exposures for humans and vegetation in rural areas can be substantial, and often higher than cumulative exposures in urban areas''(U.S. EPA, 2013a, p. 3–120). These known differences between urban and rural sites suggest that there is the potential for an inconsistent relationship between 8-hour daily peakO3 concentrations and cumulative, seasonal exposures in those areas.

Proposed Rule at 75,344 (emphasis added).

EPA cites[511] an analysis in the PA of design values at monitors for 2001-03 and 2009-11 that purportedly shows that between the two periods during which broad scale precursor reductions occurred, ozone reductions in terms of both metrics were recorded, and there was a fairly strong positive degree of correlation between the two metrics. In support, the notice says:

- in 2009-11, monitors just meeting the current standard (75ppb) had W126 values ranging from less than 3 ppm-hrs to approximately 20 ppm-hrs
- At sites with an 8-hour design value at or below 70 ppb, 3-year W126 values were above 17 ppm-hrs at no monitors, above 15 ppm-hrs at 1 monitor, and above 13-ppm-hrs at 8 monitors in the West and Southwest
- At sites with 8 hour design value at or below 65 ppb, W126 values were above 11 ppm-hrs at no monitors, above 9 ppm-hrs at 3, and above 7 at 9 (majority of which are located in the West and Southwest)

[510] CASAC Letter 2014a at 11 (emphasis added).
[511] Proposed Rule at 75,344-45.

These data do not support a claim of congruence between the primary standards being considered and the W126 options. First, the data show that there were in fact a number of monitors that would meet the various 8-hour standard levels yet violate W126 levels that are within the CASAC and PA ranges. Second, even as to sites where both standards would be met, there is no basis for concluding that there is some fundamental underlying relationship that assures meeting the 8-hour will mean meeting any of the W126 options: indeed the above quotes from the PA and CASAC refute such a suggestion. Finally, the periods examined include the recession of 2008-10: hardly a representative period, as industrial activity and emissions declined.

EPA also cites another analysis in PA that looks at data for 3-year periods back to 2006-08 and indicates that among counties that met the current standard, the number with W126 values above 15 ppm-hrs ranges from fewer than 10 to 24 (these were predominantly in the Southwest region. Again, the analysis fails to show that meeting an 8-hour standard will invariably – or even very likely – meet a W126 standard. It addresses only a comparison with the current (inadequate) 75 ppb standard, it looks only at counties that would have been over 15 ppm-hrs, and it still finds that a number of counties would violate a W126 standard: And it suffers from the same defects cited above.

Additional analysis shows that an 8-hour standard metric would in fact allow a wide spread variation in W126 values. This variability increases as the level of the standard moves away from 60 ppb to higher 8-hour averages. Further, this approach is particularly under-protective in western National Parks and other Class I areas in this region, where the relationship between EPA's target W126 values and the 8-hour metric is least robust (as reported by Wells).

EPA relies in large part on an analysis[512] that compares the W126 to 8-hour[513] form levels to assert that EPA can achieve a level of protection defined by the W126 metric but represented by the 8-hour form. This approach is flawed in both general application and when specific western US National Parks are examined.

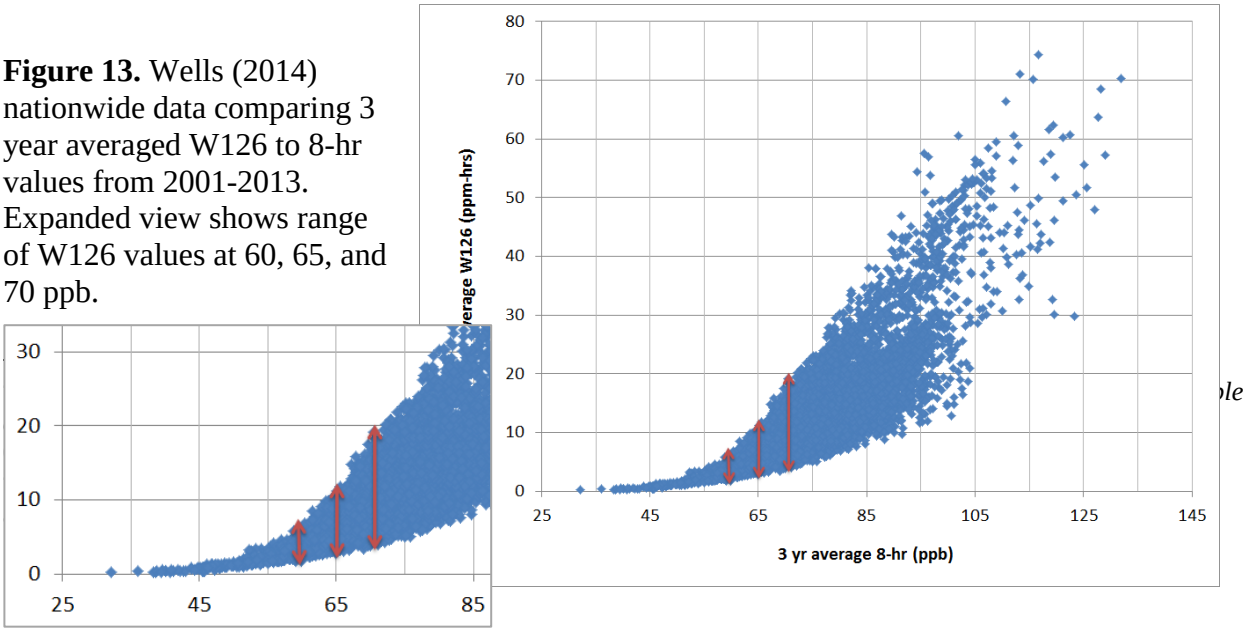

Figure 13. Wells (2014) nationwide data comparing 3 year averaged W126 to 8-hr values from 2001-2013. Expanded view shows range of W126 values at 60, 65, and 70 ppb.

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The relationship is clearly non-linear with significant spread in the data, see Figure 13. Table 25 shows the minimum, maximum, and range in W126 values observed at levels of 60, 65, and 70 ppb from a dataset used in the Wells memo.[514] Particularly noteworthy is the large variation of W126 values for each 8-hour level, with range of 4.9, 8.9 and 15.3. This level of variability shows that the 8-hour metric is simply not an accurate surrogate for W126 levels. And the decree of variability is material and substantial. For example, at the 8-hour level of 70 ppb, some sites have 3 year average W126 values higher than CASAC high end of 15 ppm-hrs and EPA's proposed high end of 17 ppm-hrs. Although the variability is less at an 8-hour level of 65 ppb, the range of W126 levels is a full 8.6 ppm-hrs: equivalent to more than 50% of the high ends of both the CASAC and EPA ranges. And the maximum W126 level at 65 ppb is 11.8 ppm-hrs, far in excess of the 7 ppm-hrs low end of the CASAC and staff ranges. The variability in these ranges is all the more significant considering the conservative approach to calculating the W126, e.g. 3 year average, limited to 12 hr and highest 3 months. For all these reasons, it is arbitrary for EPA to suggest that the Wells data shows that 8-hour form will achieve target W126 based levels of protection.

Table 25. Significant variability in three year average W126 ranges at three year average 8-hour level targets from Wells Memo dataset.

8-hour level (ppb)*	Minimum W126	Maximum W126	Range in W126
60	2.0	6.9	4.9
65	3.2	11.8	8.6
70	3.8	19.1	15.3

*Levels based on ppm values truncated to 3rd decimal place

Wells identified that the relationships between the metrics vary by region stating: "In particular, the Southwest and West regions (i.e., the southwestern U.S.) appear to have higher W126 values relative to their respective 4th max values than the rest of the U.S."[515] Here, we provide some specific analysis of 11 western National Parks included in the Wells dataset that further exemplify the inter-site variability, uncertainties in predicting W126 values, and under-protection of western park lands using the 8-hour standard. We applied linear regressions between the W126 and 8-hour metrics for individual park level data and found significant variability is present when moving from one location to another even among the western region sites.

The parks included were: Chiricahua National Monument, Grand Canyon National Park, Petrified Forest National Park, Death Valley National Park, Joshua Tree National Park, Lassen Volcanic National Park, Mesa Verde National Park, Big Bend National Park, Chamizal National Memorial, Canyonlands National Park, and Great Basin National Park. All of these parks have ozone-sensitive species (see Exhibit 14). At the 60 ppb level two parks exceeded a level of 7 ppm-hrs; Chiricahua National Monument and Joshua Tree NP had W126 values of 9.6 and 10.1 ppm-hrs, respectively. At the levels EPA proposed, 65 to 70, all of the 11 parks exceed 7 ppm-

[514] Provided by B. Wells via email.
[515] B. Wells (2014). Comparison of Ozone Metrics Considered in the Current NAAQS Review, U.S. EPA, *available at* http://www.epa.gov/ttn/naaqs/standards/ozone/data/Wells-2014-CompO3Metrics-EPA-HQ-OAR-2008-0699-0155.pdf.

hrs and 6 out of 11 exceed 15 ppm-hrs, the high end of the range recommended by CASAC, and 1 park (Joshua Tree NP) exceeded the 17 ppm-hrs at an 8-hour standard of 70.

Table 26. The range in W126 values associated with 8-hour targets, calculated from linear fits for 11 National Parks included in the Wells dataset.

8-hour level (ppb)	Minimum W126	Maximum W126	Range in W126	# of parks > W126 level
60	2.5	10.1	7.4	2 parks > 7
65	3.2	11.8	8.6	11 parks > 7 5 parks > 10
70	3.8	19.1	15.3	6 parks >15

CASAC clearly emphasized its opposition to averaging the W126 standard over 3 years pointing to the cumulative nature of ozone impacts and the fact that a 3-year average standard does not protect against damage from high levels in a single year. They recommend EPA focus on the lower end of the 7-15 ppm-hrs range if averaging across 3 years. EPA not only ignores this advice but proposed averaging with an even higher W126 range target.

We examined the annual W126 values at 4 western National Parks compared to EPA's preferred 3-year averaged 8-hour maximum form for the timeframe 2000-2013. These parks were selected because their 8-hour values have been within and just over the range of 60-70 ppb. Those parks include: Grand Canyon National Park, Mesa Verde National Park, Big Bend National Park, and Canyonlands National Park. All 4 parks have ozone-sensitive species present including Ponderosa Pine an important forest tree species for western forests that have both commercial and ecological value[516]. EPA states that 15 ppm-hrs creates 3 percent loss for ponderosa pine.[517]

It is no surprise that the variability in 1 year W126 data is greater than a metric that is averaged over 3 years. Our concern is that this year-to-year variation in W126 is significant in these parks but unaccounted for in EPA's approach. The ranges observed in W126 values on an annual basis would result in significant biomass loss of 6% or greater, see Table 27. In light of the clear recognition that ozone damage is cumulative these findings indicate that EPA's 3 year averaging approach dilutes the effectiveness of setting a protective level. Further, the ranges observed in W126 include levels that represent a median biomass loss of 6% or greater, 3 times as high as CASAC threshold of less than or equal to 2%.

Table 27. Range in annual W126 values at 4 National Parks showing significant under protection

National Park	Standard Deviation Annual W126	Standard Deviation 3 year 8-hour	Range Annual W126	Range 3 year 8-hour	# Ozone Sensitive Species
Big Bend	3.7	3.3	4.4 - 18.0	62 - 71	4
Grand Canyon	4.3	2.2	10.1 - 26.9	68 - 74	12

[516] Greater Flagstaff Forests Partnership, Ecology of Southwestern Ponderosa Pine Forests, *available at* http://www.gffp.org/pine/ecology.htm.
[517] Proposed Rule at 75,324.

Mesa Verde	3.9	2.0	10.7 - 23.4	67 - 73	7
Canyonlands	4.1	1.3	10.3 - 23.6	68 - 72	11

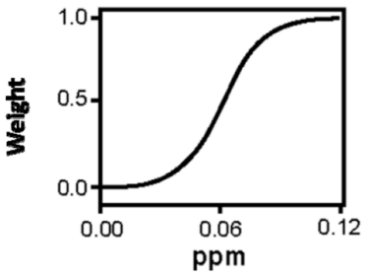
Grand Canyon, Mesa Verde and Canyonlands National Parks all had *minimum* values greater than 9 ppm-hrs, the upper end of the range cited by the National Park Service (Table 27). Further, these sites often see W126 levels greater than 15 ppm-hrs, the upper end of the range identified by CASAC, and 17 ppm-hrs (See Exhibit 14). Unfortunately, Big Bend National Park is seeing an overall rise in ozone reflected in both metrics. The peak W126 value of 18.0 at Big Bend was in 2011 while the peak 8-hr value was in 2013. In addition, 10 out of 14 years exceeded a level of 7 ppm-hrs at Big Bend, while only 6 of these years exceeded a 3 year 8-hour average of the 4th highest daily of 65 ppb. These disconnects are further examples of how the 8-hour standard is ill-suited to protect vegetation. The 8-hour standard does not ensure secondary standards are protective of the values Congress intended they protect.

4. The W126 is the right metric

a. What is the W126 and why is it more relevant to protecting vegetation?

A W126 standard is different from the form upon which the primary standard is built in a number of key ways. This is important to articulate because it explains why the primary metric of the 3 year average of the 4th highest daily 8-hour average is not scientifically defensible as a way to protect vegetation.

The W126 is a weighted value. The actual hourly ozone concentration, in ppm, is assigned a weighted value with higher concentrations counting more than the lowest values. This is based on many scientific studies that found this form best relates to plant damage that can be documented and measured. In this case the weighting formula is sigmoidal. A sigmoidal weighting metric is clearly different from the primary form where *only* the top 4 maximum values over a season determine the standard: here all values included in the summing window greater than 60 ppb are weighted more heavily. The W126 not only accounts for maximum concentrations but also factors in exceptionally high concentrations (e.g. >100 ppb) as it assigns these values the most weight.



It's cumulative (summed). This is appropriate for looking at impacts to vegetation over the growing season because damage can be attributed to the ongoing exposure to the pollutant as the plant moves through its annual growth cycle. Clearly this is different from the primary standard which is based on one peak value (the 4th highest daily 8-hour maximum) and provides little reflection of the growing season, potentially compounding the full effects of ozone exposure.

As we will discuss below it should be recognized that EPA's summation window for W126 (highest 3 month 12-hr sum) is conservative and not protective of full season exposures (the growing season is longer than 3 months) or all environments (mountains with high ozone

overnight and in the early morning). This limitation may leave some plants at higher elevations unprotected during overnight and early morning when ozone can be peak. Further, EPA favors a 3 year averaging of the cumulative exposure that is counter to the CASAC recommendation to use a 1 year metric.

b. The CASAC recommendations

The CASAC clearly concludes that the current secondary standard is not sufficient to protect vegetation from welfare effects of ozone.[518] They recommend changing the form to the W126 at the level of 7 to 15 ppm-hrs. Id. The CASAC "does not recommend the use of the three-year averaging period," finding that use of such an averaging period "is not supported by the available data."[519] They instead advise a single year standard pointing out that this is more biologically relevant, provides more protection to annual crops, protects perennials from the cumulative effects of ozone exposure, and protects against single unusually damaging years.[520] They also state that if the 3-year averaging period is used that the upper limit of 15 ppm-hrs should be reduced.

EPA fails to provide a scientific or reasoned basis for rejecting CASAC's recommendation of a single-year W126 standard. EPA cites variability in ambient ozone concentrations from year to year, but fails to explain why that justifies a 3-year average over a single year standard. As CASAC noted, the variability in a single year W126 standard is reduced by the fact that it is the sum of 3 months of data, so it is not nearly as sensitive to extreme events as an hourly or 8-hour averaging period.[521] Even if there is significant variability, EPA fails to explain why that is relevant to assuring requisite welfare protection, and if so why the 3-year average approach would be more effective at protecting public welfare. EPA further asserts that the PA found greater significance for effects associated with multiple-year exposures, but that PA observation was referring to potential carry over effects from one year to the next. It does not show that a single year metric would provide inadequate protection, or that a 3-year average would provide better protection. In fact, the PA finds that use of 3-year averages may lead to underestimation of RBL.[522] EPA further asserts that CASAC's concern about protecting against adverse effects associated with a single year's exposure "can be addressed through use of a three-year average metric, chosen with consideration of the relevant factors."[523] But EPA does not explain how such an approach can address CASAC's concern, nor does EPA commit to adopting such an approach. EPA also asserts that the Administrator recognizes greater confidence in judgments related to public welfare impacts based on a three-year average metric, but fails to explain why this is so, and fails to cite scientific evidence in support. Finally, EPA disregards CASAC's advice that if a 3-year average is used, it should be set at a lower level than a single-year standard to protect against single unusually damaging years that will be obscured in the average.[524] EPA offers no explanation for failing to follow CASAC's advice on this score. Not only does EPA fail to commit to setting such a lower level, but it proposes a range with a

[518] *E.g.*, CASAC Letter 2014a at iii.
[519] *Id*. 13.
[520] *Id*.
[521] *Id*. at 13.
[522] Proposed Rule at 75338/3.
[523] *Id*. 75347/2.
[524] CASAC Letter 2014a at 13.

high end (17 ppm-hrs) that is actually higher than the range recommended by CASAC for a 1-year W126 standard.

For all the foregoing reasons, EPA's rejection of CASAC's advice for adoption of a single-year W126 metric is arbitrary and violative of EPA's duty to adequately justify a departure from CASAC's recommendation.

Of note, CASAC advised that levels above 15 ppm-hrs should not be options for the secondary standard. By way of example, CASAC noted that at 17 ppm-hrs, the median tree species has an "unacceptably high" loss of tree biomass (6%). Further, they state that a level of "7 ppm-hrs is protective of ecosystem services" as it is the only level in which the median loss for the tree species examined is less than or equal to 2%.

c. A 12-hr, maximum 3 month W126 form of the standard is conservative

In selecting the level of the W126 to protect public welfare, the EPA Administrator must consider that a form set as a 12 hour/highest 3 month sum, provides only a conservative level of protection due to under estimating true exposure to vegetation. First limiting to only a 3 month summing window is clearly not representative of the full growing season and overlapping elevated ozone concentrations and therefore is only a partial account of the ozone exposure. Second, as we describe in depth below, by limiting to a 12 hour sum window many mountain environments are under protected because the standard fails to include overnight/early morning exposures which can be high in mountains.

To fully protect vegetation from cumulative ozone and consideration of Class I areas that have peak ozone concentrations overnight, a full 24 hour standard is needed. The 2013 ISA recognizes the issue of higher nighttime concentrations stating: "Persistently high O3 concentrations observed at many of the rural sites investigated here indicate that cumulative exposures for humans and vegetation in rural areas can frequently exceed cumulative exposures in urban areas."[525] Yet, EPA disregards overnight hours stating in the WREA that "Since plant and tree species are not photosynthetically active during nighttime hours, only O_3 concentrations observed during daytime hours (defined as 8:00 AM to 8:00 PM local time) were included in the summations."[526] This is incorrect. A number of studies[527] have shown that cumulative ozone exposure reduces stomatal control, amplifies water loss, and reduces tree growth. For instance, McLaughlin et al. (2007a) discuss that cumulative ozone exposure, including at nighttime, contributes to the physiological changes observed in tree species at mid-elevation locations in Great Smoky Mountain National Park. McLaughlin et al. shows evidence that ecosystem wide

[525] *See* ISA page 107.
[526] *See* U.S. EPA (2014). Welfare Risk and Exposure Assessment for Ozone, 4-6 [hereinafter WREA].
[527] *See e.g.*, S.B. McLaughlin et al. (2007). Interactive effects of ozone and climate on tree growth and water use in a southern Appalachian forest in the USA, *New Phytologist*, 174: 109-124; S.B. McLaughlin et al. (2007). Interactive effects of ozone and climate on water use, soil moisture content and streamflow in a southern Appalachian forest in the USA, *New Phytologist*, 174: 125-136; N.E. Grulke et al. (2004). Stomata open at night in pole-sized and mature ponderosa pine: implications for O3 exposure metrics, *Tree Physiology*.

impacts occur from cumulative ozone exposure detecting a reduction in late season stream flows from a forested watershed.[528]

Sensitive species and elevated nocturnal ozone exposures co-occur. Of the 267 National Parks that are known to have ozone sensitive plants 226 parks have at least one species that has been documented to show nocturnal conductance (*Alnus rugosa, Populus tremuloides, Pinus ponderosa, Pinus radiata, Fraxinus pennsylvanica, Liriodendron tulipifera* and *Prunus serotina*). Further, we provide evidence of the nocturnal ozone exposures for a number of National Parks and other protected lands at both high and mid-elevation monitoring sites in Table 28. While we understand that other factors, such as turbulence, are important for ozone flux into plants, there are studies that have demonstrated ozone uptake and injury from nighttime exposures.[529] We believe the following combined factors should be considered by the Administrator, reflecting how a 12-hr only standard is only a conservative estimate of ozone exposure:

- Many Class I areas have ozone sensitive species that also exhibit nocturnal conductance
- High overnight ozone levels can coincide with the presence of these species
- Ozone exposure can reduce some plants ability to control stomatal opening and closing and their overall response rate to stress
- The main anti-oxidant defensive compound, ascorbate, is produced largely in daytime, due to photo dependent enzymatic activity. As it is depleted in late afternoon and into the night, it would leave plants less protected from nighttime and early morning elevated ozone concentrations

The ozone exposures are often significant for mountain sites, sites with daylight bringing rapid increases to mid-elevations as the overnight boundary layer breaks up, mixing ozone laden air to lower elevations and in evening as the boundary layer reforms and ozone that was formed over the day is transported to rural montane areas. Damage from ozone may be significant for mid-elevation area that see dramatic increases in morning time ozone levels from downward mixing of pollution aloft in combination in with mid-day local ozone production and evening transport events. The importance of the timing of elevated ambient ozone levels in relation to diurnal stomatal conductance and defensive anti-oxidant production has been discussed in the literature.[530]

[528] S.B. McLaughlin et al. (2007). Interactive effects of ozone and climate on water use, soil moisture content and streamflow in a southern Appalachian forest in the USA, *New Phytologist,* 174: 125-136

[529] Winner et al. (1989). Plant responses to elevational gradients of O3 exposure in Virginia. Proc. Natl. Acad. Sci. USA Ecology Vol. 86 pp. 8828-8832; N.E. Grulke et al. (2004). Stomata open at night in pole-sized and mature ponderosa pine: implications for O3 exposure metrics, *Tree Physiology*; W.J. Massman (2004). Toward an ozone standard to protectvegetation based on effective dose: a review of deposition resistances and a possible metric, *Atmospheric Environment* 38: 2323–2337.

[530] R.L. Heath et al. (2009). Temporal processes that contribute to nonlinearity in vegetation responses to ozone exposure and dose, *Atmospheric Environment,* 46: 2919-2928; R.C. Musselman et al. (2006). A critical review and analysis of the use of exposure- and flux-based ozone indices for predicting vegetation effects, *Atmospheric Environment*, 40: 1869–1888.

Table 28. Average % underestimation of monthly W126 12-hr summation window compared to 24-hr by site. Includes 18 data points the months April – Sept. in the years 2006-2008. Original data from AQS & NPS, calculations by the Appalachian Mountain Club.

LOCATION	ELEVATION (M)	AVERAGE % UNDERESTIMATION (RANGE)	FEDERAL OR STATE PROTECTED LANDS
Mt. Washington Base (Camp Dodge)	452	**25** (4 – 48)	White Mountain NF/ Appalachian Trail, 2 Class I Areas
Mt. Washington Summit	1910	**55** (47 – 65)	White Mountain NF/ Appalachian Trail, 2 Class I Areas
Acadia- Cadillac Mtn.	466	**45** (26 – 58)	Acadia NP, Class I Area
Whiteface Base	625	**55** (41 – 67)	Adirondack State Park
Whiteface Summit	1480	**59** (47 – 71)	Adirondack State Park
Greylock Mountain[a]	1140	**48** (39 – 57)	Appalachian Trail
Blue Ridge Parkway-RO	675	**9** (1 – 23)	Blue Ridge Parkway
Blue Ridge Parkway-75	987	**8** (1 – 18)	Blue Ridge Parkway
Blue Ridge Parkway-FP	1585	**62** (49 – 75)	Blue Ridge Parkway
Shenandoah Big Meadow	1073	**50** (42 – 56)	Shenandoah NP, Class I Area
GSM Clingman's Dome	2021	**57** (48 – 64)	Great Smoky Mtn NP, Class I Area
GSM Look Rock	793	**48** (42 – 55)	Great Smoky Mtn NP, Class I Area
GSM Cades Cove	564	**14** (3 – 37)	Great Smoky Mtn NP, Class I Area
Rocky Mountain Long's Peak	2743	**29** (21 – 43)	Rocky Mountain NP, Class I Area
Sequoia and Kings Canyon Ash Mountain	457	**20** (8 – 29)	Sequoia and Kings Canyon NP, Class I Area
Sequoia and Kings Canyon Lower Kaweah	1890	**28** (21 – 44)	Sequoia and Kings Canyon NP, Class I Area
Crestline[b]	1387	**23** (11 – 29)	San Bernardino National Forest
Yosemite Turtle Dome	1605	**36** (27 – 48)	Yosemite NP, Class I Area

[a] Greylock Mountain data missing April for 2007-2008 [b] Crestline data includes 2007-2009

5. A Standard of 75 ppb Is Not Protective of the Public Welfare

In considering the protectiveness of the current standard we are very encouraged that the Administrator "recognizes the appropriateness and usefulness of the W126 metric."[531] We also strongly agree with the Administrator's opinion that the current standard of 75 ppb is not requisite to protect the public welfare and that revision is needed.[532] The Administrator appropriately makes this judgment with particular attention to the sensitive vegetation and ecosystems in Class I areas and other public lands providing similar public welfare benefits. We also echo the Administrators reliance on the strong science base evidence around impacts to tree growth as well as the increased protection for carbon storage and other growth related effects.

6. The Secondary Standard Must Be Set at a Level No Higher Than 7 ppm-hrs

a. Visible Foliar Injury

EPA needs to protect against foliar injury in plants that benefit most with W126 values between 7 and 9 ppm-hrs, to protect the beauty and vitality of our National Parks and other public recreation lands. This level of protection is demonstrated in the WREA FIA/FHM analysis. Wetlands, which serve as important wildlife habitat, provide ecological diversity to landscapes, and often serve as important filters for soil contaminants, are especially at risk from the stress of foliar damage from ozone.

Foliar injury due to ozone has long been used as a bioindicator of pollutant exposure and is a sign of plant stress as it signifies cellular death (See Exhibit 15). Bioindicator species are selected because of their sensitivity to ozone allowing for a visible evaluation of an area's exposure. Foliar injury, with regard to indicating plant stress, is a public welfare impact as it demonstrates that the vegetation and ecosystem are experiencing ozone induced stress. Even in plants that don't show actual injury, there is likely stress because they may have to either close stomates to protect against cell death which limits photosynthesis, or use carbon based antioxidant reserves to counter the ozone. While stress does not necessarily indicate that the tree or plant will actually be damaged the cumulative ozone stress over time, and the likely additional environmental stresses caused by air pollution, must be considered. Examples of likely co-occurring air pollution impacts include: short-term and long-term impacts of acid deposition[533] , excess nitrogen inputs[534], insect infestations[535], and increased extreme precipitation events

[531] Proposed Rule at 75335.
[532] *Id*. at 75336.
[533] N. Duarte et al. (2013). Susceptibility of Forests in the Northeastern USA to Nitrogen and Sulfur Deposition: Critical Load Exceedance and Forest Health, *J. Water, Air, & Soil Pollution,* 224; G.E. Likens & D.C. Buso (2012).. Dilution and the Elusive Baseline. *Environ. Science & Technology*, 46(8): 4382-4387.
[534] C.M. Clark, et al. (2013). Estimated losses of plant biodiversity in the United States from historical N deposition (1985–2010), *Ecology*, 94:1441–1448.
[535] K.S. Knight et al. (2013). Factors affecting the survival of ash (Fraxinus spp.) trees infested by emerald ash borer (Agrilus planipennis) http://dx.doi.org/10.1007/s10530-012-0292-z

caused by climate change (including damaging icing events, floods, and increased frequency and severity of drought[536]).

Foliar injury also has another specific public welfare impact: its obvious degradation of aesthetics especially on public lands which are visited for sightseeing and recreation and are valued for beauty and healthy ecosystems. A recent summary of National Parks visitor surveys[537] found that 88% and 90% of respondents found clean air and scenic views, respectively, to be extremely important or very important. Both features were most often in the top 2 ranked attributes that visitors value.

EPA wrongly asserts there is "a lack of guidance for federal land managers regarding what spatial scale or degree of severity of visible foliar injury is considered sufficient to trigger protective action for O3 sensitive AQRVs."[538] The National Park Service has provided guidance in a 2011 document[539] that it views W126 exposures greater than 7 ppm-hrs to represent moderate to major impacts on ozone-sensitive vegetation (including foliar injury). The Park Service based this view in part on the findings of an expert workgroup that a W126 of 5‑9 ppm-hrs would protect plants in natural ecosystems against foliar injury. More importantly, in comments to EPA, the National Park Service has repeatedly called for a secondary standard in the 7 to 9 ppm-hrs range based in part on concerns about ozone-induced foliar injury[540]. Thus EPA is simply wrong in asserting that there is a lack of guidance from federal land managers regarding the degree of ozone-induced foliar injury warranting protective action.

EPA's proposal is unprecedented in that it eliminates the use of foliar injury to inform the level of W126 that would be protective under a national welfare standard. EPA wrongly asserts in its proposal that CASAC provided no guidance on foliar injury benchmarks[541] as they did for biomass loss and crop yields. This is false. CASAC frames it's overall recommendation of 7-15 ppm-hrs on the basis of *all* the evidence of casual effects to vegetation including the foliar injury information.[542] In CASAC's comments on the second draft Policy Assessment they explicitly state: "A level *below 10 ppm-hrs is required to reduce foliar injury.*" (emphasis added).

This is consistent with the CASAC committee's consensus comments on the draft WREA where they clearly point to the significance of the USFS FHM/FIA ozone biomonitor data and the level of 10 ppm-hrs as an upper bound for foliar injury.

> Figures 7-9 and 7-11, showing the cumulative number of biosites with any injury as a function ofW126, are very clear and effective in communicating the risk due to ozone.

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[536] W.R. L. (2013). Consequences of widespread tree mortality triggered by drought and temperature stress, *Nature Clim. Change*, 3:30–36 http://dx.doi.org/10.1038/nclimate1635

[537] National Park Service (2013). Natural Resource Stewardship and Science, *available at* https://psu.uidaho.edu/files/vsp/reports/5003_NPS_rept_2013.pdf.

[538] National Park Service (2011). Technical Guidance on Assessing Impacts to Air Quality in NEPA and Planning Documents, *see* 13-14, Guidance *available at* http://www.nature.nps.gov/air/Pubs/pdf/AQGuidance_2011-01-14.pdf

[539] *Id.*

[540] NPS Letter N3615 (2301) Oct. 5th, 2007, NPS Letter N3615 (2350) March 20th, 2014.

[541] Proposed Rule at 75,334

[542] CASAC Letter 2014a at iii.

This analysis also reveals a change in the E-R slope near 10 ppm-hrs. However, this slope change is not a threshold for no injury. Based on this E-R slope change, 10 ppm-hrs is a reasonable candidate level for consideration in the WREA, along with other levels.".[543]

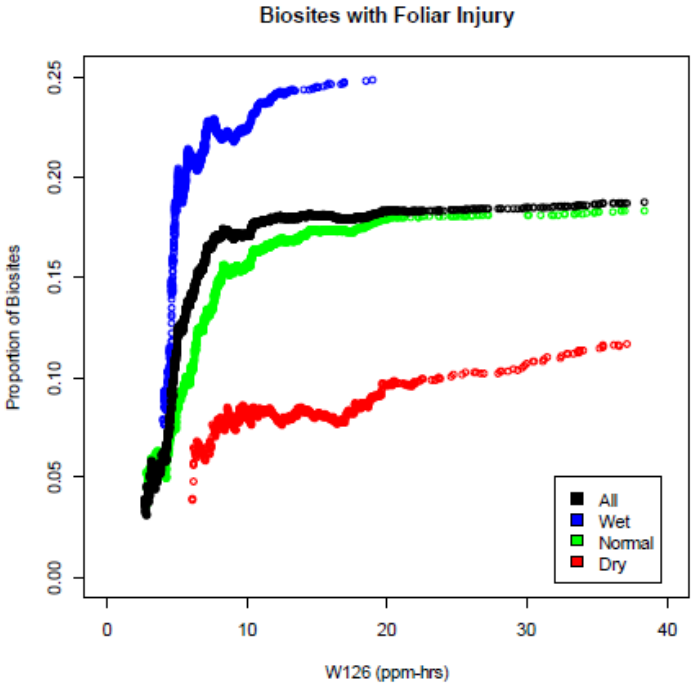

Figure 14. EPA WREA figure showing the cumulative proportion of sites with foliar injury present, by moisture category. Note that reductions in the proportion of sites, in each moisture category, largely occur below a W126 of 10 ppm-hrs.

[543] Letter from CASAC Chair Dr. H. Christopher Frey to U.S. EPA Administrator Gina McCarthy, re: CASAC Review of the EPA's *Welfare Risk and Exposure Assessment for Ozone (Second External Review Draft)* (EPA-CASAC-14-003), 7, June 18, 2014.

EPA relies on a misguided rationale that the variability of foliar injury response and the difficulty in identifying an alternative level of the standard make it too challenging to assess. EPA states: "Thus, while the PA recognizes visible foliar injury as an important O3 effect which, depending on severity and spatial extent may reasonably be concluded to be of public welfare significance, most particularly in nationally protected areas such as Class I areas, it additionally recognizes the appreciable variability in this endpoint, which poses challenges to giving it primary emphasis in identifying potential alternative standard levels." Variability should in no way discount the positive observations of foliar injury and its relationship with a W126 level. A recent example is Kohut et al. 2012 who found that the W126 3-month form was a consistent predictor of ozone foliar injury on cutleaf coneflower (*Rudbeckia laciniata var. ampla*) over 5 years in Rocky Mountain National Park. [544]

EPA's review of the FIA/FHM dataset provides a clear relationship between the W126 and foliar injury when sites that showed positive injury are sorted by soil moisture. This relationship is significant in that it allows land managers to recognize the W126 levels that can cause foliar damage that is not only unsightly to the public but also indicates plant stress. The high variability in foliar injury occurrence should not negate the positive findings where a robust relationship with W126 can be found. These positive findings necessitate action by EPA to protect the public welfare from the impacts that they represent.

To the extent EPA is suggesting that it can deem visible foliar injury as not being an adverse effect on welfare, such a suggestion is unlawful and arbitrary. EPA itself has repeatedly identified foliar injury as adverse effects in prior reviews.[545] In its 2010 reconsideration proposal, EPA stated: "In an area such as a national park, where visitors come in part for the aesthetic quality of the landscape, the Administrator recognizes that visible foliar injury incidence is an important welfare effect which should be considered in determining an appropriately protective standard level.[546] On the same page, EPA also said the 1996 consensus workshop findings (which, among other things, recommended levels of protection against foliar injury) should be given substantial weight.[547] And the 2010 proposal went on to say:

> The Administrator also believes that in order to preserve wilderness areas in an unimpaired state for future generations, **she must consider a level that affords substantial protection from known adverse O3-related effects of biomass loss and foliar injury on sensitive tree species,** as well as a level that takes into

[544] R. Kohut (2012) Foliar Ozone Injury on Cutleaf Coneflower at Rocky Mountain National Park, Colorado, *Western North American Naturalist*, 72(1)" 32-42.
[545] *See, e.g.,* 73 Fed. Reg. 16,496/2,: "[T]he Administrator finds that evidence that has become available in this review demonstrates the occurrence of adverse vegetation effects at ambient levels of recent O3 air quality, and that evidence and exposure- and risk-based analyses indicate that adverse effects would be predicted to occur under air quality scenarios that meet the current standard, taking into consideration both the level and form of the current standard. Ozone exposures that would be expected to remain after meeting the current secondary standard are sufficient to cause visible foliar injury and seedling and mature tree biomass loss in O3-sensitive vegetation."; *id*. at 16490/3 ("Staff Paper concluded that the current standard continues to allow levels of visible foliar injury in some locations that could reasonably be considered to be adverse from a public welfare perspective").
[546] 75 FR at 3,025.
[547] *Id*.

> account potential ‘‘anticipated’’ adverseO3-related effects, including effects that result in continued impairment in the year following O3 exposure (*i.e.*, carryover effects).[548]

Further, CASAC specifically identified visible foliar injury as an adverse welfare effect, as did the PA.[549] EPA offers no reasoned basis for rejecting these conclusions.

EPA asserts[550] that its refusal to protect against visible foliar injury is justified in light of "the significant challenges in judging the extent to which such effects should be considered adverse to the public welfare, in light of the variability and the lack of clear quantitative relationship with other effects on vegetation, as well as the lack of established criteria or objectives that might inform consideration of potential public welfare impacts related to this vegetation effect." Not only do these assertions lack rational support for the reasons specified above, but the rationale they reflect is essentially the same as the one rejected by the Court in *American Farm Bur. Fd. v. EPA*, 559 F.3d 512, 530 (D.C. Cir. 2009). There, EPA refused to specify a level of welfare protection for visibility because of the allegedly subjective nature of the value. The Court held EPA's refusal was unlawful and arbitrary:

> The EPA's assertion that it need not determine what level of visibility protection is requisite to protect the public welfare fails under the plain language of the statute. The CAA provides: "Any national secondary ambient air quality standard *shall specify a level of air quality* the attainment and maintenance of *which ... is requisite to protect the public welfare* from any known or anticipated adverse effects...." 42 U.S.C. § 7409(b)(2) (emphases added). The 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. Furthermore, the failure to set any target level of visibility protection deprived the EPA's decision making of a reasoned basis. Because the EPA failed to identify any target level, we need not decide whether it was reasonable for the agency to reject the target recommended by the Staff Paper and the CASAC because it was based on uncertain subjective evidence

Under the same reasoning, EPA cannot forego identifying a level of protection against foliar injury that is requisite to protect against adverse effects on public welfare, and setting a level of air quality to provide that level of protection – notwithstanding EPA's claims of difficulty in identifying such levels of protection.

Finally, CASAC and the PA were both clear that W126 values below 10 ppm-hrs are required to reduce the number of sites showing such injury – there was no material uncertainty cited on this point. CASAC further stated that the 10 ppm-hrs figure was based on its scientific

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[548] *Id.* at 3,025-26 (emphasis added)

[549] Letter from CASAC Chair Dr. H. Christopher Frey to U.S. EPA Administrator Gina McCarthy, re: CASAC Review of the EPA's *Welfare Risk and Exposure Assessment for Ozone (Second External Review Draft)* (EPA-CASAC-14-003), 7, June 18, 2014; Policy Assessment at ES-8, 5-51, 5-52.

[550] Proposed Rule at 75349.

judgment.[551] Accordingly, EPA must adopt a standard at least as protective as 10 ppm-hrs unless EPA can articulate a fully sufficient scientific basis for rejecting CASAC's advice. See Part V.C. above. EPA has failed to articulate any such basis in the record here.

b. Tree biomass; growth and productivity

CASAC has identified a 2% biomass loss as "an appropriate scientifically based value to consider as a benchmark of adverse impact for long-lived perennial species such as trees, because effects are cumulative over multiple years."[552] EPA does not offer a scientifically based or otherwise rational basis for concluding otherwise. Further, as CASAC found the level of 7 ppm-hrs is the only level analyzed for which the relative biomass loss for the median tree species is less than or equal to 2%.[553] Accordingly, EPA must protect against tree biomass loss greater than 2% annually by adopting a W126 level of 7 ppm-hrs to protect forests and ecosystems, especially in Class I areas. The WREA and other material in the record provides clear evidence that tree biomass loss increases with increasing W126 levels above 7 ppm-hrs based on concentrations exposure response curves. Further, sensitive tree species, that experience significant biomass loss when exposed to ozone, are integral to our nation's forest and even urban/suburban landscaping and greenways.

EPA incorrectly reasons that an upper level of the W126 for protecting tree biomass loss, as relative biomass loss, should be 17 ppm-hrs because this aligns with CASAC comment that >6% biomass is unacceptably high and because 17 and 15 (CASAC recommended upper limit) have the same median percent loss (5.3%). Further, EPA purports that there is little difference from 17 down to 9 ppm-hrs because there is no change in the number of tree species at or below 2% biomass loss, e.g. 5 out of 11. However, there are some very serious deviations between EPA's rationale and that of CASAC.

1. CASAC % loss judgments are based on an annual form of the W126 standard, yet EPA is proposing a 3 year averaging.
2. CASAC puts the most weight on the median % biomass loss, values that DO CHANGE dramatically over the same range that EPA says there is no difference (with 5/11 species).
3. CASAC clearly supports 7 ppm-hrs as appropriate to protect trees from significant biomass loss, at no greater than 2%, while EPA wrongly construed CASAC comment to indicate that 6% biomass loss is acceptable. Moreover, CASAC's rejection of 17 ppm-hrs as the top of the range was not based solely on the assumption of a 6% biomass loss at that level. CASAC also relied on evidence of adverse effects at 10 and 7 ppm-hrs.[554]

One CASACs' member provides more context in his individual comments where he states:

> "We favor using a measure of central tendency of the data, specifically the median across species (the green line in Fig. 5-2). This analysis provides the median of best available estimates within each species, and the median across species with all

[551] CASAC Letter 2014a at 15.
[552] *Id*. at 14.
[553] *Id*.
[554] *Id*. at 12.

species treated equally. Table 6-1 presents the RBL results for individual species for different levels of W126. This table demonstrates that a range of 7 ppm-hrs to 15 ppm-hrs will protect against RBL of 2% for at least 5 of the 12 species. We do not consider a value of 17 ppm-hrs from Table 6-1 because even though only 5 of 12 tree species are estimated to have relative biomass loss of 2 percent or less at this level, *the median species has relative biomass loss of 6.0 percent, which is unacceptably high*. With compounding over the harvest cycle or life span of these species, this will result in considerably greater cumulative RBL as discussed above. For the more sensitive tree seedlings, a value closer to the lower end of the range (7 ppm-hrs) would be more appropriate. The level of 7 ppm-hrs is the only level analyzed for which the relative biomass loss for the median tree species is less than or equal to 2 percent."[555]

EPA must not misconstrue the reasoning and position of CASAC when making a final decision on the secondary standard. Instead the Agency must consider the science squarely and set a W126 secondary standard to 7 ppm-hrs to protect tree growth and health and thereby public welfare.

c. Increase protection of carbon storage; benefits to climate change

The Administrator has specifically pointed to the fact that reduction of carbon storage is important public welfare harm from ozone, providing an important rationale for the need to revise the current standard.[556] This is supported in the recent ISA that cites consistent findings of carbon loss with ozone damage.[557] EPA estimates that transitioning to a W126 of 7 ppm-hrs would reduce carbon dioxide equal to taking 11 million cars off the road via increased carbon sequestration[558]. EPA qualifies this estimate as likely low, as their analysis did not include the forests on public lands, therefore the climate benefits are likely much greater. Given EPA's identification of the importance of ozone's negative impacts on vegetative carbon storage, such an impact plainly qualifies as an adverse welfare effect. Yet EPA does not even attempt to protect against the degree of such effects predicted to occur at levels below 13 ppm-hrs (and its proposal does not even assure protection at the 13 ppm-hrs level, given the agency's proposed use of 8-hour standards that – as demonstrated above- are not as protective as the W126 levels under consideration.).

EPA fails to rationally explain its failure to identify or provide requisite protection levels against this important welfare impact. At one point, the agency seeks to minimize the significance of additional carbon storage that would be provided with more protective W126 levels. EPA asserts that as a percent of the estimated carbon storage under the current standard, estimates of storage over 30 years under the 15, 11 and 7 ppm-hrs scenarios are less than 0.1% (13 MMtCO2e), just under 1% (593 MMtCO2e) and under 2% (1,600 MMtCO2e).[559] But by EPA's own measures, 1,600 MMt over 30 years (estimated at the 7 ppm-hrs levels) is actually

[555] *Id*. at 14 (emphasis added).
[556] Proposed Rule at 75315/2, 75321/1, 75336.
[557] *Id* . at 75,315/1.
[558] WREA.
[559] Proposed Rule at 75,325/2.

very significant. By comparison, EPA's projections for its Clean Power Plan (i.e., setting standards of performance for carbon emissions from existing power plants under CAA §111(d) are that it will reduce CO2 emissions by roughly 300-500 MMt per year.[560] So the carbon reduction benefits of a 7 ppm-hrs ozone standard over 30 years would be very roughly equal to about 4 years of the benefits from EPA's Clean Power Plan. EPA can hardly claim that such benefits are insignificant from a public welfare perspective.

7. A Range of 13 to 17 ppm-hrs Does Not Assure Requisite Protection

To justify its proposal to set the secondary standard within the same range as the primary, EPA uses a range of 13- 17 ppm-hrs as a basis for comparison. Use of such a range does not assure requisite protection against the adverse effects on public welfare for the following reasons:

i) The range is contrary to the advice of both CASAC and PA that the lower bound of the range be 7 ppm-hrs; and contrary to CASAC's advice that the upper bound be no higher than 15 ppm-hrs.[561] EPA fails to fully or rationally explain its rejection of CASAC and PA advice on the range. EPA offers an unsupported claim that there is greater uncertainty as to extent to which estimates of benefits in terms of ecosystem services and reduced effects on vegetation at lower levels might be judged significant to public welfare. But CASAC expressly cautioned against over-emphasis on uncertainty at levels below 15 ppm-hrs, finding that plant injury is clearly observed at and well below that level.[562] CASAC also expressly found that "there is quite a lot of certainty in estimates of biomass loss for forest tree seedling species for which E-R functions have been developed.[563] It further found that "based on scientific judgment of CASAC…7 ppm-hrs is protective of relative biomass loss for trees….and is protective of ecosystem services."[564] And CASAC found that "[f]or the more sensitive tree seedlings, a value closer to the lower end of the range (7 ppm-hrs) would be more appropriate. The level of 7 ppm-hrs is the only level analyzed for which the relative biomass loss for the median tee species is less than or equal to 2 percent."[565] EPA provides no scientific or reasoned basis for differing from these scientific CASAC and PA findings.

ii) EPA cites[566] the WREA's alleged showing of "relatively small additional benefits and increased uncertainty with the ecosystem services estimates" in the scenarios at and below 11 ppm-hrs. EPA claims that the PA observes similarity in the number of species with less than 2% RBL across the range from 17 to 9 ppm-hrs. The agency also claims that a similar number of species have RBL estimates below 5% for values of 13 and 11. In reality, there are significant differences in the number of species with RBL below 5% at different W126 levels. Table 29[567]

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[560] See 79 Fed. Reg. 34,830, 34,931-33 (2013).
[561] EPA also wrongly suggests that CASAC purportedly endorsed 13 ppm-hrs, when in fact CASAC simply gave it as an example of how EPA might translate a one-year W126 standard of 15 to a 3-year average standard: CASAC did not in fact endorse such an approach: It was absolutely clear that CASAC advised a single year standard.
[562] CASAC Letter 2014a at 12.
[563] *Id*. at 5.
[564] *Id*.
[565] *Id.* 14.
[566] Proposed Rule at 75,349/2.
[567] *Id*. at 75430)

shows 5 species with RBL less than 5% at 17 ppm-hrs; 8 at 11 ppm-hrs; and 10 at 9 ppm-hrs (double the number at 17). Even as between 13 and 11 ppm-hrs, there is 1 additional species showing less than 5% RBL at 11: And given that the analysis only looked at 11 species, one additional species represents 9% of the species – hardly trivial. Further, discounting the impact on even just one species conflicts sharply with EPA's stated intent to focus particular significance on adverse effects that occur on sensitive species.[568]

Likewise, EPA asserts that there is similarity in the number of species with less than 2% RBL across the W126 range from 17 to 9 ppm-hrs. While it's true the same number of species (5) suffer less then 2% RBL throughout that range, it's also the case that the median species has more than double the loss at 17 than it does at 9 (5.3% versus 2.4%) – and throughout the 9-17 range there is a steady increase in the medial species loss.[569] It is therefore arbitrary to imply that 17 provides equivalent protection.

iii) EPA wrongly suggests (79 Fed. Reg. at 75348/3) that CASAC rejected 17 ppm-hrs as the top of the range only because it assumed that the RBL at that level would be 6% - a number that was later reduced to 5.3% in the final PA. But CASAC's rejection of 17 was not based solely on the assumption of 6% biomass loss at that level. CASAC also relied on adverse effects that occur at 10 and 7 ppm-hrs.[570]

iv) EPA also rejects CASAC and PA recommendations for 7 ppm-hours as the lower end of the range on the grounds that CASAC's recommendation was a policy one, that there is greater uncertainty on the extent to which estimated benefits in terms of ecosystem services, and reduced effects on vegetation at lower ozone exposures, might be judged significant to public welfare, and on grounds of alleged relatively small additional benefits in ecosystem services at lower levels.[571] These assertions are baseless. CASAC expressly said its recommended range was scientifically based.[572] These recommendations are based on scientific evidence of adverse effect associated with the presence of ozone in ambient air. Note that these levels are based on an annual form of the standard. In reaching its scientific judgment regarding the indicator, form, averaging time, and range of levels for a revised secondary standard, the CASAC has focused on the scientific evidence for the identification of the kind and extent of adverse effects on public welfare."). Because CASAC's advice was expressly based on scientific conclusions, EPA must provide more than assertions of uncertainty to depart from CASAC's judgment: It must provide a full and reasoned scientific rationale: The agency has failed to do so here.

EPA also has no reasoned basis for claiming greater uncertainty as to welfare benefits at lower levels: As noted above, CASAC expressly cautioned against overstating such uncertainty, found "quite a lot" of certainty in the estimates of biomass loss, and expressly relied on those estimates to recommend 7 ppm-hrs as the low end of the range.

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[568] Proposed Rule at 75,348/1, 2.
[569] *Id.* at 75,340.
[570] CASAC Letter 2014a at 12.
[571] Proposed Rule at 75,349/2.
[572] *E.g.*, CASAC Letter 2014a at iii, 14 ("In our scientific judgment, it is appropriate to identify a range of levels of alternative W126-based standards that includes levels that aim for not greater than 2% RBL for the median tree species."); *id*. at 15 ("(3) the level of the standard should be between 7 ppm-hrs and 15 ppm-hrs.

Finally, EPA's claim of relatively small additional benefits at lower W126 levels is refuted in subparagraph d.ii above.

8. EPA's Proposal Fails to Specify Requisite Levels of Vegetation Protection as Required by the Act

The Act requires EPA to "specify a level of air quality the attainment and maintenance of which … is requisite to protect the public welfare from any known or anticipated adverse effects." 42 U.S.C. §7409(b)(2). The *Farm Bureau* Court held that this language requires EPA to first identify the requisite level of protection for the affected welfare value (there, visibility), and then set the secondary NAAQS to achieve that level of protection. 559 F.3d at 529-30. "EPA's failure to identify such a level when deciding where to set the level of air quality required by the revised secondary … NAAQS is contrary to the statute and therefore unlawful. Furthermore, the failure to set any target level of visibility protection deprived the EPA's decisionmaking of a reasoned basis." *Id.* 530.

In reviewing EPA's 2008 ozone NAAQS decision, the D.C. Circuit held that EPA had again failed to comply with §7409(b)(2) as construed in *Farm Bureau*. There, EPA adopted a secondary standard identical to the primary without specifying a level of protection of vegetation requisite for public welfare. The Court said:

> EPA's explanation for setting the secondary standard identical to the primary standard fails under *American Farm Bureau.* As we explained there, it is insufficient for EPA merely to compare the level of protection afforded by the primary standard to possible secondary standards and find the two roughly equivalent. EPA must expressly "determine what level of ... protection is requisite to protect the public welfare," *American Farm Bureau,* 559 F.3d at 530, and explain why this is so. Here EPA found "significant overlap" between the revised primary standard and "selected levels" of a seasonal standard, 2008 Final Rule, 73 Fed.Reg. at 16,499, and it did say that the revised primary standard "would be sufficient to protect public welfare from known or anticipated adverse effects," *id.* at 16,500. But it justified this conclusion only by comparing the revised primary standard to a seasonal level of 21 ppm-hours that EPA never "specif[ed]" was "requisite to protect the public welfare," 42 U.S.C. § 7409(b)(2)—exactly what *American Farm Bureau* held is inconsistent with the statute.
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> . . .
>
> Because EPA failed to determine what level of protection was "requisite to protect the public welfare," EPA's explanation for the secondary standard violates the Act. We therefore remand this portion of the final rule for further explanation or reconsideration by EPA.

Mississippi, 744 F.3d at 1360-62.

EPA's proposal fails to comply with the Act and the Court's remand order. The agency has completely failed to identify target levels of vegetation protection requisite for protection of public welfare and specify levels of air quality that will achieve that protection. Although the proposal discusses various potential levels of tree growth and crop impairment from ozone, and thresholds identified by CASAC for protection against such impacts, no where does it specify levels of protection that the agency itself proposes as requisite levels of protection against these adverse welfare impacts. And as already noted, the agency also fails to propose a requisite level of protection against foliar injury.

EPA does identify a range of 13 – 17 ppm-hrs, but that range is not based on any proposed level of protection against biomass loss, carbon storage loss, or foliar injury that EPA has identified as requisite for public welfare. EPA notes CASAC's views that a 2% biomass loss is protective and a 6% loss level is unacceptable, but refuses to specify what level of protection EPA itself proposes to find requisite. Accordingly, under the holdings of *Farm Bureau* and *Mississippi*, EPA's proposal violates the Act and is arbitrary.

Likewise, EPA's proposal to set the secondary standard identical to the primary is based on the same sorts of comparative analyses rejected in *Farm Bureau* and *Mississippi*. That air quality in some (or even many) areas that meet the proposed primary standards might also meet various levels of a W126 standard does not show either that the W126 levels evaluated reflect levels requisite to protect welfare, or that the primary standards will assure achievement of such levels of protection.

Finally, even if not otherwise unlawful and arbitrary, EPA's proposal to set the secondary standard as identical to primary when the agency does not even know the level at which the final primary standard will be set renders the proposal arbitrary and unlawful. The Act requires EPA to set the primary standard to achieve a different objective than the secondary: the protection of public health with an adequate margin of safety. The notion that the level chosen to protect public welfare will just by happenstance be the very same level requisite to protect public welfare is arbitrary and not credible. EPA must specify the level for the secondary standard based on what is requisite to protect public welfare against adverse welfare impacts – here damage to forests, trees and crops. EPA's basis for choosing of a given level to protect health cannot also provide a rational basis for protecting vegetation.

9. Air Quality Monitoring Requirements

a. Changes to length of monitoring seasons

We support an extended monitoring season for the secondary standard that reflects regional seasonal differences in the growing season. Photosynthesis in conifers and early emerging forest floor species begins before deciduous canopy leaf-out which should be considered in setting the length of the monitoring season. Further, the growing season can vary greatly across the U.S. EPA also must account for the extended timing of elevated ozone concentrations in the context of climate change.

b. Need for more monitors in rural and mountain areas

We are concerned that EPA is not taking the necessary steps to ensure that monitoring will be adequate to effectively implement any new secondary standard. EPA has long acknowledged that uncertainties will remain about ozone concentrations affecting sensitive natural vegetation and ecosystems until additional monitors are sited in National Parks wilderness areas and other public lands. Yet EPA does not propose to address these concerns.

EPA should identify monitoring needs in parallel to finalizing this proposal. EPA has the information necessary to identify ecosystems of concern for impacts from ozone and plan an appropriate distribution of monitors. This information should be used to outline the monitoring that will be required to protect these areas.

Moreover, while additional monitors are of great importance it is critical that existing monitors be maintained. Funding cuts in recent years have led to the removal of important monitors. Monitored data is the lifeblood of NAAQS and EPA should ensure that funding for monitors be a priority for the agency.

10. SIP Requirements

a. Attainment Schedule for Secondary Standards Must be Expeditious

The attainment of the secondary standards must be done in compliance with Clean Air Act section 172. (a) (2) (B) which states: "The attainment date for an area designated nonattainment with respect to a secondary national ambient air quality standard shall be the date by which attainment can be achieved as expeditiously as practicable after the date such area was designated nonattainment under section 7407(d) of this title. "

While the CAA does not provide the same timeline for attainment for the secondary standards as it does the primary standards, it nonetheless requires action that is as expeditious as practicable. EPA must require states to clearly define this timeline in their SIPs.

G. It is Critical For EPA To Set The NAAQS At A Truly Health Protective Level To Allow People To Engage In Protective Behavior When The Air Quality Index, Which Is Keyed To The NAAQS, Alerts Them That Safe Levels Of Ozone Have Been Exceeded

The importance of setting the Ozone NAAQS at a health-protective level of no higher than 60 ppb is further underscored by the fact that the NAAQS are directly tied to the Air Quality Index ("AQI") and therefore critical to the public's ability to engage in so-called averting behavior, i.e. behavior to reduce their exposure to ozone.[573] If the NAAQS are not set at a truly health-protective level, the AQI will not serve its purpose of informing the public about how

[573] Averting behavior typically includes reducing time spent outdoors or reducing the level or duration of outdoor activity during times of the day when ozone levels are high.

clean or polluted the air is and people will be denied the opportunity to protect themselves and their children from unhealthy air.

1. The AQI Must Accurately Inform the Public of Health Hazards From Polluted Air

a. The Primary Purpose of the AQI Is to Facilitate Averting Behavior

The AQI is an EPA-administered, nationally uniform index for reporting and forecasting daily air quality. Its very purpose is the dissemination of accurate air pollution information. The AQI for ozone[574] runs from 0 to 500, with higher AQI values corresponding with greater levels of air pollution and greater health concerns. For ease of reference, the ozone AQI is divided into six color-coded categories, each of which corresponds to a different level of health concern:

Air Quality Index (AQI) Values	**Levels of Health Concern**	**Colors**
When the AQI is in this range:	***..air quality conditions are:***	***...as symbolized by this color:***
0-50	**Good**	**Green**
51-100	**Moderate**	**Yellow**
101-150	**Unhealthy for Sensitive Groups**	**Orange**
151 to 200	**Unhealthy**	**Red**
201 to 300	**Very Unhealthy**	**Purple**
301 to 500	**Hazardous**	**Maroon**

Green: Air quality is considered satisfactory, and air pollution poses little or no risk.
Yellow: Air quality is acceptable; however, for some pollutants there may be a moderate health concern for a very small number of people. For example, people who are unusually sensitive to ozone may experience respiratory symptoms.
Orange: Although general public is not likely to be affected at this AQI range, people with lung disease, older adults and children are at a greater risk from exposure to ozone, whereas persons with heart and lung disease, older adults and children are at greater risk from the presence of particles in the air. .
Red: Everyone may begin to experience some adverse health effects, and members of the sensitive groups may experience more serious effects. .
Purple: This would trigger a health alert signifying that everyone may experience more serious health effects.

[574] EPA also calculates the AQI for four other major air pollutants regulated by the Clean Air Act: particle pollution (also known as particulate matter), carbon monoxide, sulfur dioxide, and nitrogen dioxide.

Maroon: This would trigger health warnings for emergency conditions. The entire population is more likely to be affected.[575]

Under the proposed revision, an AQI value of 100 would correspond with the revised ozone NAAQS, i.e. the level set by EPA to protect public health. Accordingly, when AQI values are below 100, EPA encourages the public to consider air quality as generally healthy, while when AQI values are above 100, the public is cautioned to consider air quality as unhealthy, first for certain sensitive groups of people, and eventually, as AQI values get higher, for the entire population.[576]

Accordingly, a primary function of the AQI is to notify the public about when ozone levels are or are expected to be high, in order to give people the opportunity to reduce their exposure to ozone through so-called averting behavior. To that end, the AQI is widely disseminated: it can be found on the internet (on the EPA-developed AIRNow website as well as many weather reporting websites), in local and national media (including *USA Today*, The Weather Channel, and CNN), and through *EnviroFlash*, a free e-mail alert system that sends daily air quality forecast to its subscribers.[577]

b. AQI Values Are Widely Disseminated Through a Robust Infrastructure

In addition to the AQI's generally wide dissemination through media and internet outlets, state and local actors--including schools, community organizations, and city, county and state agencies and governments--have also developed a robust infrastructure whose aim is to broadcast AQI values and the attendant public health information to the public. Most notably, this infrastructure includes air alert programs, which were established to warn residents when levels of air pollution reach unhealthy levels and which are currently operated by hundreds of cities and air pollution control agencies across the country. Alert days,[578] which can be declared by a local municipality, county or state, are typically set when air quality enters the unhealthy ranges, i.e. when the AQI exceeds 100. When an alert is issued, the issuing agency directly contacts a set list of recipients, including local schools, TV stations and newspapers. According to the most recent information provided by the EPA, at least 292 cities in 35 states participate in air quality alert days.[579] Another widely implemented program is the School Flag Program, through which schools are alerted to the local air quality forecast and instructed to take steps to protect students' health from air quality hazards. The program requires schools to raise a flag that corresponds to each day's air quality, with the flag color matching the AQI colors. On unhealthy air days, schools use the air quality information to encourage averting behavior in order to reduce students' exposure to air pollution. Thus, for example, schools might adjust physical activities on unhealthy air days by either shortening, cancelling or moving outdoor activities indoors, or they might require teachers to take longer breaks during athletic activity or ensure that asthma quick-

[575] U.S. EPA, Air Quality Index (AQI)—A Guide to Air Quality and Your Health, *available at* http://www.epa.gov/airnow/aqi_brochure_02_14.pdf.
[576] *See id*.
[577] *Id*.
[578] Also called Ozone Action Days, Clean Air Alert, and Air Quality Alert, among others.
[579] *See* U.S. EPA, AIRNow Home: Action Days, *available at* http://www.airnow.gov/index.cfm?action=airnow.actiondays (accessed Feb. 19. 2015).

relief medicine is on hand. According to the most recent information provided by the EPA, at least 551 schools in 29 states participate in the School Flag Program.[580]

c. The AQI Is Relied On By Large Numbers of the Population.

The information provided by the AQI is important to and relied on by large portions of the public, as evidenced by multiple studies showing widespread and significant averting behavior in response to air quality advisories. For example, a national study analyzing air-quality alert programs found that on average, individuals reduce the time they spend in vigorous outdoor activities by 18% on air-quality alert days, and are 3% less likely to participate in any vigorous outdoor activities on alert days.[581] An earlier study investigating whether individuals varied outdoor activities in response to air-quality alerts found that that 40% of respondents stayed indoors on poor-quality air days and that individuals with smog-related symptoms significantly reduced time spent outdoors (shortening outdoor activities by about 40 minutes) when ozone concentrations exceeded the national standard.[582] A study of data at outdoor facilities in the Los Angeles area showed significant pollution-avoidance behavior, with attendance dropping by as much as 15% on air-quality alert days.[583]

Studies also show that averting behavior is particularly pronounced among sensitive populations, such as children, the elderly and those with respiratory illnesses.[584] For example, a survey of parents at a pediatric clinic found that 88% of parents were aware of air pollution advisories, 71% reduced pollution and 55% sometimes restricted children's play because of advisories.[585]

d. The Air Quality Index is Used to Reduce Emissions as Well, And Requires the Concentration that Triggers Actionable Thresholds Be Set at Much Lower Levels

The Air Quality Index not only provides information to the public that will allow them to reduce their exposure to ozone as described above, the AQI also plays a critical role in the State Implementation Plans for meeting the ozone standard. That role requires that the EPA should strengthen certain key thresholds for the ozone AQI that trigger the emission reduction actions.

Many states have incorporated special actions on days when ozone levels were forecasted to reach higher levels in an effort to change behaviors or prevent emissions, in order to reduce the production of precursor emissions for ozone. For example, Utah has a system to alert

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[580] U.S. EPA, School Flag Program Registered Schools, *available at*http://www.airnow.gov/index.cfm?action=airnow.schoolflagprogramlist (accessed Feb. 19, 2015).
[581] A.L. Sexton (Responses to Air Quality Alerts: Do Americans Spend Less Time Outdoors? (2011), *available at*http://www.apec.umn.edu/prod/groups/cfans/@pub/@cfans/@apec/documents/asset/cfans_asset_365645.pdf.
[582] *See* B. Bresnahan,, M. Dickie, & S. Gerking (1997). Averting Behavior and Urban Air Pollution, *Land Economics*, 73(3): 340-357 .
[583] M.J. Neidell (2010). Information, Avoidance Behavior, and Health: The Effect of Ozone on Asthma Hospitalizations, *J, of Human Resources*, 44(2): 450-478.
[584] *See, e.g., id.* (noting that children and the elderly showed a greater response than adults); *see also* Sexton 2011.
[585] M. McDermott, J. Srivastava, & S. Croskell (2006). Awareness of and compliance with air pollution advisories: A comparison of parents of asthmatics with other parents, *J. of Asthma*, 43: 235-239.

residents for high ozone days that breaks action into 3 categories[586]: Unrestricted action, Voluntary Action and Mandatory Action which requires employers to limit driving by their employees. The timing for these actions depends on the AQI forecast. Likewise, Washington State law also incorporates mandatory action into its episode avoidance plan for days when where air pollution levels are forecast.[587]. Such mandatory actions are included in permits for industry that specify actions to be taken at each alert level.[588] The San Joaquin Valley Air Pollution Control District, which serves one of the most-ozone polluted areas in the nation, also incorporates similar action levels, triggered by the forecasted air quality, that puts enforceable requirements on permitted, stationary sources of ozone precursors.[589]

With these and similar requirements and permits in place, the AQI often used by these systems to trigger behavior change begins at the Unhealthy (red) breakpoint, not the Unhealthy for Sensitive Groups (orange) breakpoint that usually corresponds to the level of the NAAQS. Therefore, it is critical that the level of Unhealthy be set in the AQI to ensure that it recognizes the higher risk at that level.

The final AQI levels needs to provide adequate recognition of the higher levels of risk at the Unhealthy level. Under the proposal, EPA has provided a range of levels for the theshold into the unhealthy for sensitive groups category, based on the proposed standard. Unfortunately, the proposed AQI Breakpoints[590] incorporate too great a range of ozone concentration into the Unhealthy for Sensitive Groups classification, pushing the threshold for the Unhealthy level far above where the actions they trigger should begin.

Instead of having the threshold for Unhealthy begin at 85 ppb, we urge EPA to adopt a much lower threshold that would trigger these pollution reduction mechanisms that states and local agencies have been using to prevent the release of ozone precursors. Setting the breakpoint for Unhealthy at 85 ppb would, by EPA's own rationale, not require the triggering of these pollution reduction measures until the air quality threatened to impact 25 percent of exposed people.[591] However, the exposed people in the discussion in McDonnell et al, 2012, that EPA cites,[592] are healthy adults and as we discuss elsewhere the burden of ozone harems the health of many people are far lower thresholds than the thresholds for healthy adults. Since McDonnell et al. state that this threshold should be shifted to recognize the impact on different populations, the AQI should also have the threshold shifted to recognize harm to the diverse population.

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[586] http://air.utah.gov/forecastLegend.html

[587] State of Washington. RCW 70.94.715. Air pollution episodes — Episode avoidance plan — Contents — Source emission reduction plans — Authority — Considered orders.

[588] For example, see the Air Operating Permit from the Puget Sound Air Pollution Control Agency for the U.S. Oil and Refining Company, issued December 11, 2011. Accessed at http://wwwdev.pscleanair.org/library/Air%20Operating%20Permits%20Library/12593-faop.pdf on March 16, 2015.

[589] San Joaquin Air Pollution Control District. Current District Rules and Regulations. Regulation VI: Air Pollution Emergency Contingency Plan. Accessed at http://www.valleyair.org/rules/1ruleslist.htm on March 16, 2015

[590] Proposed Rule at 75,311

[591] *Id.*

[592] *Id.*; McDonnell WF, Stewart PA, Smith MV, Kim CS, and Schelegle ES. Prediction for lung function response for populations exposed to a wide range of ozone conditions. *Inhal Tox* 20101; 24(10) 619-633.

For these reasons—the important role that the Unhealthily Category plays in efforts to reduce ozone precursors, and the need to recognize that the threshold needs to be shifted to accommodate the more sensitive populations—EPA should reconsider its AQI Breakpoints and select a much lower threshold than 85 ppb as the breakpoint of for Unhealthy Category.

2. Failing to Set the NAAQS At the Health Protective Level of 60 ppb Would Lead to the Dissemination of False Information and Rob People of Their Ability to Protect Themselves and Their Children From Unsafe Air.

The purpose of the AQI, the extent of its infrastructure, and the high degree of reliance exhibited by the public require that the NAAQS be set at a truly health protective level of no higher than 60 ppb. Setting the NAAQS any higher than 60 ppb will have the direct effect of depriving the public of knowing what health impacts they and their children might suffer on any given day, as the information conveyed to them will not accurately reflect the degree to which the air in their surroundings presents a health risk. Consequently, it will also prevent the public from engaging in averting behavior (to the extent that doing so is possible) and thereby exercising their right to protect themselves and their children from the health effects of polluted air. Failing to set the ozone NAAQS at 60 ppb will therefore result in a perversion of the purpose of the AQI and the waste of its attendant infrastructure.

The need for an accurate and reliable AQI is further underscored by the fact that episodes of unsafe air will occur *even if* the current round of ozone NAAQS revisions results in the setting of a health protective standard. First, because areas will (and are allowed to) take years to come into compliance with existing standards, unsafe air will continue to plague residents of those areas long after any new standards are issued.[593] Further, because the current (and proposed) ozone NAAQS use the annual 4th-highest daily maximum 8-hour concentration averaged over 3 years as the form of the standard, even areas that are in attainment of the (current or revised) standard will see numerous 8-hour periods (and are likely to see many more shorter periods) that exceed the standard in any given year. People living in such areas have the right to know whether and when the air they breathe is safe. Setting the NAAQS at the health protective level of 60 ppb is the only way to ensure that people living in these and all other areas have the information they need to protect themselves and their children.

H. Appendix U: EPA's Proposed Changes to Implementation Rules Would Undermine the Health and Welfare-Protectiveness of the Revised Standard and Are Inconsistent with the Act and its Regulations

1. The Language of the NAAQS Should Not Be Limited to Monitoring Sites; The Language Needs to Be Broad Enough to Take into Account the Potential Use of Modeling for Evaluating Attainment, Which Should Utilize a Full Receptor Grid Reflecting the Fact the NAAQS Are National Ambient Air Quality Standards

[593] Air quality in 10% of the original 113 areas designated as non-attainment for the 1997 Ozone NAAQS still does not meet the 1997 standards and as of 46 areas were failing to attain the 2008 standards as of 2012. Proposed Rule at 75,370.

EPA's proposed language in the NAAQS itself, that is 40 C.F.R. § 50.19, is problematic. Specially, the references to reference and equivalent methods and monitoring sites in 40 C.F.R. § 50.19(a), (b), (c), and (d) should be removed. Then, an additional subsection (e) should be added which says something to the effect of, "When a monitor is used to determine compliance with a NAAQS, it must be a reference method based on appendix D to this part and designated in accordance with part 53 of this chapter or an equivalent method designated in accordance with part 53 of this chapter."

EPA should make this change for three reasons. The first is that the proposed language invites challenges from polluters to the use of methods other than air monitors in determining compliance with the NAAQS. For example, polluters used a similar argument in trying to challenge the 2010 sulfur dioxide NAAQS and its implementation.

The second is that such language is used to justify modeling only at model receptors which are representative of ambient monitor locations. Such a modeling approach is illegal and not adequately protective of public health and welfare. Rather, in modeling, a full receptor grid should be used because this is a "national" standard.

The D.C. Circuit has recognized the geographic limitations of monitoring. *See e.g. Catawba County v. EPA*, 571 F.3d 20, 30 (D.C. Cir. 2009) ("EPA's selection of the county as the unit of analysis resolved a problem inherent in the monitoring process, namely, that a monitor only measures air quality in its immediate vicinity."). There is no rational reason to export the inherent problem in monitoring into the modeling process. However, the proposed language of 40 C.F.R. § 50.19(a), (b), (c), and (d) invites arguments for such an ill-conceived approach to modeling.

Modeling only at receptors representing ambient monitor locations also violates the plain language of the statute and regulations. In *National Ass'n of MFRS v. EPA*, 750 F.3d 921, 926 (D.C. Cir. 2014) the D.C. Circuit explained: "The point of the NAAQS program is to safeguard the quality of the "ambient air," which is defined as the "portion of the atmosphere, external to buildings, to which the general public has access." 42 U.S.C. § 7409(a); 40 C.F.R. § 50.1(e)". It is contrary to the definition of ambient air to redefine ambient air as only the air at monitoring sites. EPA therefore, should take out references to air monitors in 40 C.F.R. § 50.19(a), (b), (c), and (d).

The third reason is EPA is proposing to use this language to weaken public health and welfare protection even further, and move further away from what the science says is the necessary level of protection, by not calculating design values for each monitor but rather for each site.[594] This issue is discussed in detail below.

2. Data Completeness: EPA Needs to Create a Methodology that Fills in Missing Data, Similar to 40 C.F.R. Part 75 of the Acid Rain Program

[594] Proposed Rule at 75,351.

EPA needs to create a methodology that fills in missing data, similar to 40 C.F.R. § Part 75 of the Acid Rain program. Without this, the NAAQS cannot ensure the protection that EPA says it is choosing to provide.

The metro-Atlanta ozone nonattainment area, for example, has missing data. Numerous times, hourly ozone levels trend significantly upward so that it looks almost certain that there will be an 8-hour average, but then the monitor becomes "unavailable" for some reason before enough data is gathered to get a violating 8-hour average.[595] Currently, states do not have an external incentive to ensure they gather complete ambient monitoring data. EPA needs to create incentives and consequences for states having complete monitoring data.

The current and proposed form and averaging time do not make adjustments for missing data. However, the old one-hour average did. While there is a minimum number of days that monitoring sites are required to collect, there is no consequence if the monitoring site fails to meet this standard. In any event, because the minimum standard is not 100% of the required days or hours, people can be and are exposed to significantly more short term periods about the level that EPA says is the appropriate level. EPA should add a data filling mechanism based on protective assumptions.

3. EPA Should Not Decrease the Number of Monitors Used to Calculate Design Values

EPA is proposing to no longer consider design values from monitors other than the primary monitor that are located at multi-monitor sites.[596] Not considering design values from monitors at multi-monitor sites is the same as reducing the number of monitors. EPA presents no reason why the number of monitors should be decreased while the standard is being made more protective, nor is there one. Rather, it is arbitrary to ignore data for monitors other than the primary monitor which EPA has in making regulatory decisions.

EPA seems to imply that there is consistency across monitors at the same site. That is not always true. For example, AQS reports the 4th high for 2013 for monitor 1 at site 060430003 in Yosemite National Park at 0.073 ppm, or above the proposed NAAQS.[597] However, AQS reports the 4th high for 2013 for monitor 2 at that same site as 0.056 ppm or below the proposed NAAQS.[598] This is a 30 percent spread which could not be fairly described as consistent. Nor is this an isolated example.

We do applaud EPA's desire to make sure there are more complete sets of ambient monitoring data. However, as we explained in section V.H.2, the appropriate way to address this is to create a protocol for filling in missing data similar to the one used in the Acid Rain Program.

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[595] Data available at http://www.air.dnr.state.ga.us/amp/ and incorporated herein by reference.
[596] *See* Proposed Rule at 75,351.
[597] *See* Exhibit 17 at 27.
[598] *Id.*

4. Data Handling: EPA Should Take an Approach that Is Rational, Protective and Consistent with the Statute

EPA's current and proposed data handling conventions weaken the NAAQS by increasing the risk that people will actually be exposed to ozone levels above the levels EPA deems appropriate. We recommend EPA take another approach. Consistent with the Congressional intent that the Clean Air Act use a precautionary approach when it comes to exposing innocent people to toxic air pollution, EPA should use data handling conventions that err on the side of protecting people and the public welfare rather than ones that arbitrarily assume certain amounts of ambient ozone does not exist when we in fact know that it does. Below we discuss elements of the data handling convention and how EPA can implement them consistent with this guiding principle.

EPA's proposed data handling convention would require that any decimal digits reported beyond three decimal digits will be truncated.[599] EPA's stated reasons for this are (1) consistency with past practice and (2) typical measurement uncertainty.[600]

EPA must, at a minimum round the third decimal place rather than truncate. Past practices do not provide a rational basis to truncate in this context because monitoring equipment has changed over the decades since EPA started truncating. As to measurement uncertainty, truncating and rounding both address this uncertainty. However, rounding is more consistent with Congress' clearly expressed will that NAAQS be addressed in a precautionary manner. *See* 42 U.S.C. § 7409(b)(1)("allowing an adequate margin of safety"). Whatever convention EPA uses, that method must be accounted for in setting the level of the standard. As shown in part V.B.3.b.ii above, the use of such conventions can result in the standard being insufficiently protective to prevent adverse effects.

Similarly, EPA should not substitute zero for one-half of the minimum detection level (MDL) as it is proposing in calculating 8-hour averages from the hourly ozone data when fewer than six hourly ozone concentrations are available during an 8-hour period.[601] Substituting zero is arbitrary because there is no reason to believe the value is actually zero. Rather, a review of AQS shows hours with zero ozone levels are extremely rare.

The most rational approach is to extrapolate the most reasonable approximation of the hourly value based on trajectory of the hourly values closest in time to the missing hourly value or some other mathematically acceptable way to approximate. This approach is also more consistent with Congress' intent for a precautionary implementation of the NAAQS. This approach also moves the actual implementation of the standard closer to providing the protection against exposures of concerns which EPA claims to the public and CASAC that EPA is providing. Moreover, this need not be complicated, time consuming or resource intensive. It can be done using automated computer programs.

[599] Proposed Rule at 75,352.
[600] *Id.*

[601] *Id*. 75,352.

EPA cannot lawfully or rationally get rid of the rule that provides for 8-hour daily maximums to include overlapping hours from two days.[602] Again, EPA's proposal moves the standard away from the protection EPA is claiming, especially since the human controlled exposure studies are often based on 6.6 hours of exposures not 8 hours so the exposure of concern is really shorter than the NAAQS form. In addition, this is not double counting from an individual exposures point of view. People come and go from areas. Moreover, EPA's claim that post-sunset ozone peaks should not be counted because they "are assumed to be caused by transport of O3 molecules"[603] is doubly irrational. First, it's based expressly on an unexplained assumption. Second, wherever the ozone came from, it's present in the area and available for people to breathe. EPA must provide people the protection the Clean Air Act guarantees. Overall, using only the daily maximum is not supported by the controlled human exposure studies or by EPA's regulatory stability argument. EPA shouldn't make the problem of using only the daily maximum worse by its new proposal of only 17 8-hour periods in a day rather than 24.

EPA also proposes that there must be 13 of 17 8-hour periods in a day in order to determine a valid daily maximum.[604] EPA says that it is including this requirement because 13/17 is consistent with the 75% data completion requirement used for daily and annual NAAQS-related statistics.[605] However, EPA proposes to keep the provision in 40 C.F.R. § 50, Appendix P which says that a daily maximum 8-hour average is valid if it is greater than the NAAQS. *Id.* The Appendix P language is mandatory: "a day shall be also be counted as valid". 40 C.F.R. § 50, Appendix P 2.1. The preamble used an unclear term "allowing."[606] We believe that the preamble was just lacking in precision and that the language in Appendix P 2.1 will remain mandatory. We support and believe the Clean Air Act mandates that any day with an 8-hour average above the level of the NAAQS must be included in calculating the design value.

5. Changes to Length of Monitoring Seasons

a. The Length of the Ozone Season Should be a Uniform 12 Months

Over five years in the making, EPA is proposing to expand the length of the required ozone monitoring seasons in a number of states.[607] We strongly support expanding the length of the ozone monitoring seasons. As further detailed below, ozone exceedances are recorded outside of the traditional ozone season in a number of states, and would likely be recorded in other states if the monitoring season was expanded.

EPA's proposal, however, does not go far enough. EPA is trying to set the ozone season to monitor when conditions are conducive to ozone formation.[608] EPA tries to do this by looking at data from monitors which were operated outside of their ozone season. But, this approach

[602] *Id*.
[603] *Id*. at 75,352-53
[604] *Id*. at 75,353.
[605] *Id*.
[606] *Id*.
[607] *Id*. at 75,358.
[608] *Id*.

almost certainly misses many situations in which ozone exceedances occur outside the traditional ozone season because approximately 700 monitors do not operate year round.[609] In addition, EPA's current methods for determining what conditions are conducive to ozone formation are overly conservative. For example, EPA used to believe that ozone was not formed in the winter time in colder climates. However, the designation of part of southwest Wyoming as nonattainment for the 2008 ozone NAAQS demonstrates the error of that belief.[610] Climate change is also likely to increase the length and severity of ozone seasons. To assure more complete identification of the periods in which ozone exceedances occur, EPA needs to require all monitors to operate year round.

Furthermore, EPA's use of only one year of data in some instances to determine if they ozone season needs to be extended arbitrarily excludes situations in which looking at more than one year would show the need for such extended monitoring. *See* 79 Fed. Reg. at 75,358.

The fact that EPA's proposed increase in ozone seasons would only cost $230,000[611] is a strong indication that increasing all ozone monitors to year-round would also cost a modelst amount. Furthermore, the Clean Air Act requires that this cost be covered by permit fees, not by state agencies' general budgets. The cost of increasing all monitors to year round monitoring would be truly trivial when spread across all permitted polluters.

In addition, the regulations cannot allow the Regional Administrators to change the regulations regarding ozone season without notice and comment. This would be an APA and/or CAA violation. It also would be very misleading for the public who often rely on the ozone monitoring data which is communicated to the public to make decisions to protect the health and lives of their families. If an ozone season is changed without public notice, people may assume that ozone levels are safe when in fact they are not safe but there is no monitoring being conducted to report ozone levels to the public. Note, however, that we do fully support revocation of previous Regional Administrator-granted waiver approvals.

Also, EPA proposes not to increase the PAMS monitoring season from the current 3-month June-August period.[612] EPA does so even though it acknowledges that "in many areas the highest O3 concentrations are observed outside of the PAMS season." Id. CASAC recommended extending the season and making it more flexible.[613] Countering those considerations, EPA points only to "the potential burden associated with a lengthening of the PAMS season" and the value of a uniform season to "provide a consistent data set."[614] That is not a rational explanation for refusing to gather more, better data.

b. Barring Year-Round Monitoring in All Areas, EPA Should Ensure that States Where There Is or May Be Winter-Time

[609] *Id.* at ftnt. 232.
[610] In addition, states sometimes claim that their ozone problems are caused by long range transport of ozone, including international transport. To the extent this is true, an ozone season based on local conditions would be arbitrary because it fails to consider conditions where the ozone is allegedly created.
[611] Proposed Rule at 75,360
[612] *Id*. at 75,365.
[613] *Id*.
[614] *Id*.

Ozone Season Problems Including All of the Region 8, 9 and 10 States, Should Have Year Round Ozone Monitoring

Even if EPA were to reject our recommendation to increase the ozone monitoring season to 12 months for all monitors, EPA still needs to make changes to its proposed ozone monitoring seasons. EPA claims that places like Montana and South Dakota only have 4 month long ozone seasons. This is inconsistent with winter time ozone monitoring data when it was actually done in the winter for similar places like Wyoming as evidenced by the fact that SW Wyoming is now a designated nonattainment area. In fact, EPA plainly states: "As an example, the highest O3 concentrations in the Mountain-West often occur during the winter months."[615]

Yet for the Mountain-West state of Wyoming, EPA's proposal is to not require monitoring during the winter months of October, November and December.[616] While it is true that winter technically begins on December 21st, it is common knowledge that Wyoming experiences what anyone would describe as winter weather during the months of October, November and December. For example, in Rock Springs, Wyoming, the average high and average low temperature in December is the same as in January.[617] The average snowfall in December is just one inch different than the average snowfall in January.[618] Thus, it is arbitrary to not require ozone monitoring in December but require it in January when the ozone formation factors are very similar. October and November, while slightly warmer on average, also have almost the same snowfall average as January, February and March. Since snowfall is probably an important factor in wintertime ozone formation, it makes sense to also require ozone monitoring in October and November.

Montana, South Dakota and North Dakota are in a similar situation to Wyoming. The Bakken oil patch has resulted in an exponential increase in ozone precursor emissions in this area. Montana is actually in the "Mountain-West" which EPA says often experiences its highest ozone concentrations in winter months.[619] Yet, contrary to its own admitted fact, EPA is not proposing to require ozone monitoring Montana during winter months.[620]

It is not clear whether EPA believes the Mountain-West to include South Dakota and North Dakota. It is clear that the Bakken oil patch is mainly in North Dakota and its ozone precursor emissions are skyrocketing. In 2013, the most recent year of undisputedly final ozone data, all 9 of North Dakota's ozone monitoring sites experienced 8-hour ozone values of 0.060 ppm or greater according to EPA's AQS. Although slightly colder, Watford City, North Dakota, for example, gets similar amounts of wintertime precipitation and snow as Wyoming.[621] Thus, EPA should create a year round ozone monitoring season for Wyoming, Montana, North Dakota and South Dakota.

6. Changes to Monitoring Network

[615] *Id*. at 75,365.
[616] *Id*. at 75,410.
[617] *See* Exhibit 19.
[618] *Id*.
[619] *Id.* at 75,365.
[620] *Id.* at 75,410.
[621] See Exhibit 19 and Exhibit 20.

The current ozone monitoring network is inadequate. One example of this is the recent discovery that the Upper Green River Basin area in Wyoming is violating the 2008 ozone NAAQS. Another example is Metropolitan Statistical Area (MSAs) are not required to have monitors and 105 of these MSAs, with a combined population of 18 million people, did not have monitors that gathered enough data to determine a design value.[622] EPA has concluded that there is a reasonable likelihood that many of these MSAs would have violated the 2008 NAAQS.[623] Moreover, the monitoring network is inherently inadequate because monitors only measure air quality in their immediate vicinity. *Catawba County v. EPA*, 571 F.3d at 30. EPA acknowledged the inadequacy of the monitoring network before by proposing to modify the minimum monitoring requirements in urban areas and adding new minimum monitoring requirements in non-urban areas. 74 Fed. Reg. 34,525 (July 16, 2009). Unfortunately, EPA never finalized the 2009 Proposal. EPA should use this rulemaking as an opportunity to begin to address this problem.

First, consistent with the 2009 Proposal, EPA should modify the minimum ozone monitoring requirements to require at least one monitor to be placed in MSAs of populations ranging from 50,000 to less than 350,000. This would help ensure that an additional 18 million people or more are not being exposed to dangerous or deadly ozone pollution.[624]

Second, to ensure protection consistent with both the secondary and primary NAAQS, and again consistent with the 2009 Proposal, EPA should require states to have ozone monitors in three categories of non-urban areas. The first required non-urban monitor should be located in areas such as some federal, state, or tribal lands, including wilderness areas that have ozone-sensitive natural vegetation and/or ecosystems. Exhibit 21 lists ozone -sensitive natural vegetation.[625]

The second required non-urban monitor category should be required to be placed in a MSA expected to have ozone design value concentrations of at least 85 percent of the NAAQS. This is important because EPA's rules should not discriminate against people who live in rural areas. In addition, many MSAs may have low residential numbers and yet are tourist areas where many more people spent time engaged in outdoor physical activity which EPA has repeatedly acknowledges makes them more at risk of injury ozone population. For example, evidence indicates that the elevated ozone levels in the front range of Colorado extend all the way up to the continental divide.[626] Yet, there are no regulatory monitors in Western Boulder County, Gilpin, Clear Creek, Summit, Park, Teller, Fremont, Custer, and Huerfano Counties and thus none of the front range mountain counties are designated non-attainment except Boulder County based on urban monitors. While these counties have relatively low residential populations, millions of people engage in outdoor activities in these counties. These areas

[622] 74 Fed. Reg. 34,525, 34,527 (July 16, 2009)(2009 Proposal).
[623] *Id.* at 34,528.
[624] To the extent EPA is concerned about a procedural challenge to such a requirement, EPA could finalize the 2009 proposal in the same rule that finalized the ozone NAAQS.
[625] *See* Exhibit 21 at 11-13.
[626] *See e.g.* Exhibit 18. Note that the NCAR Frappe study and NASA Discovery AQ studies, to which this article refers have not released all of their data to the public yet. However, EPA is participating in the NCAR Frappe study. Thus, all data from this study currently in EPA's files are incorporated herein by reference.

include ski areas which host year round activities as well as wilderness areas and national and state parks. Many of the activities engaged in these areas such as hiking, backpacking, mountain biking, and skiing are very physically demanding resulting in increased respiration. Even moderate exercise or no exercise in these communities can result in significant increases in respiration for people not used to the elevation, thus increasing their ozone exposure. Yet the current monitoring network design completely fails to address this situation. Similar situations can be found in other Western states.

The third required non-urban monitor should be in the area of expected maximum ozone concentration outside of any MSA, potentially including the far downwind transport zones of currently well-monitored urban areas. This expectation should consider any creditable evidence of potential high ozone levels. This includes monitors that are not formally a part of EPA's regulatory system as well as credible data from computer models, satellites and aircraft measuring ozone.

7. Implementation Issues

a. Prevention of Significant Deterioration: Significant Emission Rates and Significant Impact Levels

In the Proposal, EPA briefly discusses various screening tools used to assess whether or not a source must undertake Prevention of Significant Deterioration (PSD) review. The agency notes that it has established a "Significant Emission Rate" (SER) for ozone precursors (VOCs and NOx) of 40 tons per year (tpy), and that the agency uses 100 tons per year as a substitute of sorts for a Significant Impact Level (SIL), which it has not yet set for ozone.[627] The agency further notes that it "intends to consider whether it is appropriate to make any revisions to the PSD regulations related to the screening tools for [ozone] in a separate rulemaking…"[628] It is of critical importance that EPA undertake revisions to the SER and SIL-like tools (though not create a SIL). Although we applaud the agency for planning to undertake the analysis required to revise these tools, we believe that these revisions should be undertaken contemporaneously with this and any other update to NAAQS.

i. Significant Emission Rates

A Significant Emission Rate set at 40 tpy is entirely inappropriate in the context of the proposed rulemaking. A SER set at 40 tpy for NOx and VOCs has no rational relationship to a revised 8-hour ozone standard. SERs are used to determine "where pollutant emissions or ambient impacts could be considered *de minimis*," and there is nothing to suggest that 40 tons per year is a *de minimis* level of pollution under any revised standard.[629]

EPA claims authority to set *de minimis* exceptions to NSR permitting based on from *Alabama Power v. Costle*. 636 F.2d 323 (D.C. Cir. 1979). In that case, the D.C. Circuit suggested that EPA could exempt from PSD review some emission increases on *de minimis* grounds. 636

[627] Proposed Rule at 75,379.
[628] *Id.*
[629] *Id.*

F.2d at 400. As the Court made clear, however, the burden of justifying any such exemption would be on EPA, and the agency's inquiry must focus on the statutory goals:

> Unless Congress has been extraordinarily rigid, there is likely a basis for an implication of de minimis authority to provide exemption when the burdens of regulation yield a gain of trivial or no value. *That implied authority is not available for a situation where the regulatory function does provide benefits, in the sense of furthering the regulatory objectives, but the agency concludes that the acknowledged benefits are exceeded by the costs*.

Id. 360-61 (emphasis added).

When implementing *Alabama Power,* EPA acknowledged that it could not label any pollution levels "*de minimis*" unless it first determined "the cumulative effect on increment consumption of multiple sources in an area each making the maximum *de minimis* emissions increase (thereby going unreviewed under PSD at the time of the change)."[630] That is, the agency recognized (1) that the Clean Air Act proscribes increment violations; (2) that PSD is a vital mechanism for enforcing increment restrictions; and thus, (3) that the agency lacks authority to exempt *de minimis* pollution increases from PSD if there is any chance that such exemptions could, individually or cumulatively, lead to increment violations.

> While one source may modify its facility and not cause a significant air quality impact, a number of sources making such a change could cause a significant impact. If the sources were located near to each other, the cumulative air quality impact could consume a significant amount of the increment. Since the extent of the impact is directly proportional to the number of sources and their relative proximity to each other, it is important to determine the potential air quality impact from a number of existing sources making *de minimis* changes in emissions.[631]

The same analysis must, logically, hold for NAAQS violations.

Fundamentally, the methodology utilized by EPA for determining NSR applicability must be consistent with the agency's obligation to ensure that the PSD permitting program prevents modified sources from "caus[ing], or contribut[ing] to" an increment or NAAQS violation reflecting actual NAAQS in place as a matter of law. The last time the Agency apparently undertook such exercise was in 1980 when the ozone NAAQS was 120 ppb.[632] The Agency has done nothing to show that the current SER of 40 tpy, as keyed to an updated standard, would ensure that such increment or NAAQS violations were prevented.

As such, we urge EPA to move forward with revisions to its PSD screening tools as quickly as possible. We also ask that the Agency include in this rulemaking any information on

[630] 45 Fed. Reg. 52,676, 52,707 (Aug. 7, 1980).
[631] U.S. EPA (1980). Impact of Proposed and Alternative De Minimis Levels for Criteria Pollutants, 7 (EPA-450/2-80-20).
[632] 45 Fed. Reg. 52,676, 52,732 (Aug. 7, 1080).

the sufficiency of a 40 tpy SER to prevent air quality violations relating to the range of standards proposed in the rulemaking. The SER cannot be disconnected from the level of the NAAQS as is the case with the levels set in 1980.

Should EPA lower the NAAQS for ozone, it must provide a rational basis for concluding that the existing SER continues to be appropriate.

ii. Significant Impact Levels

EPA notes that SILs are used to "determine the extent to which an ambient impact analysis must be completed for the applicable pollutant," and that the Agency has yet to set a SIL for ozone. 79 Fed. Reg. 75,379. We do not agree that SILs are lawful or rational for reasons given in comments on EPA's PM2.5 increment rule, which we adopt by reference. EPA-HQ-OAR-2006-0605-0040. In the event that EPA persists with using SILs and SIL-like tools, we offer the following comments.

Current regulations state that "any net emissions increase of 100 tons per year or more of [VOC] or [NOx] subject to PSD would be required to perform an ambient impact analysis, including the gathering of air quality data." 40 CFR § 51.166(i)(5)(i). EPA goes on to note that these values

> do not reflect a categorical conclusion by the EPA that sources emitting less than 100 tpy of VOCs or NOx will not cause or contribute to a violation of the current (or any revised) O_3 NAAQS, nor does it reflect a conclusion that such sources should be categorically excluded from the requirement for an ambient impact analysis.

Id.

The ambiguous and vague nature of this statement underscores that EPA *must* revise the 100 tons/year threshold for ambient impact analysis found in 40 C.F.R. § 52.21(i)(5)(i) and 51.166(i)(5)(i). There is no record in the docket for this rulemaking or elsewhere to demonstrate that NOx and VOC emission levels set at 100 tpy are *de minimis* in relation to any *new* ozone NAAQS, nor even, as the Agency itself notes, the current NAAQS. In a recent response to a petition for rulemaking on this issue, the Administrator noted that in the 1990 NSR Workshop Manual, at page C.28 footnote b, the Agency

> sa[id] the following with respect to the then-applicable one-hour ozone NAAQS: 'No significant ambient impact concentration has been established. Instead, any net emissions increase of 100 tons per year of VOC subject to PSD would be required to perform an ambient impact analysis.'

The Administrator allowed that "based on these statements, this 100 [tpy] value has been used by some permitting authorities in a manner similar to a SIL to assess whether a detailed air quality analysis should be conducted for ozone."[633]

[633] Letter from Gina McCarthy, U.S. EPA Assistant Administrator, to Robert Ukeiley, at 4 (Jan. 4, 2012), *available at* http://www.epa.gov/scram001/10thmodconf/review_material/Sierra_Club_Petition_OAR-11-002-1093.pdf.

The Agency acknowledges in the same letter that the 100 tpy level has "not been revisited by the EPA since the promulgation of the 8-hour ozone NAAQS and do not reflect a categorical conclusion by the EPA that every source emitting less than 100 TPY of NOx or VOCs will not cause or contribute to a violation of the current ozone NAAQS."[634]

The Agency itself has, in this rulemaking and others, allowed that the 100 tpy threshold functions "in a manner similar to a SIL" but at the same time asserts that "the EPA has not established a SIL for O_3."[635] As noted above, the court in *Alabama Power* made clear that the Agency's discretion does not extend beyond the confines of the statutory language. For reasons stated in the briefs for Sierra Club in Sierra Club v. EPA, 705 F.3d 458 (D.C. Cir. 2013) (incorporated by reference), we contend that the Clean Air Act forecloses the use of SILs to avoid or truncate the demonstration required by 42 U.S.C. §7475(a)(3). Accordingly, EPA needs to make clear that sources must undertake an individualized and comprehensive analysis in consultation with the appropriate EPA regional office to ensure that pollutant emissions and ambient impacts from the source are fully evaluated and considered, and demonstrate that the source's emissions will not cause or contribute to a violation of the NAAQS.

b. SIP requirements, Schedules and Attainment Dates

In setting a schedule to implement updated NAAQS, the Clean Air Act's statutory language itself provides the requirements that states must meet in preparing state implementation plans to attain and maintain compliance with the updated ozone standards. The deadlines the Act establishes are outer time limits, however: nothing prevents EPA from encouraging states to coordinate their submissions of SIP elements by submitting some of them earlier than the last possible day. For the purposes of the rulemaking at issue here, nothing prevents EPA from issuing schedules for states contemporaneously with the issuance of an updated NAAQS, to facilitate states' timely submissions. We urge EPA to take that approach here, as it ensures timely achievement of the goals laid out in the Act, and assists the states in ensuring that their recommendations to EPA are as well-informed by appropriate information and methodologies as possible.

Specifically, EPA notes that it intends to propose guidance or rules for "assisting with implementing any revised O_3 NAAQS resulting from this proposal within 1 year after a revised NAAQS is established."[636] In particular, EPA notes that it is considering rules relating to "nonattainment area classification methodologies, SIP due dates, attainment dates, and required implementation programs such as NNSR and conformity."[637] The agency estimates that the rules will be finalized 2 years after the NAAQS issue.[638]

To the extent EPA intends to issue such rules we urge EPA to issue SIP requirements, schedules, and attainment dates as quickly as possible, and strongly suggest that the agency

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[634] *Id.*
[635] *Cf. McCarthy Letter* at 4, 79 Fed. Reg. 75,379.
[636] Proposed Rule at 75,373.
[637] *Id.*
[638] *Id.* at 75,374.

consider finalizing these requirements contemporaneously with any updated NAAQS. However, compliance with statutory time frames for implementing the NAAQS is not contingent on issuance of EPA rules, and any delay in issuance of such rules will not in any way excuse such compliance.

c. EPA Has No Authority to "Grandfather" Sources Out of PSD Requirements

EPA proposes to amend the federal PSD permitting regulations in 40 C.F.R. § 52.21 to allow certain PSD permit applicants to avoid demonstrating that their project will not cause or contribute to violations of a new ozone NAAQS.[639] The plain and unambiguous language of Clean Air Act section 165, however, does not confer on EPA any authority to exempt, or "grandfather" permit applicants from the statute's PSD permitting requirements.[640]

EPA admits to no ambiguity in the requirements of the statute. 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"). EPA acknowledges that it interprets the language of the CAA "to require that PSD permit applications must include a demonstration that new major modifications will not cause or contribute to a violation of any NAAQS that is in effect as of the date the PSD permit is issued."[641] 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). This plain meaning is reinforced by the purposes of the PSD program, the statutory structure around the program, and the legislative history behind it.

The express purposes of the PSD program include:

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[639] *Id.* at 75378 and 75404 (proposing amendments to 40 C.F.R. § 52.21(i)(12))

[640] To the extent EPA intends to issue such rules, because of this potentially compressed timetable, we urge EPA to issue SIP requirements, schedules, and attainment dates as quickly as possible, and strongly suggest that the agency consider finalizing these requirements contemporaneously with any updated NAAQS. However, compliance with statutory time frames for implementing the NAAQS is not contingent on issuance of EPA rules, and any delay in issuance of such rules will not in any way excuse such compliance.

[641] Proposed Rule at 75,377.

> (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 ensure 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 ozone 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 ensure economic growth is consistent with the preservation of air resources, and precludes careful decision-making and informed public participation.

When EPA adopted regulations implementing the PSD program in 1980, the Agency expressly rejected similar requests for "grandfather" exemptions based on these same clearly stated statutory purposes.[642] Specifically, in its final rule, EPA rejected a commenter's suggestion that EPA "promulgate a grandfather provision that would use the date of complete application instead of the date of permit issuance" in determining the applicability of section 165's requirements.[643] As the Agency noted, the "[u]se of such date, however, might exempt more projects from review" and "fail to give adequate expression to the interests behind section 165, especially the goal of protecting air quality." *Id.*

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

[642] 45 Fed. Reg. 52,676 (Aug. 7, 1980).
[643] *Id*. at 52,683.

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 ozone 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. Unless the source can meet these criteria, it may not be built.

EPA does not suggest that there is ambiguity in the statutory language of section 165(a)(3). Yet EPA maintains that it is nonetheless free to waive these requirements through rulemaking following the adoption of a new NAAQS.[644] EPA suggests that such discretion derives from the fact that, under CAA section 165(c), the agency must grant or deny a permit within one year of a completeness determination, and its general rulemaking authority in CAA section 301.[645] EPA's arguments lack merit.

[644] *See* 79 Fed. Reg. at 75377.
[645] *See id.*

The initial premise of EPA's argument is false. Even assuming the promulgation of a new NAAQS might lead to the inability to *approve* a permit within the deadlines of section 165(c) – a conclusion that has no actual record basis – such inability does not create a conflict or tension with the requirements of section 165(a)(3). First there is no actual conflict between these sections. If EPA cannot approve the project within the applicable deadline while also finding that the source has met its statutory obligations regarding air quality protection, the appropriate resolution is either to deny the permit application because it does not meet the requirements of the statute, or to acknowledge that with the promulgation of a new NAAQS the application is no longer complete.

Second, even if it turned out to be impossible to comply with both sections, that failure does not create a conflict that allows EPA to pick which section it is going to ignore. A fundamental canon of statutory construction is that "[a] statute should be construed so that effect is given to all its provisions, so that no part will be inoperative or superfluous, void or insignificant." *Hibbs v. Winn*, 542 U.S. 88, 101 (2004) (internal quotation and citation omitted); *see Bernier v. Bernier*, 147 U.S. 242, 245 (1893) ("[I]t is a general rule, without exception, in construing statutes, that effect must be given to all their provisions, if such a construction is consistent with the general purposes of the act, and the provisions are not necessarily conflicting."). As the Supreme Court recently explained, the agency's "authority and responsibility to resolve some questions left open by Congress that arise during the law's administration" does not extend to "include a power to revise clear statutory terms that turn out not to work in practice." *Utility Air Regulatory Grp. v. EPA*, 134 S.Ct. 2427, 2446 (2014). "An agency confronting resource constraints may change its own conduct, but it cannot change the law." *Id.*

EPA's faulty line of reasoning has already been considered and refuted by the U.S. Supreme Court in *General Motors Corp. v. United States*, 496 U.S. 530 (1990). In *General Motors*, industry argued that EPA's failure to act on a state implementation plan (SIP) revision within the statutory period for review under section 110 of the Act precluded EPA from enforcing the existing provisions of the SIP under section 113. *Id*. at 535. The Supreme Court rejected the contention that there was a conflict in the statute, holding that delay on the part of EPA does not affect the Agency's ability to enforce the other requirements of the Act. *Id*. at 539-42. In other words, a violation of one provision does not affect the applicability of other requirements unless the statute provides otherwise. *General Motors*, 496 U.S. at 540-42; *see also United States v. James Daniel Good Real Property*, 510 U.S. 43, 63 (1993) ("We have held that if a statute does not specify a consequence for noncompliance with statutory timing provisions, the federal courts will not in the ordinary course impose their own coercive sanction").

Third, to the extent there is any real problem, it is a problem within EPA's ability to manage. There is no reason that modeling cannot be conducted now by permit applicants, pending final promulgation of the ozone standard, that assesses compliance with an ozone standard down to 60 ppb. If a particular proposed project would cause or contribute to a violation of a lower standard it can prepare accordingly without delaying review of the permit.

With no ambiguity or gap in the statutory language, EPA has no ability allow sources to avoid the statutory mandates. Nothing in the statute gives EPA the power to waive the requirements of section 165(a), which are self-effectuating and directly enforceable against the source.

EPA suggests that it has general rulemaking authority in section 301 to alter these otherwise plain requirements. The courts, however, have repeatedly rejected such a notion. Section 301(a)(1) authorizes EPA to "prescribe such regulations as are necessary to carry out [its] functions under [the Act]." 42 U.S.C. § 7601(a)(1). The D.C. Circuit, however, has "consistently held that EPA's authority to issue ancillary regulations is not open-ended, particularly when there is statutory language on point." *NRDC v. EPA*, 749 F.3d 1055, 1063 (D.C. Cir. 2014); *see also Am. Petroleum Inst. v. EPA*, 52 F.3d 1113, 1119 (D.C. Cir.1995) ("the general grant of rulemaking power to EPA cannot trump specific portions of the CAA"); *NRDC v. Reilly*, 976 F.2d 36, 41 (D.C. Cir.1992) (rejecting EPA's use of general rulemaking authority to add to a statutorily specified list); *Sierra Club v. EPA*, 719 F.2d 436, 453 (D.C.Cir.1983) (same). As the court explained: "Th[e]se precedents establish a simple and sensible rule: EPA cannot rely on its gap-filling authority to supplement the Clean Air Act's provisions when Congress has not left the agency a gap to fill." *NRDC*, 749 F.3d at 1064.

EPA's reliance on the fact that it has adopted similar illegal exemptions in the past also does not provide any authority to continue such practices here. *See, e.g.*, *New Jersey v. EPA*, 517 F.3d 574, 583 (D.C. Cir. 2008) ("[P]revious statutory violations cannot excuse the one now before the court."); *F.J. Vollmer Co. v. Magaw*, 102 F.3d 591, 598 (D.C. Cir. 1996) ("[W]e do not see how merely applying an unreasonable statutory interpretation for several years can transform it into a reasonable interpretation.").

Nor does *dicta* from the Ninth Circuit's Avenal decision support the notion that EPA has any authority to waive plain statutory requirements.[646] The Ninth Circuit found the statute unambiguous on the question of whether section 165(a)(3) required sources to demonstrate compliance with the NAAQS in effect at the time of permit issuance. *See Sierra Club*, 762 F.3d at 981 (holding that permitting delay did not endow EPA with authority to waive NAAQS by rewriting unambiguous statutory terms). With no authority to change these unambiguous requirements, that is the end of the statutory analysis.

The Avenal court's continued discussion of past grandfathering exemptions reflects a misunderstanding of EPA's actions. The court seemed to believe that EPA had avoided imposing new NAAQS by adjusting the operative dates of those NAAQS. *See id.* at 983 (noting "[o]n almost every prior occasion, EPA grandfathered a limited set of applications, in effect, by specifying an operative date (or dates) for each new regulation, as it was formally adopted. In contrast to the ad hoc waiver here, the former procedure does not, on its face, violate the plain statutory mandate to enforce whatever regulations are in effect at the time the agency makes a final decision."). As EPA is well aware, it is not proposing to "grandfather" sources by changing the operative date of the new ozone NAAQS. Instead it is waiving "the plain statutory mandate to enforce whatever [NAAQS] are in effect at the time the agency makes a final decision." *Id.* As the Ninth Circuit found, EPA has no such statutory authority.

[646] *See* Proposed Rule at 75,377 (citing *Sierra Club v. EPA*, 762 F.3d 971, 983 (9th Cir. 2014)).

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. Unless a source can meet these requirements, it may not be built. The statute provides no authority for EPA to waive these requirements, so the proposed grandfathering exemption should be dropped from the final rule.

d. Designations

EPA explains that it intends to issue guidance and rules that will help implement the 2015 NAAQS in a timely manner.[647] We applaud this goal. However, EPA's track record on timeliness when it comes to implementing the Clean Air Act is not good. If EPA truly wants to take timely action, it seems like EPA would need to make fundamental changes in its processes in order to achieve this goal.

Specially, EPA claims that it will issue guidance for the designation process within 4 months of promulgating the NAAQS.[648] In order to do that, EPA should be working on that guidance now.

As to designations for the secondary standard, if the standard is mainly driven by the protection of ecologically sensitive areas, EPA should include in its rationale for designations that same considerations. For example, if there is a violating monitor in a multi-county national park or wilderness area, EPA should include the entire national park or wilderness area in the nonattainment area.

Furthermore, for the secondary standard EPA should use a more expansive definition of "nearby" when considering what areas contribute to ambient air quality in a nearby area that does not meet the secondary standard. In the context of intrastate pollution, it is critical that EPA designate areas that contain sources that contributing to nonattainment. For example, if a power plant in a rural area several counties over from a violating monitor in a national park is contributing to that violating monitor, EPA needs to designate the area around the power plant as nonattainment. Failure to do so will undermine the Act's scheme for bringing the nonattaining national park into attainment by excluding the power plant from ozone control mandates that apply only inside the nonattainment area. Therefore, for the secondary standard, nearby should be defined to include anywhere within the state.

In the context of interstate pollution, it is true that there will be CAA §§ 126 and 110(a)(2)(D)(i)(I) to address this situation. However, in practice these tools have been extremely slow to achieve actual results. A much more effective and efficient approach would be to again, in the context of the secondary NAAQS, use a more expansive definition of nearby to go beyond contiguous counties. While this is a more expansive definition, it is not unprecedented. For example, for the 2006 PM2.5 NAAQS, EPA included non-contiguous areas in the Cincinnati nonattainment area to ensure that power plants that where contributing to violating monitors could be appropriately controlled.

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[647] 79 Fed. Reg. at 75,372.
[648] *Id.*

In the context of the primary standard, EPA should seriously reconsider its position on a multistate nonattainment designation as has been repeatedly requested by Mid-Atlantic states such as Delaware and Maryland which receive the majority of their ozone and ozone precursors from upwind states on many days. EPA has repeatedly rejected these requests stating that the Good Neighbor provisions are the appropriate way to deal with this situation. Yet, at the same time, EPA has steadfastly refused to move forward with the timely implementation of the Good Neighbor provisions for the 2008 ozone NAAQS. This does not serve the states' or the public's interest.

e. Background Ozone Levels

EPA includes a discussion of background ozone levels. That is, EPA claims that in some areas on some days, ozone and its precursors from international and natural sources could prevent reaching attainment levels, especially in locations with "few remaining untapped opportunities for local emission reductions."[649] Yet EPA acknowledges that: "modeling indicates that U.S. anthropogenic emission sources are the dominant contributor to the majority of modeled O3 exceedances of the NAAQS across the U.S."[650] EPA also correctly notes that the Clean Air Act already contains provisions to address these situations in the extremely rare case that there is a credible claim.

Moreover—though it is not legally relevant, see API, 661 F.2d at 1185 ("Houston's argument that because natural factors make attainment impossible the Administrator acted arbitrarily and capriciously in setting the primary ozone standard at an 'unattainable' level is addressed in part by our analysis of API's attainability argument. Attainability and technological feasibility are not relevant considerations in the promulgation of national ambient air quality standards.")[651] The significant cost-effective emission reductions available and resultant ambient air quality improvements are discussed in Part VII below.

Background ozone levels are not the issue here. Nevertheless, air pollution needs to be reduced to levels that don't harm public health, period. When EPA sets the standard, by law it must base that decision solely on what it takes to protect public health with an adequate margin of safety. In other words, the only legitimate concern is the impact on human health.

Looking just at natural levels of ozone, spring and summer, when background levels are typically highest, mean levels are found to range from 15-35 ppb, including high elevation sites (where natural background is typically higher).[652] For most locations, median levels fall between 20 -25 ppb. Even when taking into account emissions from outside of the U.S. and during the spring and summer at high elevation, average background levels still do not go above 50 ppb. [653]

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[649] Proposed Rule at 75,382.
[650] *Id*.
[651] To the extent there is any conflict between this case and later cases like ATA III, the "earlier decision"—API—binds. *United States v. Old Dominion Boat Club*, 630 F.3d 1039, 1045 (D.C. Cir. 2011).
[652] Policy Assessment at 2-13.
[653] *Id*. at 2-14.

Background levels of ozone are typically well below air quality standards. [654] Furthermore, it is U.S. man-made emissions that are responsible for pushing concentrations to levels above current and proposed air quality standards – even in high elevation areas.[655] Models estimate that background ozone contributes to between 59 and 66 percent of seasonal mean ozone. Even in Denver, at high elevation, man-made emissions from U.S. sources are responsible for about 45 percent of total ozone. [656] With nearly half, or often more, of ozone stemming from U.S. man-made sources, there is plenty of room to take action.

There are rare events where background sources cause a sudden surge in ambient ozone levels, however, the CAA expressly plans for excluding these emissions. Section 179(b) of the CAA allows for exclusion of air quality data when exceedances result from international emissions. EPA's exceptional events policy allows the agency to exclude ambient monitoring data when calculating design values, and determining attainment, when background levels from natural or international sources cause a spike in emissions.

Even though background ozone levels are not a significant concern here, it is worth noting that EPA can't set air quality standards based on attainability. In 1979, the City of Houston challenged EPA's setting of a one-hour ozone standard at 120 ppb as being too close to natural background levels. This argument was rejected by the court, which found that attainability is not a relevant consideration in setting air quality standards and that EPA does not need to tailor standards to meet the needs of different locations. *API v. Costle*, 665 F.2d 1176, 1185-1186 (D.C. Cir. 1981). In other words, claims that standards are hard to meet because they are close to background levels are nothing new and are not appropriate for EPA to consider in setting a health-protective standard.

In short—background ozone levels are nowhere near proposed air quality standards and if there are exceptional events--cases where emissions from natural or international sources do result in especially high levels—there are processes by which EPA can exclude these from calculations.

f. EPA Must Complete Its Reconsideration of the 2008 Ozone NAAQS

As discussed above in Section II.B, in 2010, EPA proposed to reconsider the 2008 NAAQS.[657] In 2011, EPA abruptly halted the reconsideration process. When several public health and environmental organizations challenged the decision, EPA moved for dismissal on the ground that it was actually just deferring its reconsideration:

> EPA decided to coordinate further proceedings on its voluntary rulemaking on reconsideration with that ongoing periodic review, by deferring the completion of its voluntary rulemaking on reconsideration until it completes its statutorily-

[654] Background levels also fall below EPA's proposed secondary, or welfare-based, standard of from above 15 ppm-hrs to 7 ppm-hrs, expressed in terms of the W126 index, falling below 3 ppm-hrs. Policy Assessment at 2-21.
[655] Policy Assessment at 2-20.
[656] Policy Assessment at 2-16, 2-21.
[657] 75 Fed. Reg. 2,938 (Jan. 19. 2010).

> required periodic review. Because EPA's deferral neither concludes its voluntary rulemaking reconsidering the 2008 Ozone NAAQS nor establishes legal rights or obligations, it does not constitute judicially reviewable final agency action under the Clean Air Act.

EPA Mot. to Dismiss at 2, *American Lung Ass'n v. EPA*, No. 11-1396 (D.C. Cir. Dec. 8, 2011); *accord id.* at 11. The court granted the motion, accepting EPA's characterization of its action as a "non-final decision to defer action on the 2008 voluntary revision of the national ambient air quality standards for ozone." Order, *American Lung Ass'n*, No. 11-1396 (D.C. Cir. Feb. 17, 2012); *see also Mississippi*, 744 F.3d at 1341-42 (summarizing history).

EPA has assured the D.C. Circuit that it would complete the reconsideration of the 2008 NAAQS along with the completion of this mandatory review. EPA must follow through on that commitment and conclude the reconsideration rulemaking as it promised, bearing in mind that it may not consider implementation costs in making determinations on the strength of the NAAQS. *See Whitman*, 531 U.S. at 468-71.

VI. THE NEW NAAQS IS, AT ITS CORE, ALSO AN ENVIRONMENTAL JUSTICE ISSUE: EPA MUST SET A STANDARD THAT IS PROTECTIVE OF MINORITIES AND ELIMINATES THE ROLE THAT OZONE PLAYS IN THEIR HEALTH OUTCOMES

EPA's proposed environmental justice analysis also falls far short of an analysis that complies with the applicable Executive Order. Executive Order 12,898 calls for agencies to "identify[] and address[], 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 and its territories and possessions, the District of Columbia, the Commonwealth of Puerto Rico, and the Commonwealth of the Mariana Islands." Yet EPA acknowledges that the analysis that it performed "cannot be used to draw any conclusions regarding potential disparities in exposure or risk across populations of interest from an EJ perspective."[658] Nevertheless, EPA claims that "EPA believes the human health or environmental risk addressed by this action will not have potential disproportionately high and adverse human health or environmental effects on minority, low-income or indigenous populations because it does not affect the level of protection provided to human health or the environment."[659] This statement is doubly arbitrary: first, by its own admission, EPA has no basis whatsoever for its stated belief; second, the claim that the ozone NAAQS "does not affect the level of protection provided to human health or the environment" is entirely false, for the NAAQS' entire purpose is to protect public health and the environment. EPA's claim that it has done any meaningful environmental justice analysis is thus astounding in its chutzpah.

As EPA sets a new ozone NAAQS standard, it is critical that EPA carefully consider impacts and health outcomes in minority and lower socioeconomic communities. Minority and lower socioeconomic communities--which can and often do overlap—are frequently

[658] U.S. EPA (2014). Regulatory Impact Analysis of the Proposed Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone (EPA-452/P-14-006), at 9A-1 [hereinafter RIA]; *accord id.* at 9A-6.
[659] Proposed Rule at 75,387/1.

disproportionately exposed to higher levels of ozone air pollution, to more types of elevated air pollution and to more chronic air pollution. And, perhaps not surprisingly, minorities and lower socioeconomic communities suffer a disproportionately higher asthma burden in the United States --particularly African-Americans, Puerto Ricans, and Native Americans.

EPA has an Interim Process Guide on how to incorporate environmental justice into its actions.[660] Notably, the Interim Process Guide explicitly contemplates a cumulative impacts analysis, which as discussed below is a highly important part of addressing environmental justice issues. EPA should utilize its Interim Process Guide and engage in a cumulative impacts analysis to ensure that the 2015 Ozone NAAQS standard effectively addresses environmental justice issues.

A. Across the Nation Minorities Disproportionally Live in Higher Levels of Ozone and Thus Will Disproportionately Suffer the Impacts of an Insufficient Ozone Standard

Setting a new ozone standard is inescapably an issue with significant environmental justice implications for a number of reasons. Among other things, more minorities are over represented in areas with lower air quality and higher levels of air pollution that whites. More specifically, by cross referencing census data, EPA's nonattainment designations for the 2008 ozone standard, and levels of ozone (2011-2013 "design values" for the 2008 ozone standard), a clear and persistent trend becomes evident.

African-Americans in particular may be at higher risk of early death from ozone pollution than the general population. Bell et al. (2008) [661] examined 98 urban communities in the U.S. and reported that the risk between ozone and mortality was greatest in areas with high unemployment, a higher percentage of African-Americans, higher public transportation use, and a lower availability of air conditioning. These results indicate that some segments of the population may face higher health burdens of ozone pollution. The mean long-term ozone concentration in this study was 26.8 ppb.

For example, in Tennessee, blacks are approximately 30% overrepresented in areas that fail to meet the 2008 ozone standards, and whites are approximately 30% underrepresented in such nonattainment areas when compared to average state wide racial demographics. Nor is Tennessee alone in this dubious distinction. In the South alone, Alabama, Arkansas, North Carolina, Georgia, Kentucky and Texas all have higher relative concentrations of blacks—sometimes far higher--living in areas that fail to meet the minimum ozone standards than concentrations of whites. This is reflected in the graphs below.

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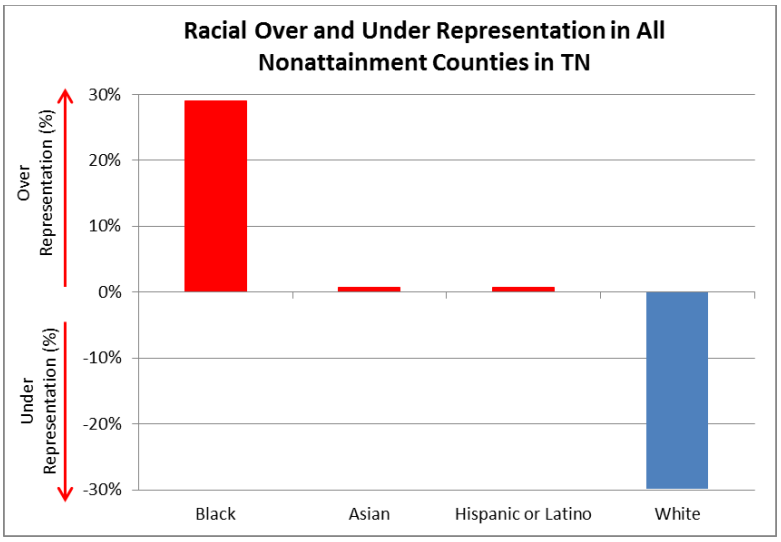
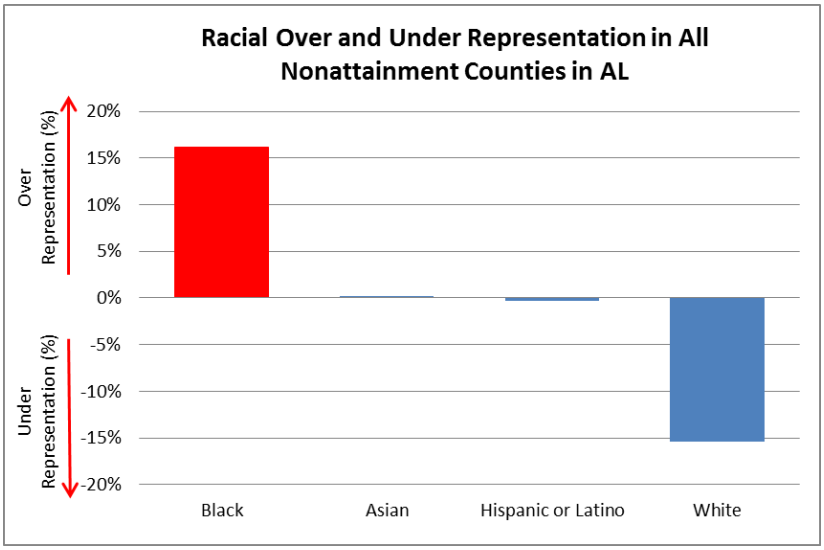
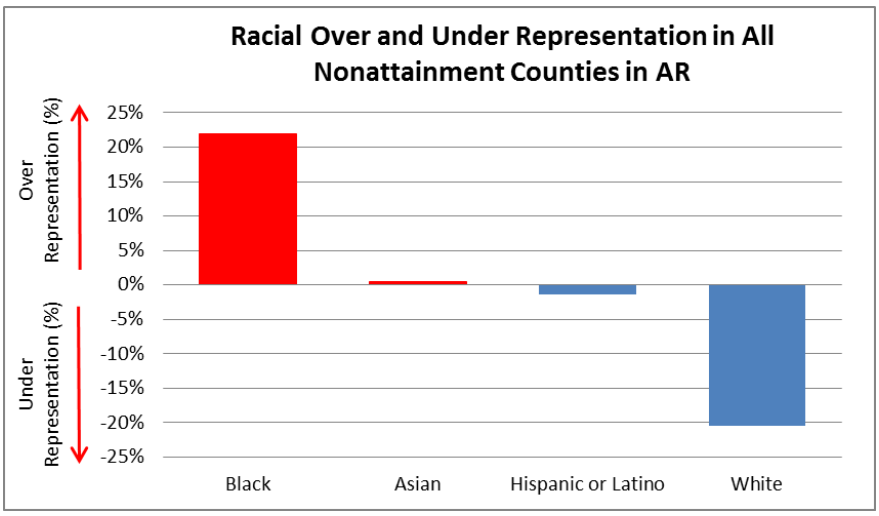
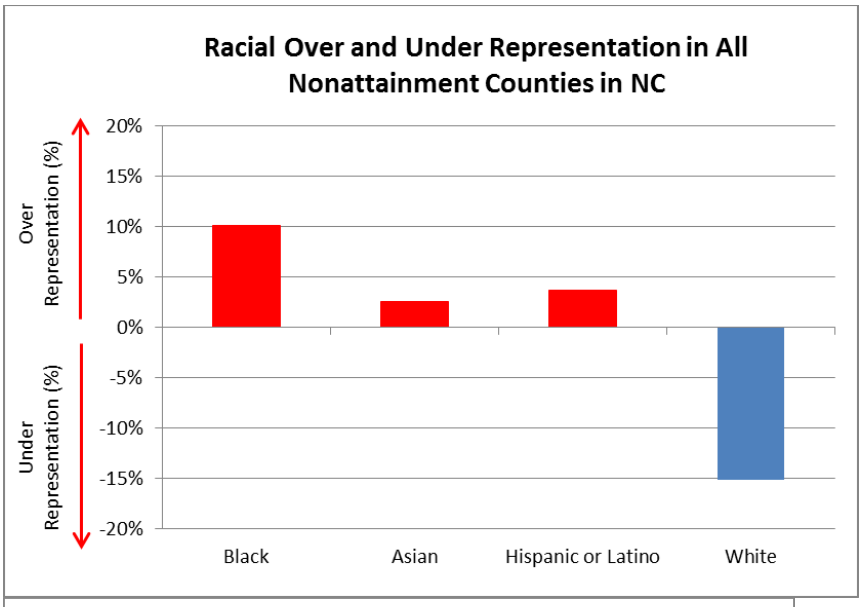
[660] *See* EPA's Action Development Process: Interim Guidance on Considering Environmental Justice During the Development of an Action, http://www.epa.gov/environmentaljustice/resources/policy/considering-ej-in-rulemaking-guide-07-2010.pdf; *see also* EPA, Draft Technical Guidance for Assessing Environmental Justice in Regulatory Analysis, http://www.regulations.gov/#!docketDetail;D=EPA-HQ-OA-2013-0320.

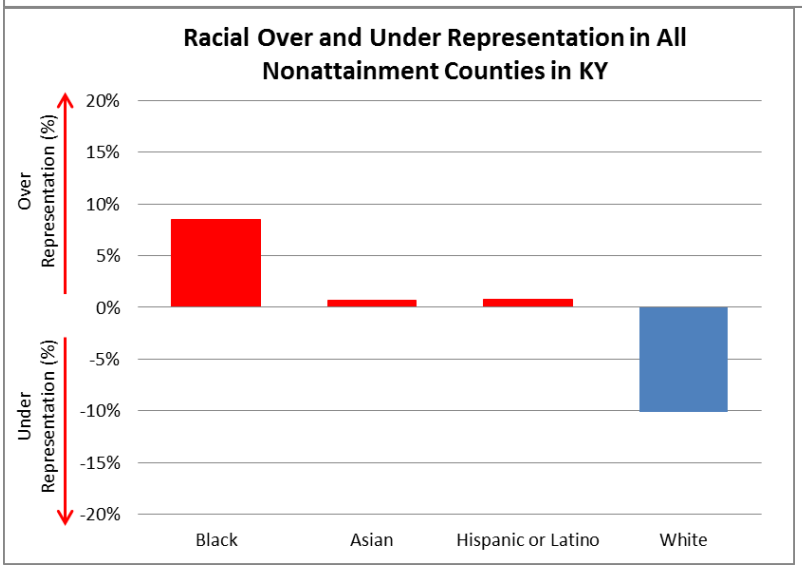
[661] M.L. Bell & F. Dominici (2008). Effect modification by community characteristics on the short-term effects of ozone exposure and mortality in 98 US communities, *Am. J. Epidemiol.*, 167: 086-997.

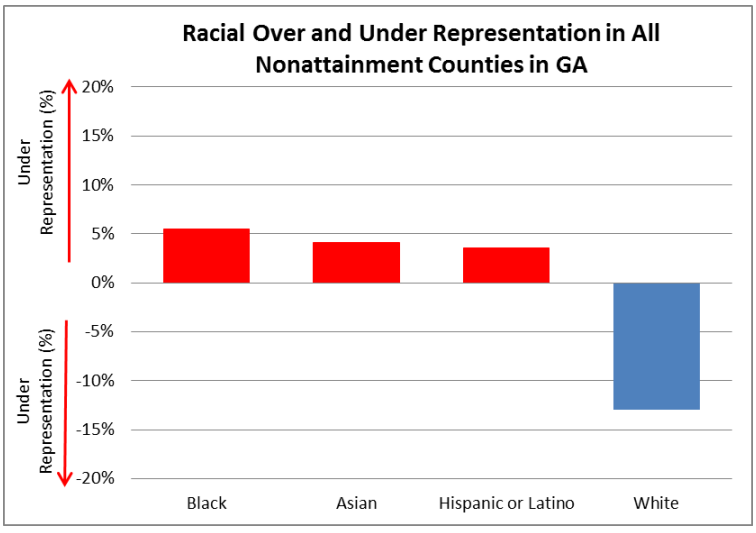

Racial Over and Under Representation in All Nonattainment Counties in TN
Over Representation (%)
Under Representation (%)
30%
20%
10%
0%
-10%
-20%
-30%
Black
Asian
Hispanic or Latino
White

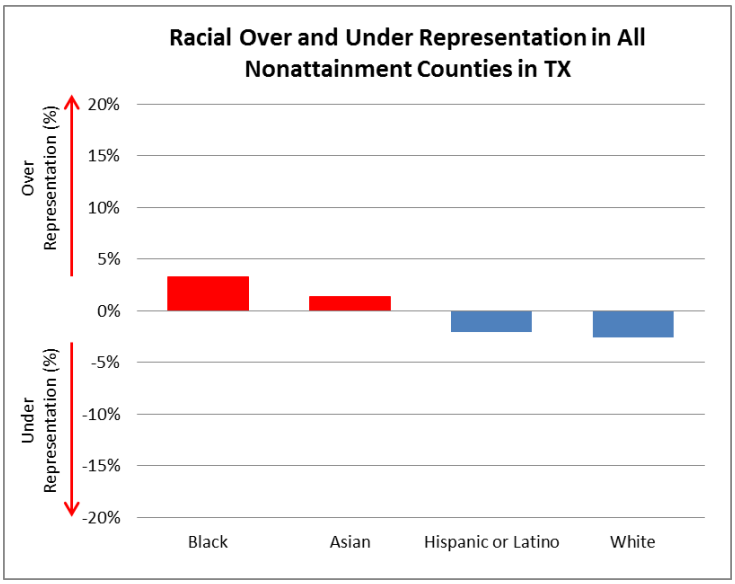

Racial Over and Under Representation in All Nonattainment Counties in AL
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White


Racial Over and Under Representation in All Nonattainment Counties in AR
Over Representation (%)
Under Representation (%)
25%
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
-25%
Black
Asian
Hispanic or Latino
White

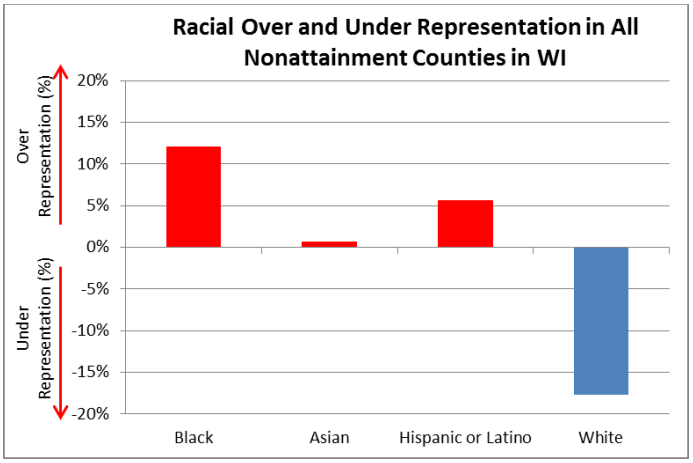

Racial Over and Under Representation in All Nonattainment Counties in NC
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White

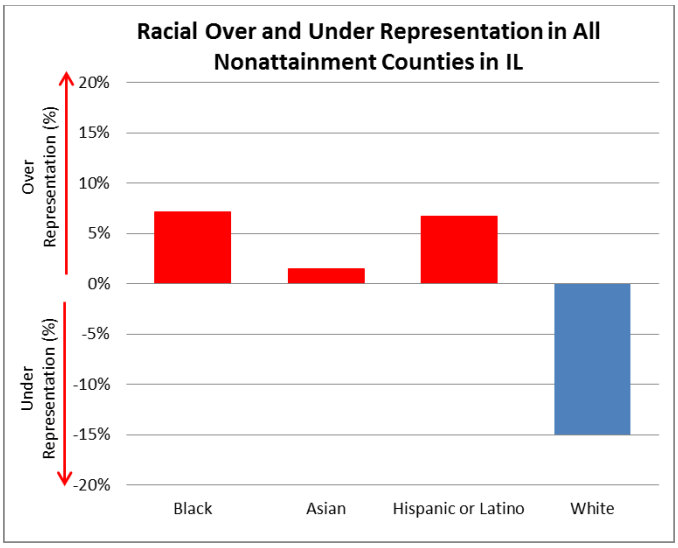

Racial Over and Under Representation in All Nonattainment Counties in KY
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White

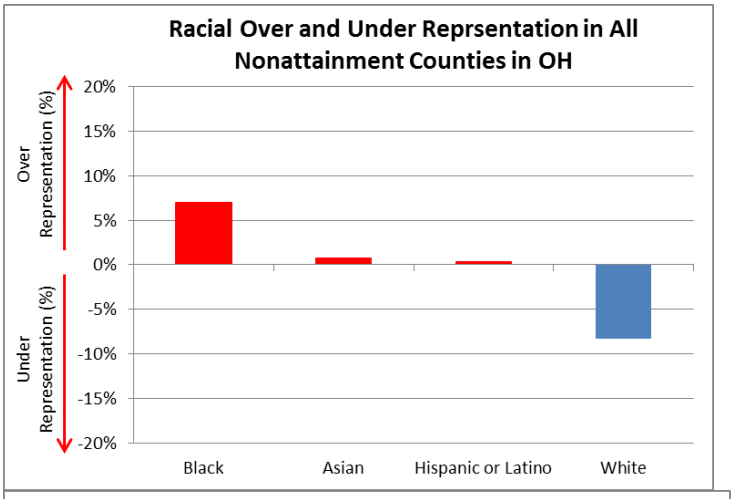

Racial Over and Under Representation in All Nonattainment Counties in GA
Under Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White

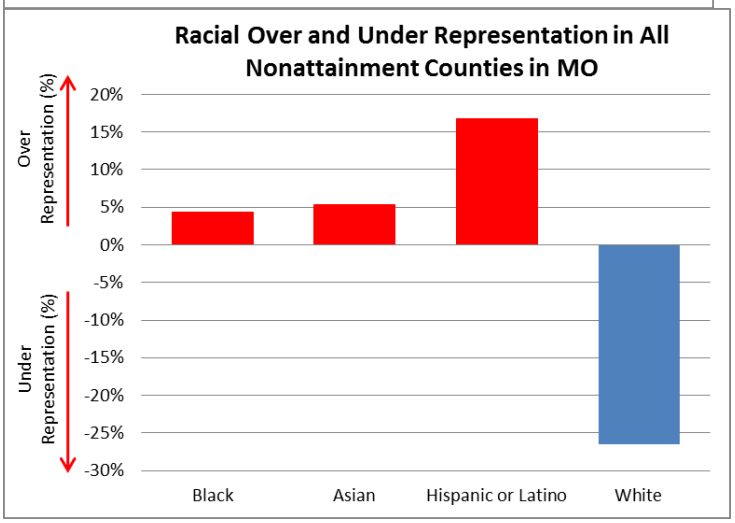


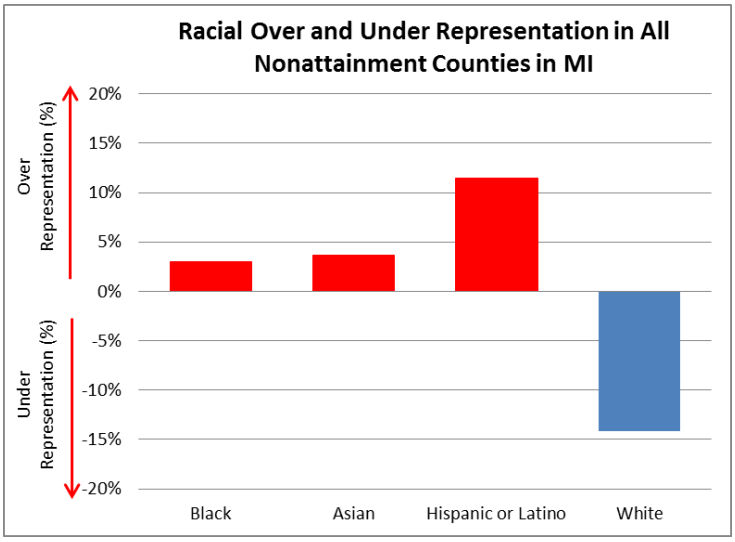
The same holds true for central and mid-west states. As reflected in the graphs below, Wisconsin, Illinois, Michigan, Missouri, Ohio, Kansas, and Oklahoma are particularly notable in the over representation of blacks living in areas that fail to meet minimum air quality standards for ozone. Nevada also follows this trend.

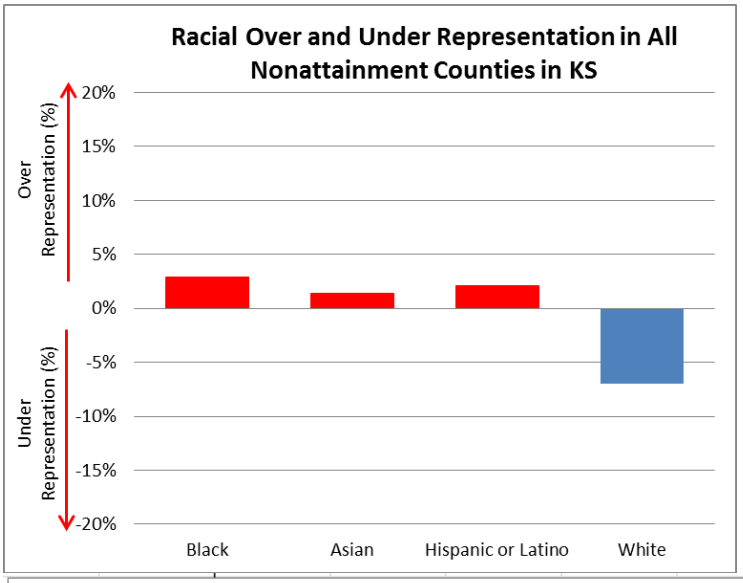


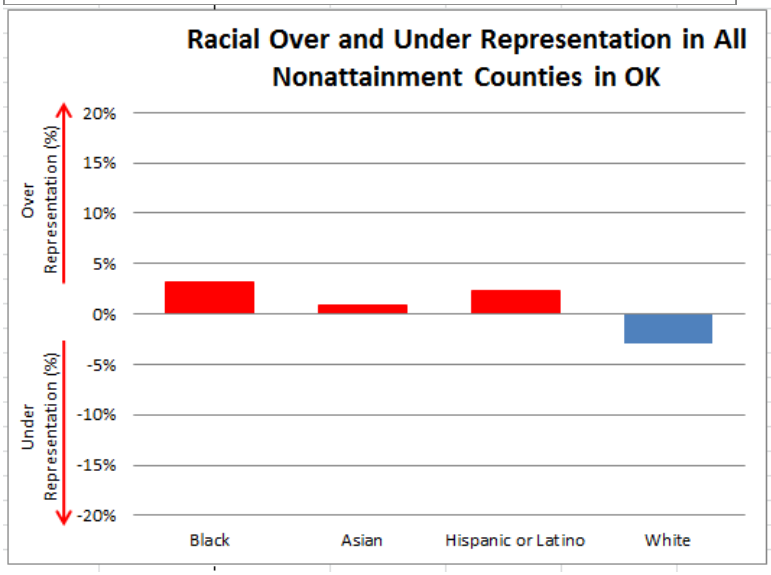

Racial Over and Under Representation in All Nonattainment Counties in IL
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White

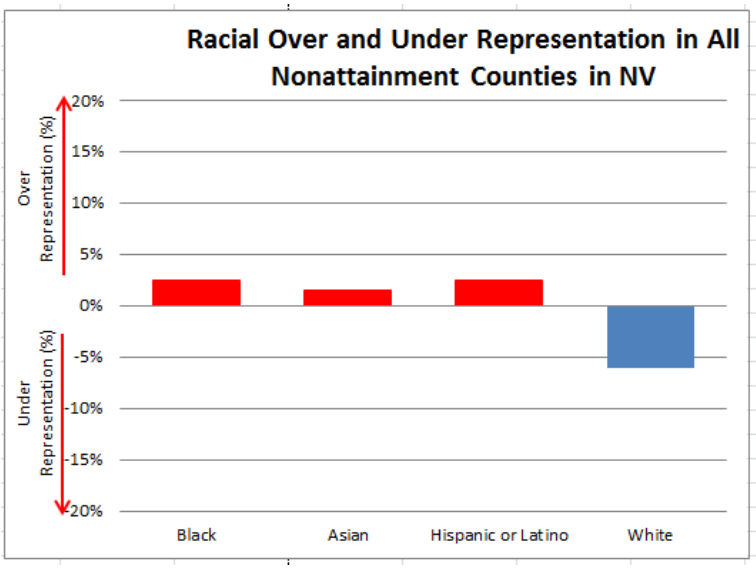

Racial Over and Under Reprsentation in All Nonattainment Counties in OH
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White


Racial Over and Under Representation in All Nonattainment Counties in MO
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
-25%
-30%
Black
Asian
Hispanic or Latino
White

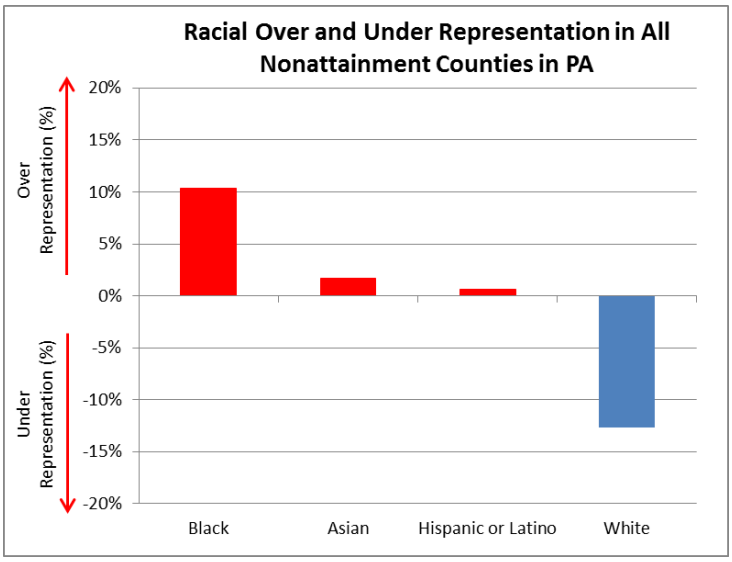

Racial Over and Under Representation in All Nonattainment Counties in MI
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White

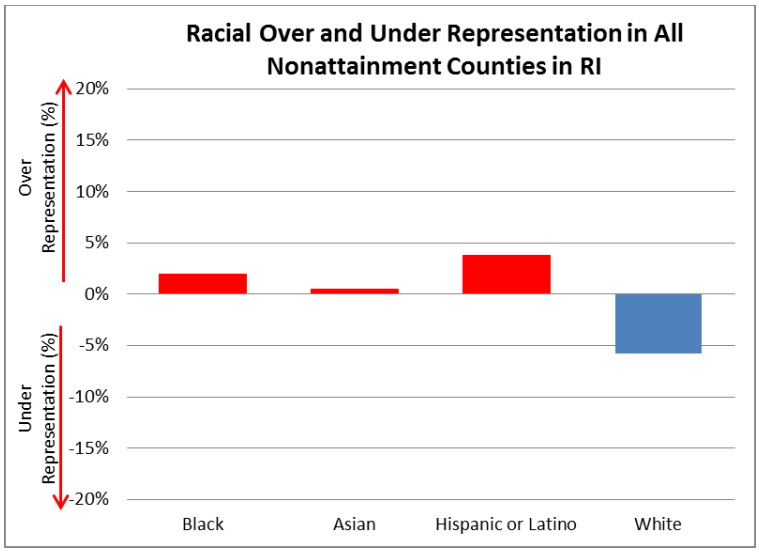

Racial Over and Under Representation in All Nonattainment Counties in KS
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White

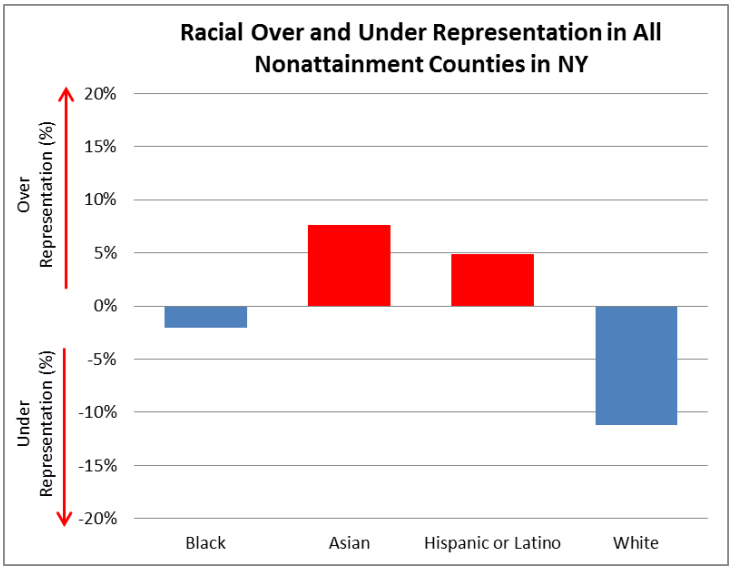

Racial Over and Under Representation in All Nonattainment Counties in OK
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White

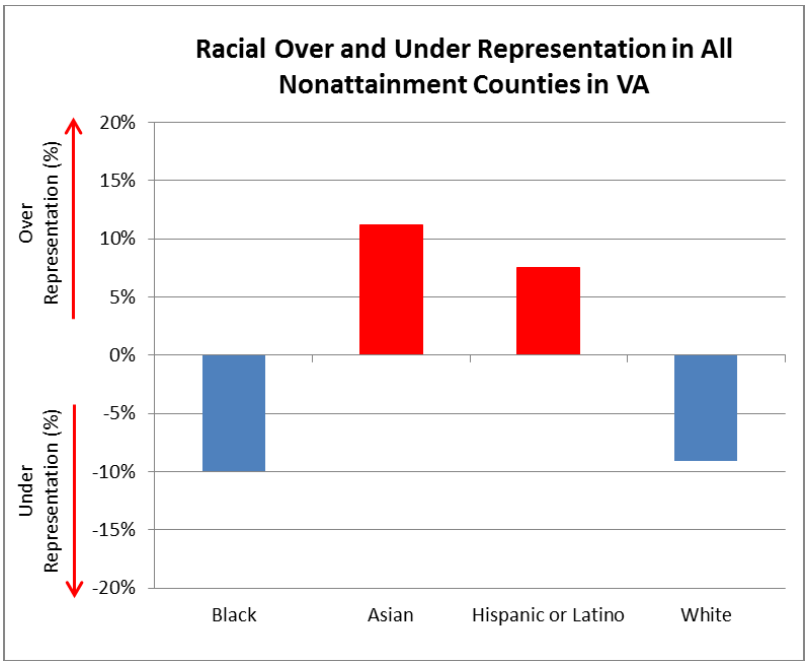


The trend can also be seen in eastern and mid-Atlantic states, where Pennsylvania and Rhode Island have higher relative exposures among blacks than whites, though in other states such as New York and Virginia minorities that are at greater relative exposure than whites are Hispanics and Asians.

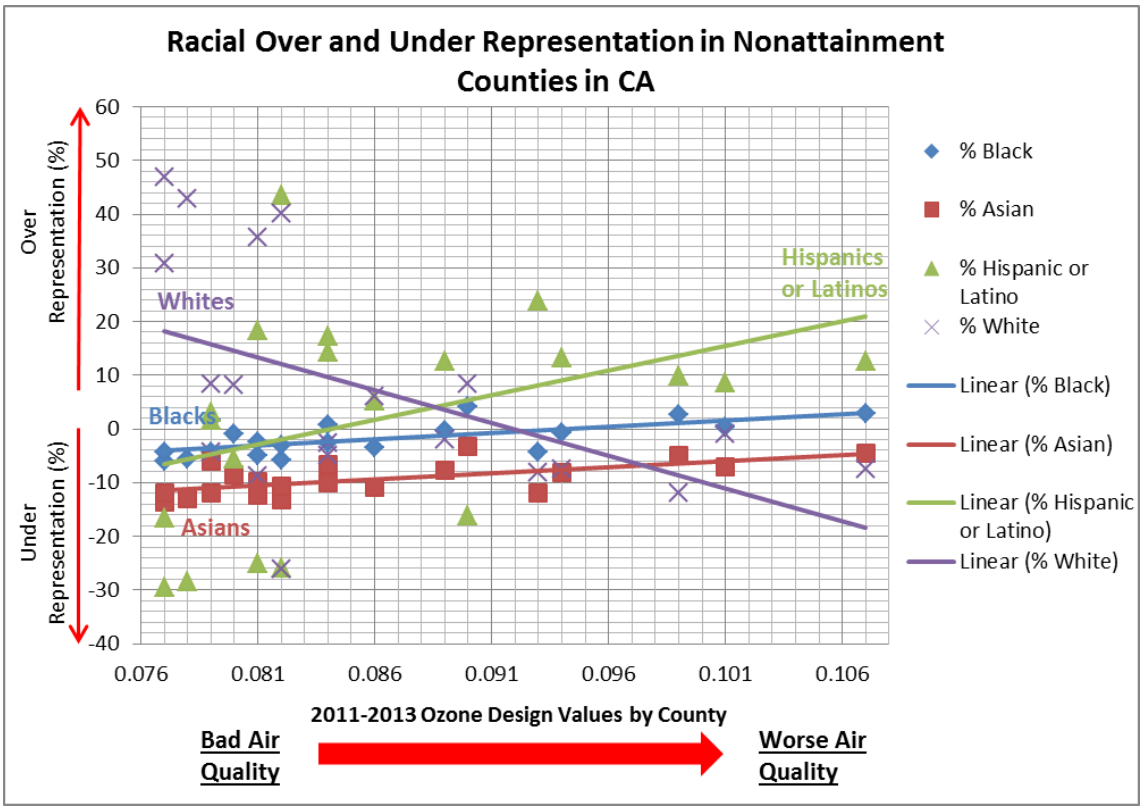


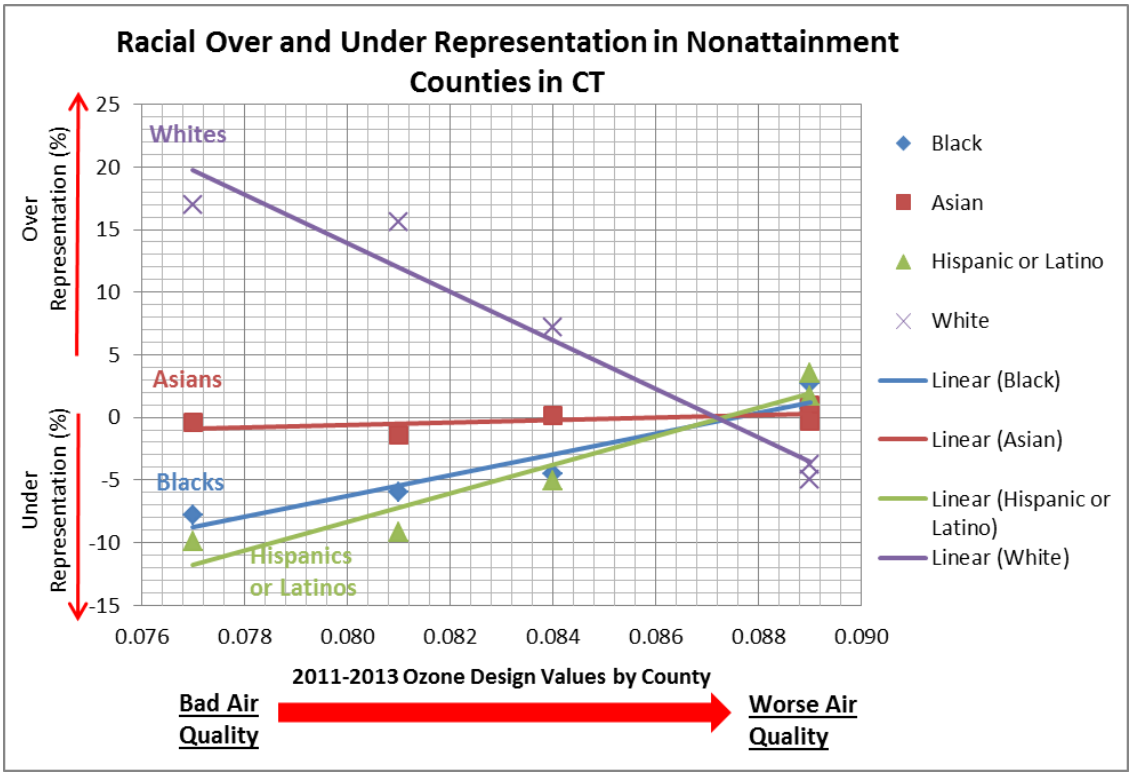

Racial Over and Under Representation in All Nonattainment Counties in RI
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White

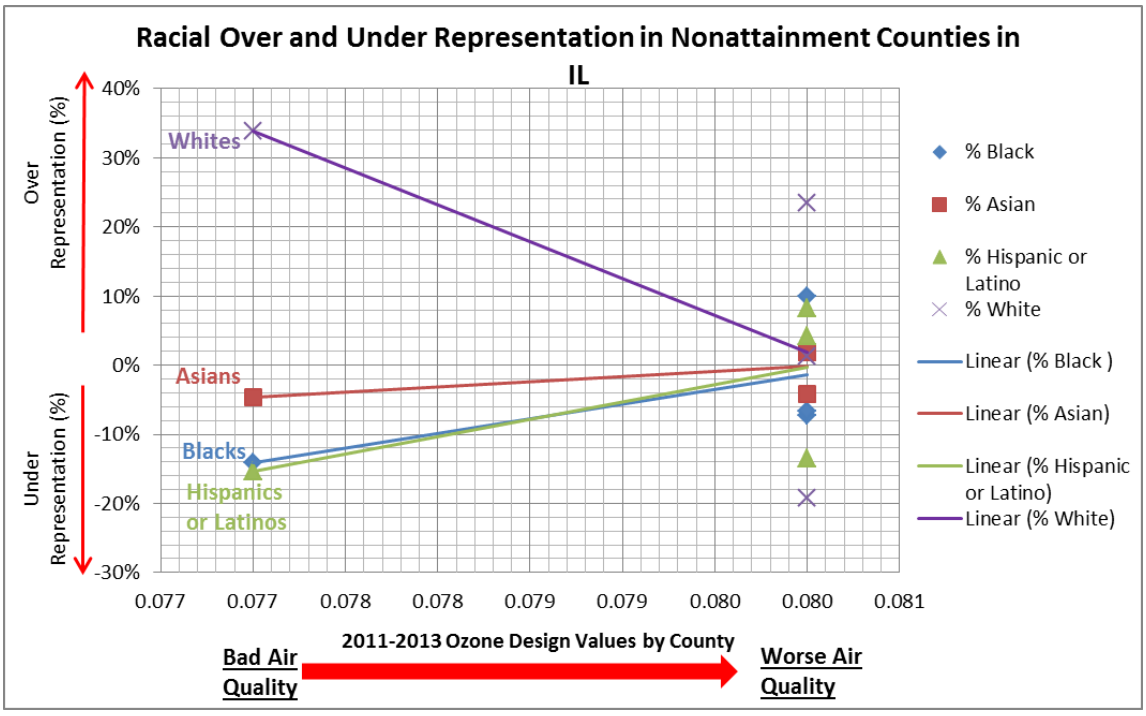

Racial Over and Under Representation in All Nonattainment Counties in NY
Over Representation (%)
Under Representation (%)
20%
15%
10%
5%
0%
-5%
-10%
-15%
-20%
Black
Asian
Hispanic or Latino
White

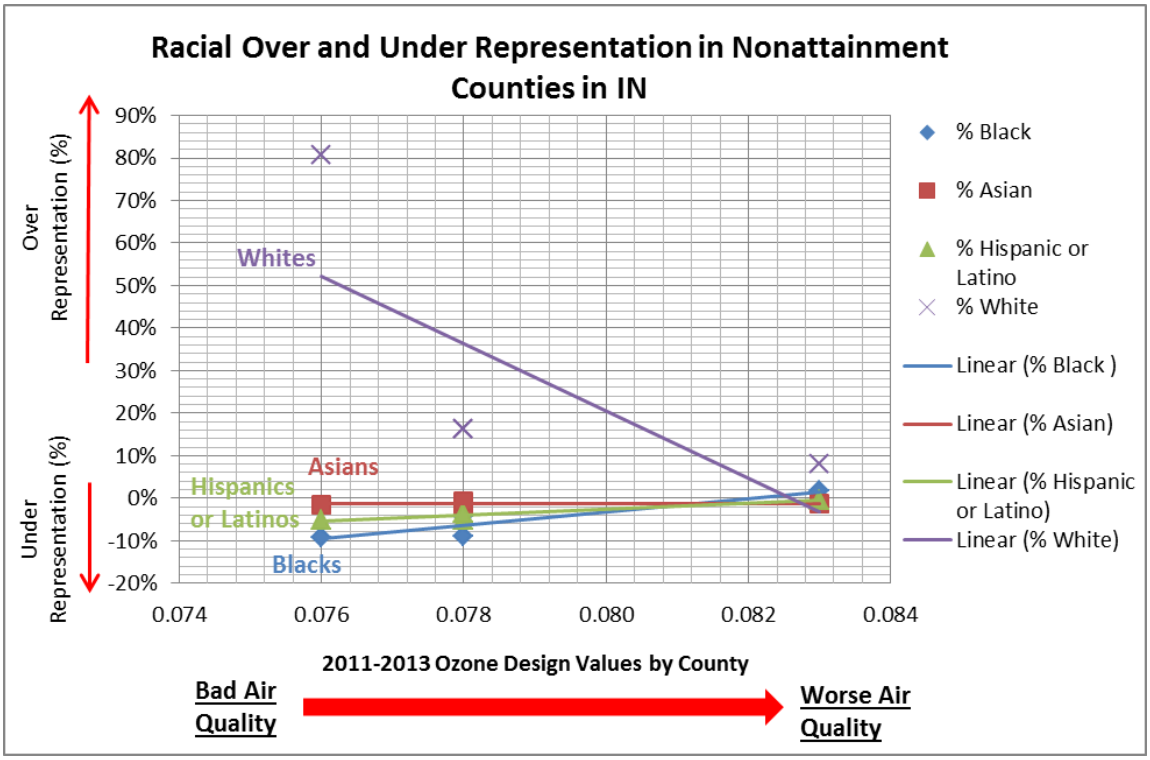


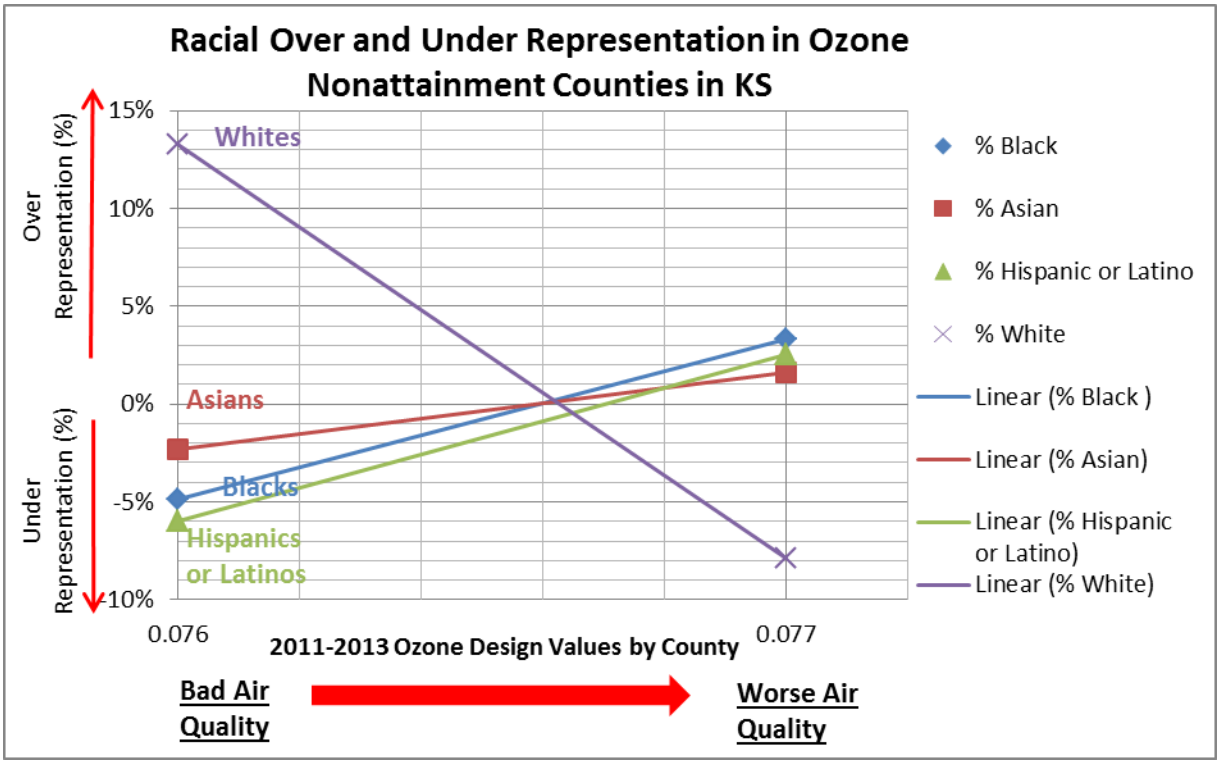
Indeed, as the line graphs below demonstrate, for many states there is a striking correlation between increasing concentrations smog, increasing concentrations of minorities and decreasing concentrations of whites in areas that fail to meet minimum air quality standards. Stated another way, as air quality progressively worsens, representation of blacks and other minorities in the population increases while representation of whites in the population decreases.

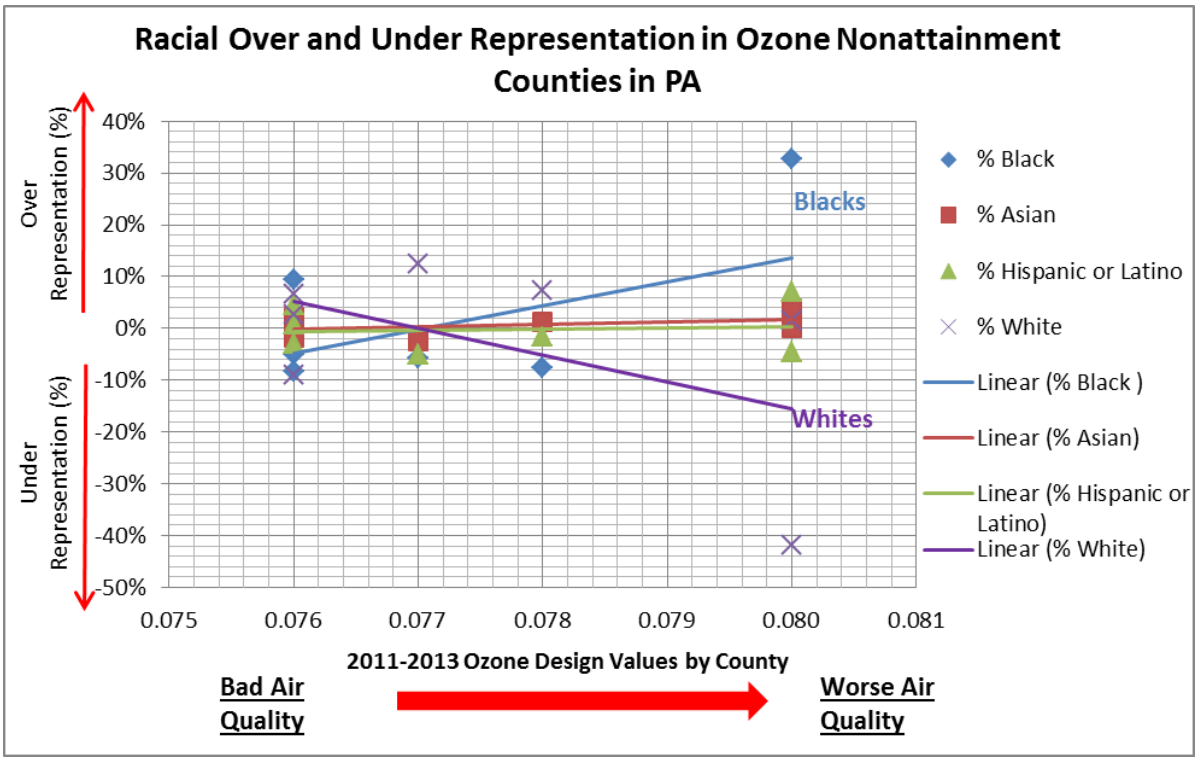


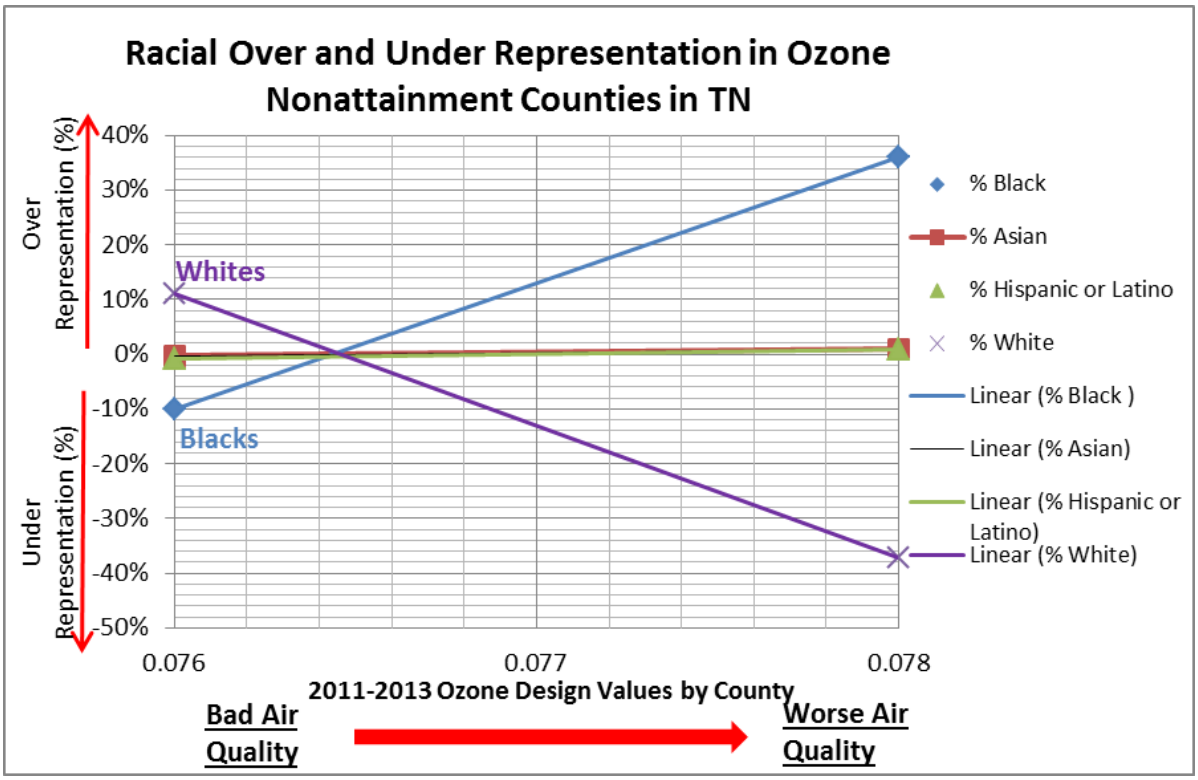

Racial Over and Under Representation in Nonattainment Counties in CT
Over Representation (%)
Under Representation (%)
25
20
15
10
5
0
-5
-10
-15
Whites
Asians
Blacks
Hispanics or Latinos
0.076
0.078
0.080
0.082
0.084
0.086
0.088
0.090
2011-2013 Ozone Design Values by County
Bad Air Quality
Worse Air Quality
Black
Asian
Hispanic or Latino
White
Linear (Black)
Linear (Asian)
Linear (Hispanic or Latino)
Linear (White)

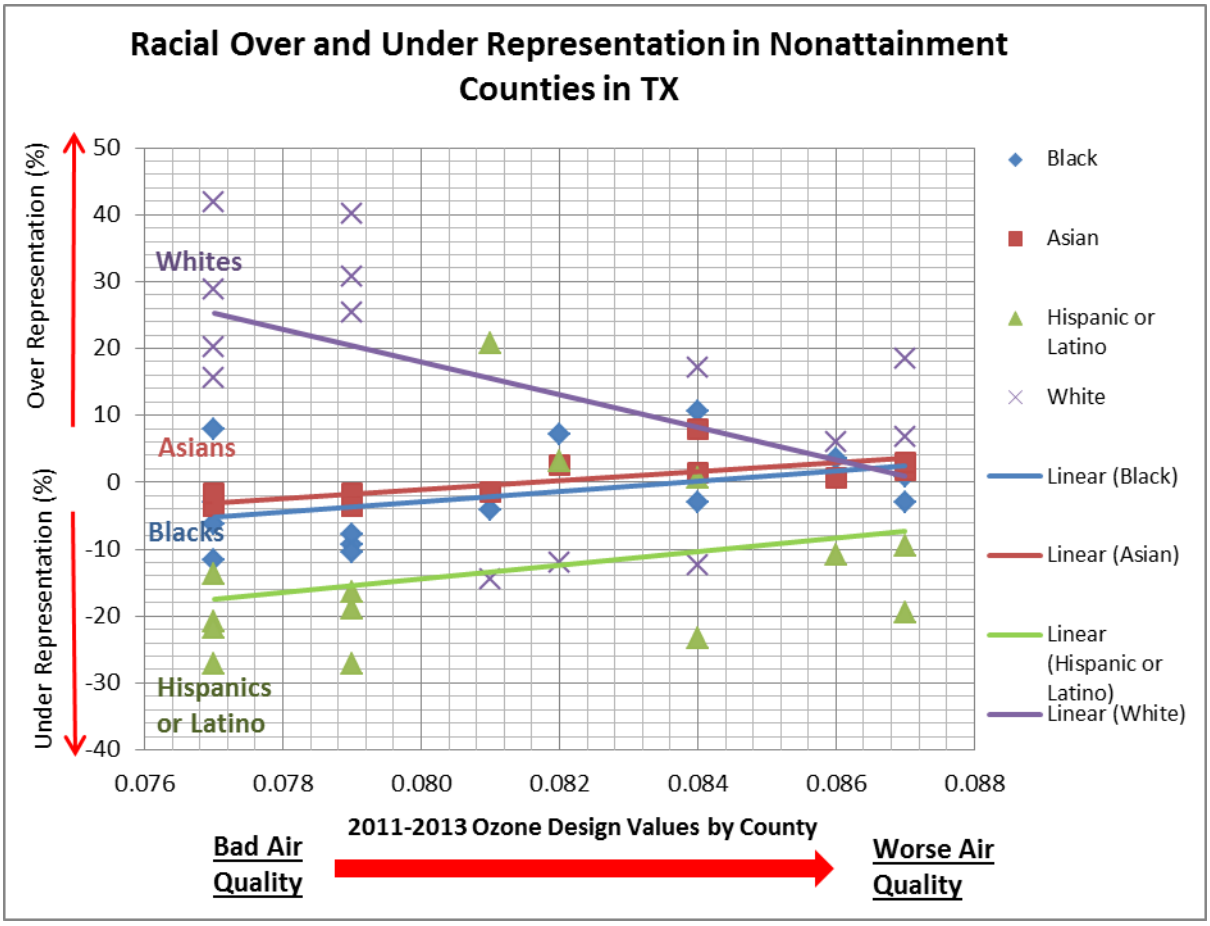

Racial Over and Under Representation in Nonattainment Counties in IL
Over Representation (%)
Under Representation (%)
40%
30%
20%
10%
0%
-10%
-20%
-30%
Whites
Asians
Blacks
Hispanics or Latinos
0.077
0.077
0.078
0.078
0.079
0.079
0.080
0.080
0.081
2011-2013 Ozone Design Values by County
Bad Air Quality
Worse Air Quality
% Black
% Asian
% Hispanic or Latino
% White
Linear (% Black)
Linear (% Asian)
Linear (% Hispanic or Latino)
Linear (% White)


Racial Over and Under Representation in Nonattainment Counties in IN
Over Representation (%)
Under Representation (%)
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
-10%
-20%
Whites
Asians
Hispanics or Latinos
Blacks
0.074
0.076
0.078
0.080
0.082
0.084
2011-2013 Ozone Design Values by County
Bad Air Quality
Worse Air Quality
% Black
% Asian
% Hispanic or Latino
% White
Linear (% Black)
Linear (% Asian)
Linear (% Hispanic or Latino)
Linear (% White)


Racial Over and Under Representation in Ozone Nonattainment Counties in KS
Over Representation (%)
Under Representation (%)
15%
10%
5%
0%
-5%
-10%
Whites
Asians
Blacks
Hispanics or Latinos
0.076
0.077
2011-2013 Ozone Design Values by County
Bad Air Quality
Worse Air Quality
% Black
% Asian
% Hispanic or Latino
% White
Linear (% Black)
Linear (% Asian)
Linear (% Hispanic or Latino)
Linear (% White)


Racial Over and Under Representation in Ozone Nonattainment Counties in PA
Over Representation (%)
Under Representation (%)
40%
30%
20%
10%
0%
-10%
-20%
-30%
-40%
-50%
0.075
0.076
0.077
0.078
0.079
0.080
0.081
Blacks
Whites
% Black
% Asian
% Hispanic or Latino
% White
Linear (% Black)
Linear (% Asian)
Linear (% Hispanic or Latino)
Linear (% White)
2011-2013 Ozone Design Values by County
Bad Air Quality
Worse Air Quality


Racial Over and Under Representation in Ozone Nonattainment Counties in TN
Over Representation (%)
Under Representation (%)
40%
30%
20%
10%
0%
-10%
-20%
-30%
-40%
-50%
0.076
0.077
0.078
Whites
Blacks
% Black
% Asian
% Hispanic or Latino
% White
Linear (% Black)
Linear (% Asian)
Linear (% Hispanic or Latino)
Linear (% White)
2011-2013 Ozone Design Values by County
Bad Air Quality
Worse Air Quality



B. Setting An Ozone Standard That Protects Environmental Justice Communities Requires EPA To Consider The Cumulative Impacts Including Synergistic Exposures To Multiple Pollutants

It is critical to consider the cumulative impacts of multiple stressors when assessing health impacts, including a population's exposure to multiple pollutants, exposure to higher levels of multiple pollutants, and chronic exposure to lower levels of multiple pollutants. This is particularly important if the 2015 Ozone NAAQS is to adequately address environmental justice issues, as it must.

As CASAC noted in its June 2014 letter to EPA concerning the promulgation of the 2015 ozone NAAQS:

> EPA should consider how review and revision of the NAAQS can be done synergistically for logical, scientifically relevant groupings of criteria pollutants. For example, O3 and NO2 are both criteria pollutants that are inter-related via atmospheric chemistry, and human exposure to these pollutants is often in the form of a mixture that includes both, and other pollutants such as particulate matter. The National Research Council and the North American Research Strategy for Tropospheric Ozone have both made detailed recommendations for

> multipollutant approaches to air quality management. . . CASAC encourages EPA to explore multipollutant approaches for review of the primary standards . . .[662]

This is particularly true when evaluating sensitive sub-groups such as minority communities and low-income communities that frequently experience higher exposure to air pollution and disproportionate impacts.[663] Minorities and lower income communities are more likely to live or work near pollution sources and to have higher pollution burdens from mobile and stationary sources, which are only exacerbated by factors such as health care access, housing market dynamics, and predisposed traits.[664] These higher pollution burdens are associated with health outcomes such as respiratory and cardiovascular disease, low birth weight, and premature mortality.[665]

Epidemiological studies similarly suggest that socioeconomic status ("SES") is associated with higher risks of ozone-related health outcomes.[666] EPA concludes that "most studies of individuals have reported that individuals with low SES and those living in neighborhoods with low SES are more at risk for O3-related health effects, resulting in increased risk of respiratory hospital admissions and ED visits."[667] For example, a New York City study showed that children with lower socioeconomic status had greater risk of ozone-induced hospital admissions for asthma.[668] Accordingly, the ISA noted that "evidence is suggestive of SES as a factor affecting risk of O3-related health outcomes."[669]

To be sure, controlled human exposure studies are valued for their ability to control and eliminate confounding factors such as temperature, co-pollutants, or allergens and the epidemiological studies EPA relies upon are subjected to rigorous statistical analysis to control for confounding effect of multiple pollutant exposures.[670] Yet in the real world, physiological impacts are likely to be even worse than what is experienced in the exposure studies *because* of the addition of these other factors. The combined effects among air pollutants produce important

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[662] CASAC Letter 2014a at v.
[663] Policy Assessment at 1-15; ISA at 8-1, 8-2, 8-2.
[664] Morello-Frosch et al. (2011). Understanding the Cumulative Impacts of Inequalities in Environmental Health: Implications for Policy, *Health Affairs*, 30(5): 879-887.
[665] American Lung Association, State of the Air-Disparities in the Impact of Air Pollution (2013), *available at* http://www.stateoftheair.org/2013/health-risks/health-risks-disparities.html#_ftn1.
[666] S. Lin, X. Liu, L.H. Le, & S. Hwang (2008). Chronic exposure to ambient ozone and asthma hospital admissions among children, *Env. Health Perspect.*, 116(12): 1725-1730.; J.T. Lee, J.Y. Son, H. Kim, & S.Y. Kim (2006). Effect of air pollution on asthma-related hospital admissions for children by socioeconomic status associated with area of residence, *Arch. Environ. Occup. Health*, 61(3): 123-120; S. Cakmak, R.E. Dales, M.A. Rubio, M& C.B. Vidal (2011). The risk of dying on days of higher air pollution among the socially disadvantaged elderly, *Environ. Res.*, 111(3): 388-393; M. Pastor, R. Morello-Frosch, & J. Sadd (2010). Air Pollution and Environmental Justice: Integrating Indicators of Cumulative Impact and Socio-Economic Vulnerability into Regulatory Decision-Making, California Air Resources Board.
[667] ISA at 8-27.
[668] Lin. et al. 2008, *supra* note 654.
[669] *Id.* at 8-28.
[670] *See* Proposed Rule at 75,251: "Most O3 effect estimates for lung function were robust to adjustment for temperature, humidity, and copollutants such as PM2.5, PM10, NO2, or SO2."

physiological effects.[671] Air pollutants are inhaled as a mixture of different sources, yet focus has historically been placed on monitoring and regulating individual pollutants in isolation.[672]

Research is beginning to show how the cumulative effects of environmental stressors can work to produce health disparities and regulatory agencies, including EPA, have begun considering ways of addressing cumulative impacts in decision-making.[673] As EPA notes,

> "For example, although exposure to ozone may primarily target the respiratory system, "real-world" combined exposures to particulate matter, ozone and hazardous air pollutants may affect multiple target organs. Multipollutant exposures may elicit acute, adaptive responses across the respiratory, cardiac, vascular, immunologic, neurological and other organ systems."[674]

In taking a precautionary approach to protect the health of at-risk groups, EPA should not consider ozone exposure in isolation, but should also consider the combined burdens of multi-pollutant exposure and additional environmental stressors. Indeed, doing so is consistent with EPA's mandate to set a NAAQS standard that protects the public health with an adequate margin of safety. And EPA can use its cumulative impacts framework as a roadmap for doing so.[675]

C. The Environmental Justice Implications of Setting the 2015 NAAQS Are Evident in the Asthma Burdens of Minorities, Including in Particular African-Americans

The environmental justice implications of the 2015 Ozone NAAQS is evident in the asthma burdens of minority populations. To be sure, ozone is not the only cause of asthma, nor the only thing that can trigger an asthma attack. However, research has shown that it is an important cause of asthma incidence, prevalence and attacks. As such, the level of ozone that EPA decides to allow under the 2015 Ozone NAAQS is clearly of immediate and direct importance to the health of environmental justice communities.

In absolute number terms, African-Americans are most heavily burdened by asthma in the U.S. Nationally, the current asthma prevalence rate for non-Hispanic blacks is 11.9%, compared to 8.1% for non-Hispanic whites and 7.0% for Hispanics.[676] While the prevalence rate reflects a relatively significant disparate impact, it actually understates asthma's true burden on the African-American community. Other key statistical measures of asthma's impact – including

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[671] J. Mauderly& J. Samet (2009). Is there Evidence for Synergy Among Air Pollutants in Causing Health Effects?, *Environ. Health Perspect.*, 117(1):1-6; ISA sec. 4.3.4.
[672] U.S. EPA, Exposure and Health Effects of Mixtures of Air Pollutants, *available at* http://www2.epa.gov/air-research/exposure-and-health-effects-mixtures-air-pollutants (accessed Mar. 16, 2015).
[673] D.O. Johns et al. (2012). Practical Advancement of Multipollutant Scientific and Risk Assessment Approaches for Ambient Air Pollution, *Env. Health Perspectives*, 120(9): 1238-1242 (2012).
[674] U.S. EPA, Exposure and Health Effects of Mixtures of Air Pollutants.
[675] *See* U.S. EPA (2003), Framework for Cumulative Risk Assessment (EPA/630/P-02/001F) *available at* http://www2.epa.gov/sites/production/files/2014-11/documents/frmwrk_cum_risk_assmnt.pdf.
[676] CDC, Asthma Surveillance Data, *available at* http://www.cdc.gov/asthma/asthmadata.htm (accessed Mar. 13, 2014).

hospitalization rates, emergency department visit rates, and mortality rates – show a much starker contrast. These other measures typically show disproportionate impacts of approximately 200-400% when comparing non-Hispanic blacks to non-Hispanic whites. The following table, which includes statistics from states that have recent data in at least three of the four major categories, illustrates this pattern:

State	Current Prevalence among Adults		Hospitalization Rate*		Emergency Department Visit Rate*		Mortality Rate*	
	White Non-Hispanic	Black Non-Hispanic	White Non-Hispanic	Black Non-Hispanic	White Non-Hispanic	Black Non-Hispanic	White Non-Hispanic	Black Non-Hispanic
Connecticut[677]	8.3%	15%	86	405	342	1273	0.77	2.61
Texas[678]	9.2%	10.2%	88	195	N/A	N/A	1.0	1.9
North Carolina[679]	7.2%	10%	75	210	N/A	N/A	0.68	1.8
Indiana[680]	8.7%	13.7%	85	306	344	1293	N/A**	N/A**
Wisconsin[681]	8.6%	15.9%	63	346	N/A	N/A	0.79	3.54

* Per 100,000 persons

**Indiana data provides raw mortality numbers but not mortality rates. In 2011, 73 Indiana residents died from asthma, 54 of whom were white and 18 of whom were black. African-Americans thus comprised approximately 24% of asthma deaths despite accounting for only 9% of Indiana's total population.

As the data summarized in the table above shows, asthma's disproportionate impact is greater for the most serious, life-threatening asthma-related complications. In Wisconsin, the mortality rate for non-Hispanic blacks is nearly 450% higher than the mortality rate for non-Hispanic whites. Connecticut's hospitalization rate discrepancy is also well over 400%. Even the states listed above that have the most equitable asthma burdens – North Carolina and Texas – have near or over a 200% discrepancy in both hospitalization rates and mortality rates. Stated another way, current prevalence rate is a measure of who has been recently diagnosed with asthma, while the other statistical categories are measures of who suffers the worst asthma-related complications (those complications that necessitate hospital visits or result in death). And the data shows that not only are African Americans more likely to have asthma, but even among asthma sufferers, they are more likely to have worse outcomes: not all individuals who

[677] Connecticut Dept. of Health (2012). The Burden of Asthma in Connecticut 2012 Surveillance Report, *available at* http://www.ct.gov/dph/lib/dph/hems/asthma/pdf/full_report_with_cover.pdf.
[678] Texas Dept. of State Health Services, Asthma Health Facts 2011, *available at* https://www.dshs.state.tx.us/asthma/data.shtm#New_Asthma (accessed Mar. 13, 2014),..
[679] North Carolina Dept. of Health and Human Services (2010).The Burden of Asthma in north Carolina 2010, available at http://www.asthma.ncdhhs.gov/docs/TheBurdenOfAsthmaInNorthCarolina-2010.pdf; North Carolina Dept. of Health and Human Services, African Americans and Asthma in North Carolina (Mar. 12, 2014), http://www.asthma.ncdhhs.gov/docs/factsheets/2011/AfricanAmericansAndAsthmaInNorthCarolina.pdf.
[680] [680] Indiana State Dept. of Health, Asthma Fact Sheet, *available at* http://www.in.gov/isdh/files/ISDH_FactSheet_Asthma_Nov2013_FINAL(1).pdf (accessed Mar. 123, 2014).
[681] Wisconsin Dept. of Health (2013). Burden of Asthma in Wisconsin 2013.

have asthma suffer from it equally. A higher percentage of African-Americans have asthma, but an even higher percentage suffer from its most serious symptoms and complications.

Minority groups other than African-Americans are also disproportionately affected by asthma. Nationally, Puerto Ricans and American Indians/Native Alaskans have a much higher current asthma prevalence rate than even African-Americans, at 16.7% and 14.3% respectively.[682] [683] In Hawaii, the prevalence rate for Native Hawaiians is 14.9%, compared to only 9.0% for whites living in Hawaii.[684] Asthma's heavy burden on these groups is also evident from other statistical measures. Nationally, the mortality rate for Puerto Ricans is four times higher than the mortality rate for whites.[685] Similar trends exist at the state level for Native Americans. In Oregon and Wisconsin, for example, the American Indian hospitalization rate is double the rate for non-Hispanic whites.[686] And while asthma prevalence among the total Hispanic population is actually lower than the national average, Hispanics also have higher hospitalization and mortality rates than non-Hispanic whites, and thus also suffer disproportionately.[687] Hispanics are 30% more likely to visit the hospital for asthma, as compared to non-Hispanic whites, and Hispanic children are 40% more likely to die from asthma.

As is evident from the statistics, asthma has an extremely disproportionate impact on minorities across all parts of the United States. It is undisputed that ozone is a trigger for asthma attacks. And the ozone standard that CASAC and EPA develop must be protective of minorities and eliminate the role that ozone plays in the above health outcomes.

VII. ADDITIONAL CONSIDERATIONS

A. Emission Reductions Needed to Improve Air Quality Are Readily Available

As discussed in Section II above, the Supreme Court has been absolutely clear that the primary NAAQS must be established based on health considerations alone and must not be based on cost or technological feasibility. *Whitman v. Am. Trucking Ass'ns*, 531 U.S. 457, 464-71 (2001); *see also Am. Lung Ass'n v. EPA*, 134 F.3d at 389 (Administrator must promulgate national standards that limit emissions sufficiently to establish that margin of safety "without reference to cost or technological feasibility"). Even if they were relevant, however, industry statements regarding both the feasibility of meeting a lower ozone standard and the costs of doing so are greatly overstated. Significant cost-effective VOC and NOx reductions are readily

[682] The Office of Minority Health, Asthma and American Indians/Alaskan Natives, *available at* http://minorityhealth.hhs.gov/templates/content.aspx?lvl=3&lvlID=532&ID=6172 (accessed Mar. 13, 2014).
The Office of Minority Health, Asthma and Hispanic Americans, *available at* http://minorityhealth.hhs.gov/templates/content.aspx?lvl=3&lvlID=532&ID=6173 (accessed Mar. 13, 2014),..
[684] Hawaii State Dept. of Health, Hawai'i Asthma Plan 10 (2013).
[685] The Office of Minority Health, Asthma and Hispanic Americans (Mar. 13, 2014), http://minorityhealth.hhs.gov/templates/content.aspx?lvl=3&lvlID=532&ID=6173.
[686] Oregon Health Authority, Asthma Emergency Department Visits and Hospitalizations (Mar. 12, 2014), *available at* https://public.health.oregon.gov/DiseasesConditions/ChronicDisease/Asthma/Documents/burden/ch7.pdf (Mar. 12, 2014); Wisconsin Dept. of Health 2013, *supra*.
[687] The Office of Minority Health, Asthma and Hispanic Americans, *supra*.

available using available and proven control technologies, many of which are already employed on major emission sources and are simply not being operated consistent with best prior levels.

1. Significant Emission Reductions Are Already Underway and Further Emission Reductions Are Readily Achievable

Ozone forms when nitrogen oxides and volatile organic compounds react in the presence of heat and sunlight. Over 70% of annual NOx emissions in the United States come from two source sectors: mobile sources and coal fired power plants.[688] Significant emission reductions are already underway in both of these source sectors due to the ongoing implementation of existing rules and the turnover of the vehicle fleet.

EPA in multiple rulemakings has identified significant reductions in ozone levels that are occurring and will continue across the country. In its Regulatory Impact Analysis for the Cross-State Air Pollution Rule (CSAPR), EPA projected that in the absence of the rule, total NOx would decline by over 7 million tons between 2005 and 2014, that EGU NOx would decline from over 3.7 million tons to under 2.1 tons, and that the rule would drive an incremental 200,000 tons of annual NOx reductions.[689] EPA stated that these "significant aggregate reductions" in EGU NOx emissions "would lower overall ambient levels of . . .ozone across much of the eastern U.S."[690] Due to litigation delays, these emission reductions have not been fully implemented to date, so additional reductions from CSAPR can be anticipated over the next two years.[691]

Also in the EGU sector, implementation of the Best Available Retrofit Technology (BART) and Reasonable Further Progress (RFP) requirements of the regional haze provisions of the Clean Air Act have resulted State and Federal Implementation Plans are driving significant further reducing emissions of NOx. BART and RFP requirements are driving numerous control installations of Selective Catalytic and Selective Non-Catalytic Reduction (SCR and SNCR) technology. BART determinations for the following units drove installation of SCR or SNCR.

Table 30: BART Determinations Driving SCR or SNCR

State	Facility	BART determ.
AZ	AEPCO Apache Unit 2	**Gas conversion**
AZ	AEPCO Apache Unit 3	**SNCR**
AZ	APS Cholla Unit 2	**SCR + LNB/OFA**
AZ	APS Cholla Unit 3	**SCR + LNB/OFA**
AZ	APS Cholla Unit 4	**SCR + LNB/OFA**

[688] U.S. EPA, 2011 National Emission Inventory, *available at* http://www.epa.gov/ttnchie1/net/2011inventory.html.
[689] U.S. EPA, Office of Air and Radiation, Regulatory Impact Analysis for the Federal Implementation Plans to Reduce interstate Transport of Fine Particulate Matter and Ozone in 27 States; Correction of SIP Approvals for 22 States, 51 Tbl. 3-7, 55 Tbl. 3-10 (June 2011).
[690] *Id.* at 74.
[691] In December 2014, EPA issued rulemaking establishing a revised implementation schedule for CSAPR, which shifted CSAPR's original 2014 compliance deadline out to 2017. *See* EPA, Rulemaking to Amend Dates in Federal Implementation Plans Addressing Interstate Transport of Ozone and Fine Particulate Matter, 79 Fed. Reg. 71,663 (Dec. 3, 2014). (See Exhibit 22).

AZ	SRP Coronado Unit 1	**SCR + LNB/OFA**
AZ	SRP Coronado Unit 2	**SCR + LNB/OFA**
CO	Craig Station 1	**SCR + existing LNB/OFA**
CO	Craig Station 2	**SCR + existing LNB/OFA**
CO	Hayden Station 1	**SCR**
CO	Hayden Station 2	**SCR**
MT	Colstrip 1	**SOFA+SNCR**
MT	Colstrip 2	**SOFA+SNCR**
ND	Coal Creek Unit 1 & Unit 2	**LNB+SOFA+SNCR**
ND	Leland Olds 1	**SNCR + Basic SOFA**
ND	Leland Olds 2	**SNCR + ASOFA**
ND	Milton R. Young 1	**SNCR + ASOFA**
ND	Milton R. Young 2	**SNCR + ASOFA**
ND	Stanton 1 with Lignite Coal	**LNB+OFA+SNCR**
ND	Stanton 1 with Powder River Basin Coal	**LNB+OFA+SNCR**
NM	Four Corners 1	**Shutdown**
NM	Four Corners 2	**Shutdown**
NM	Four Corners 3	**Shutdown**
NM	Four Corners 4	**SCR**
NM	Four Corners 5	**SCR**
NM	San Juan Generating Station Unit 1	**SNCR**
NM	San Juan Generating Station Unit 2	**Shutdown**
NM	San Juan Generating Station Unit 3	**Shutdown**
NM	San Juan Generating Station Unit 4	**SNCR**
NV	Reid Gardner 1	**ROFA + Rotamix or LNB+OFA+SNCR**
NV	Reid Gardner 2	**ROFA + Rotamix or LNB+OFA+SNCR**
NV	Reid Gardner 3	**ROFA + Rotamix or LNB+OFA+SNCR**
NV	Tracy 3	**LNB + SNCR**
WA	TransAlta Centralia Units 1 and 2	**Flexfuel + SNCR**
WY	Dave Johnson Unit 3	**SCR or shutdown**
WY	Laramie River Unit 1	**SCR**
WY	Laramie River Unit 2	**SCR**
WY	Laramie River Unit 3	**SCR**
WY	Laramie River Unit 4	**SCR**
WY	Naughton Unit 3	**SCR**

Although these decisions are driving reductions in NOx as a condensable to fine particulate matter that mars vistas in our national parks and wilderness areas, the NOx reductions resulting from these BART and RFP determinations will have significant benefits for ozone formation as well.

In addition, the Mercury and Air Toxics Standard, while not directly targeting reductions in NOx emissions has nevertheless led a number of older, highly polluting facilities to elect to retire rather than upgrade outdated emission control systems for mercury and other hazardous air pollutants, with attendant benefits for NOx emissions.

Although not yet final, there are significant synergies between a low ozone standard and the Clean Power Plan (CPP), which projects large reductions in NOx emissions. EPA expects the CPP to have the following estimated human health co-benefits:

> In addition to CO2, implementing these proposed guidelines is expected to reduce emissions of SO2 and NO_X, which are precursors to formation of ambient PM2.5, as well as directly emitted fine particles. Therefore, reducing these emissions would also reduce human exposure to ambient PM2.5 and the incidence of PM2.5-related health effects. In addition, in the presence of sunlight, NOX and VOCs can undergo a chemical reaction in the atmosphere to form ozone. Depending on localized concentrations of volatile organic compounds (VOCs), reducing NOX emissions would also reduce human exposure to ozone and the incidence of ozone-related health effects.[692]

According to EPA estimates, the CPP is projected to reduce emissions of pollutants that contribute to ground-level ozone by over 25 percent in 2030.[693] This includes emission reductions of 407,000 to 428,000 tons of nitrogen dioxide[694] and 128,000 tons of ozone-season NO_X in 2030.[695] Thus, costs of implementing the CPP should be appropriately excluded from cost estimates of a 60 ppb ozone standard.

In the mobile source context, further reductions will be achieved as the nation's vehicle fleet becomes more efficient, cleaner, and electrified. California has been at the cutting edge of cleaning up emissions from vehicles. At least 14 states have adopted California's Clean Car Standards, which in addition to reducing greenhouse gas emissions from passenger vehicles, achieve significant reductions in NOx and VOCs as well.[696] In those states, the standards become effective for vehicles with model years between 2005 and 2011,[697] meaning that ongoing fleet turnover will continue to drive improvements in vehicle fleet emission of ozone precursors.

EPA's Tier 3 Vehicle Emission and Fuel Standards Program (Tier 3) (see Exhibit 23 and 24) is anticipated to have huge benefits for reducing NOx and VOC emissions. EPA predicts

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[692] U.S. EPA, Regulatory Impact Analysis for the Proposed Carbon Pollution Guidelines for Existing Power Plants and Emission Standards for Modified and Reconstructed Power Plants, 4-14 (June 2014).
[693] U.S. EPA, EPA Fact Sheet: Clean Power Plan, *available at* http://www2.epa.gov/sites/production/files/2014-06/documents/20140602fs-important-numbers-clean-power-plan.pdf
[694] *Id*.
[695] U.S. EPA, Memorandum: Emission Reductions, Costs, Benefits, and Economic Impacts Associated With Building Blocks 1 and 2
[696] In order of adoption: New Jersey, Connecticut, Washington, Vermont, New York, Maine, Rhode Island, Massachusetts, Oregon, Arizona, Pennsylvania, Maryland, Florida, and New Mexico. *See* Maryland Department of the Environment, States Adopting California's Clean Cars Standards,, *available at* http://mde.maryland.gov/programs/Air/MobileSources/CleanCars/Pages/states.aspx.
[697] *Id.*

that upcoming Tier 3 standards will reduce onroad NO_x emissions—the largest single category of NOx emissions—by nearly 10% in 2018, 25% by 2030, and 80% by 2050. [698] For VOCs, EPA predicts a 3% reduction by 2018, 16% by 2030, and 28% by 2050.[699] EPA projects that by 2018, Tier 3 will reduce ozone design values substantially against a 2018 reference case in certain key urban areas.[700] For example, the design values in Washington D.C. and surrounding counties in 2018 are anticipated to be decreased by over 1 ppb[701] and the design values for many counties in the Atlanta area approaches or exceeds 1.5 ppb.[702] Benefits are anticipated to be even greater by 2030. For example, the modeled design values in the Washington D.C. metro in 2030 are decreased by more than 2.5 ppb in 2030 compared to the reference case,[703] and decrease by nearly 3.0 ppb in 2030 compared to the reference case in the metro Atlanta area.[704]

Catalytic converter technology is now capable of eliminating over 95% of NOx emissions from on-road passenger vehicles. As the California Air Resource Board (CARB) explained eight years ago:

> Current catalytic converter designs are more than 95% efficient in removing the hydrocarbons (HC), carbon monoxide (CO), and oxides of nitrogen (NOx) from engine exhaust before they reach the atmosphere. Improvements in catalytic converter washcoats, precious metal loading, and substrate designs over the years, in combination with better vehicle fuel control systems, are the primary factors that have made compliance with California's very low emission standards possible.[705]

New York adopted the CARB standards for aftermarket catalytic converters in 2013[706] and Maine adopted the CARB standards, although has delayed implementation until 2018.[707] As catalytic converters achieving these high levels of emission reductions are more widely employed on new vehicles, and as the fleet turns over, there will be further reductions in NOx emissions from the mobile source sector.

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[698] U.S. EPA (2014). Office of Transportation and Air Quality, Regulatory Impact Analysis: Control of Air Pollution from Motor Vehicles: Tier 3 Motor Vehicle Emission and Fuel Standards Final Rule, 7-48.
[699] *Id*., 7-49.
[700] U.S. EPA (2014). Air Quality Modeling Technical Support Document: Tier 3 Motor Vehicle Emission and Standards (EPA-454/R-14-002), Appendix B: 8-Hour Ozone Design Values for Air Quality Modeling Scenarios.
[701] *Id*.
[702] *See id.* (Cobb County, GA: 1.28 ppb; De Kalb County, GA: 1.43 ppb; Douglas County, GA: 1.27 ppb; Fayette County, GA: 1.39 ppb; Gwinnett County, GA: 1.51 ppb; Henry County, GA: 1.56 ppb)
[703] *See id*. (Washington, D.C.: 2.73 ppb; Montgomery County, MD: 2.40 ppb; Prince Georges County, MD: 1.87 ppb; Arlington County, VA: 2.70 ppb; Fairfax County, VA: 2.68 ppb; Alexandria City, VA: 2.50 ppb).
[704] *See id*. (Cobb County, GA: 2.35 ppb; De Kalb County, GA: 2.74 ppb; Douglas County, GA: 2.08 ppb; Fayette County, GA: 2.45 ppb; Gwinnett County: 2.82 ppb; Henry County, GA: 2.54 ppb)
[705] State of California Air Resources Board, Staff Report: Initial Statement of Reasons for Rulemaking, Public Hearing to Consider Amendments to Regulations Regarding New Aftermarket Catalytic Converters and Used Catalytic Converters Offered for Sale and Use in California (Sept. 7, 2007). (See Exhibit 25).
[706] *See* New York State Dept. of Env. Conservation, Fact Sheet Prohibition of Used Catalytic Converters/New Aftermarket Catalytic Converter Standards, *available at* http://www.dec.ny.gov/chemical/87411.html.
[707] *See* Maine Dept. of Env. Protection, Rulemaking Fact Sheet (5 MRSA §8057), *available at* http://www.maine.gov/tools/whatsnew/attach.php?id=635480&an=1. (See Exhibit 26).

Additional reductions in ozone precursors can be achieved through increased electrification of the vehicle fleet. President Obama in 2008 set a goal of 1 million electric vehicles (EVs) on the road by 2015.[708] According to data from the Electric Drive Transportation Association, sales of plug-in vehicles have increased significantly in recent years.[709]

Table 31

Year	Plug-in Vehicle Sales[a]
2014	118,773
2013	96,702
2012	52,835
2011	17,735
2010	345

[a] = Includes plug-in hybrid electric vehicles and extended-range electric vehicles

The benefits of electrification of the vehicle fleet depend, of course, on reducing the NOx-intensity of the power generation sector. However, there are a number of reasons why the NOx-intensity of power generation will continue to decline. New generation comprises almost exclusively NOx-free renewable and relatively low-NOx new gas generation, displacing older, dirtier units. Moreover, the share of new generation from NOx-free renewables has been dramatically increasing in recent years. According to a recently-released National Renewable Energy Laboratory (NREL) analysis, "In 2013, renewable electricity accounted for more than 61% of all new electricity capacity installations in the United States."[710] As cleaner, largely NOx free generation replaces our oldest and dirtiest power plants, the ozone benefits of vehicle electrification will continue to increase.

Finally, it is important to bear in mind that societal changes and changes in patterns of urban development will also reduce the cost of compliance with a new ozone standard. As urban areas become denser, public transit improves, ride-sharing options increase, and cities become more walkable and bikable, reliance on NOx and VOC-emitting motor vehicles will decrease.

2. Further Reductions in VOC Emissions from the Oil and Gas Sector Are Forthcoming

EPA has also taken recent steps to limit VOC emissions from the oil and gas sector. In 2012, the agency finalized new source performance standards (NSPS) for specified equipment in the oil and gas industry that will reduce VOC emissions by approximately 190,000 tons annually starting in 2015.[711] Taking into account voluntary emission reductions anticipated through EPA's Natural Gas STAR program, this figure increases to 290,000 tons annually.[712] The

[708] *See* U.S. White House, FACT SHEET: President Obama's Plan to Make the U.S. the First Country to Put 1 Million Advanced Technology Vehicles on the Road, a*vailable at* www.whitehouse.gov/sites/default/files/other/fact-sheet-one-million-advanced-technology-vehicles.pdf.
[709] Electric Drive Transportation Association, Electric Drive Sales Dashboard, *available at* http://electricdrive.org/index.php?ht=d/sp/i/20952/pid/20952. (See Exhibit 27).
[710] National Renewable Energy Laboratory, 2013 Renewable Energy Data Book at 4 (released Jan. 2015).
[711] 77 Fed. Reg. 49,490, 49,533 (Aug. 16, 2012).
[712] *Id.* at 49,534.

measures required under the oil and gas NSPS include (but are not limited to) reduced emission completions (RECs) at new hydraulically fractured natural gas wells, steep cuts in emissions at new storage tanks, and the installation of lower-emitting designs for new compressors and pneumatic controllers in various segments of the industry.[713] Notably, the REC component of the rule—the largest driver of its emission reductions—did not take effect until January 1, 2015.[714] Accordingly, the full scope of the rule's benefits is only now materializing.

Furthermore, the White House and EPA recently announced plans for a strategy to reduce the oil and gas industry's emissions of methane, a potent greenhouse gas that is often released into the atmosphere concurrently with VOCs.[715] As part of its methane control strategy, the administration expects to issue another NSPS for oil and gas sources targeting methane emissions from new equipment not covered under the 2012 rule, which may include "completions of hydraulically fractured oil wells, pneumatic pumps, and leaks from new and modified well sites and compressor stations."[716] EPA plans to publish a proposed rule in the summer of 2015 and a final rule in 2016.[717]

The agency also expects to develop "control technique guidelines" under section 183b of the Clean Air Act for existing oil and gas systems in ozone non-attainment areas and states in the Ozone Transport region.[718] These guidelines will help affected states achieve the ozone NAAQS by "provid[ing] an analysis of the available, cost-effective technologies for controlling VOC emissions from covered oil and gas sources"[719] and by establishing a "presumptive norm" for RACT determinations in their non-attainment SIPs. *Connecticut Fund for Envt., Inc. v. E.P.A.*, 672 F.2d 998, 1003 (2d Cir. 1982). The administration's other forthcoming actions to reduce methane pollution from oil and gas sources, such as EPA rules requiring leak detection and repair and BLM rules targeting waste and flaring of natural gas on federal land, can also be expected to reduce co-occurring emissions of VOCs.

3. There are Significant, Economic Reductions to Be Achieved from Coal Fired Electricity Generating Unites in the Electric Power Sector

Despite the ongoing progress in the electric power sector, massive emission reductions are still readily achievable from the U.S. coal fleet. According to EPA's 2011 National Emission Inventory, over 85% of electric sector NOx emissions are from coal plants.[720] Although

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[713] *See id.* at 49,496-99.
[714] *Id.* at 49,492.
[715] *See* Press Release, The White House, FACT SHEET: Administration Takes Steps Forward on Climate Action Plan by Announcing Actions to Cut Methane Emissions (Jan. 14, 2015), *available at* http://www.whitehouse.gov/the-press-office/2015/01/14/fact-sheet-administration-takes-steps-forward-climate-action-plan-anno-1; U.S. EPA, Press Release: EPA's Strategy for Reducing Methane and Ozone-Forming Pollution from the Oil and Natural Gas Industry (Jan. 14, 2015), *available at* http://yosemite.epa.gov/opa/admpress.nsf/0/BA7961BF631C87BF85257DCD00526FF7
[716] Press Release, EPA (Jan. 14. 2015), *supra* .
[717] *Id.*
[718] *Id.*
[719] *Id.*
[720] U.S. EPA, National Emissions Inventory, *available at* http://www.epa.gov/ttnchie1/net/2011inventory.html. Approximately 1.8 million out of 2.1 million tons of electric sector NOx were from coal-fired electric generating units.

adoption of SCR control technology has become increasingly widespread on U.S. coal plants, with 327 coal units having installed SCR or having announced plans to install SCR (representing nearly 42% of units 100 MW or larger and over 177 GW or 56% of coal capacity),[721] significant further progress is achievable. There are still 451 coal units accounting for 140 GW of capacity that lack SCR or plans to install SCR.

More insidiously, many of the plants that have installed SCR controls, often at the expense of ratepayers, are not operating these controls at previously demonstrated control levels. In 2013, more than 20 coal units had annual NOx emission rates that were over five times greater than their best consistently demonstrated historical emission rate ("best demonstrated emission rate").[722] In other words, these facilities emitted five times as much NOx in 2013 as they could have if they had simply operated their SCR at proven control efficiencies. More than 50 coal units had annual NOx emission rates that were over three times greater than their best demonstrated emission rate.[723] At least 88 coal units had NOx emission rates that were more than double their best demonstrated emission rate.[724] At least 135 coal units had NOx emission rates more than 50% higher than their best demonstrated rate.[725] And over 170 units had NOx emission rates more than 25% higher than their best previously demonstrated rate.[726] In other words, coal plants around the country have been routinely turning down their existing NOx emission controls—emission controls often directly paid for (with interest) by ratepayers—causing the public to pay not only with their pocketbooks but also with their lungs.

The consequences for NOx emissions and air quality are staggering. As documented in the accompanying analysis by Dr. Ranajit Sahu, if coal units that have already installed SCR controls simply operated those controls to replicate best demonstrated control efficiency, they could reduce annual ozone season emissions by more than 136,000 tons and overall annual emissions by more than 296,000 tons each year. Given that total coal EGU NOx emissions in 2011 were approximately 1.8 million tons, it follows that emissions from the largest stationary source sector of NOx emissions could be reduced by 1/6th simply by operating existing emission controls.

The emission reductions achievable from cleaning up the other half of the coal fleet are also enormous. We analyzed emissions from coal plants in CSAPR states and New England[727]

[721] Compiled using data from Energy Information Agency Form 860 and EPA's Clean Air Markets Database (ampd.epa.gov/ampd/).
[722] Best demonstrated historical rate was calculated by looking at daily average NOx rates reported by the facility to EPA and available from EPA's Air Markets Program Database (ampd.epa.gov/ampd/) and identifying, post-installation of SCR, the lowest NOx emission rate that was achieved on a daily basis for 30 consecutive operating days. Because this analysis required the facility to have met the rate as a daily average every day during a 30 consecutive operating day period, it is a very conservative rate, especially when compared to an annual average. Additional data on average 2013 NOx rate was also retrieved from EPA's Air Markets Program Database.
[723] Data from EPA's Air Markets Program Database.
[724] *Id.*
[725] *Id.*
[726] *Id.*
[727] Alabama, Arkansas, Connecticut, Delaware, Florida, Georgia, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Nebraska, New Hampshire, New Jersey, New York, North Carolina, Ohio, Oklahoma, Pennsylvania, South Carolina, Tennessee, Texas, Virginia, West Virginia, and Wisconsin. (Note that Maine, Rhode Island and Vermont have no coal plants.)

using publicly available emission data. During the ozone season of 2011, coal plants in these states emitted over 570,000 tons. These ozone season emissions could be reduced by 61% (350,000 tons) through operation of existing controls, as described above, and installation of SCR controls on the remaining coal units.[728] This figure excludes emissions from coal plants that have ceased operating between 2011 and present.[729] Annual emission reductions are even greater: NOx emissions from these states could be reduced by nearly 780,000 tons each year (from 1.28 million tons down to 500,000 tons) simply by upgrading to SCR and operating existing SCR controls. There is no reason to expect that emissions reductions from plants in the remaining states would be any less significant, indicating that coal EGU emissions as a whole can still be reduced by more than 3/5th with existing and widely-used control technologies.

Table 32: **Achievable Annual and Ozone Season NOx Emission Reductions from Coal Plants in CSAPR and New England States (2011)**

	Actual NOx (tons)	**Achievable NOx Tons: Installation and Operation of SCR (tons)**	**NOx Reduction (Actual - Achievable) (tons)**	**NOx Reduction Percentage (%)**
Ozone season	572,724	223,179	349,545	61.0%
Annual	1,280,129	502,852	777,277	60.7%

4. Additional NOx Reductions from Coal Plants Will Have a Significant Ameliorative Effect on Ozone Levels

Coal EGUs are by far the largest stationary source category of NOx emissions in the United States, with 2011 emissions of approximately 1.8 million tons. However, coal plants' contributions to air quality are likely to be even more pronounced due to the coincidence of emissions from these plants with conditions favoring peak ozone formation. The conditions most conducive to the formation of ozone are also conditions that lead to increased utilization of the dirtiest coal EGUs: hot summer days when air conditioners are running and energy demand is at its highest. Coal units lacking SCR controls are often operating on these high energy demand days and, due to their lack of controls, can have an outsize impact on total NOx emissions and poor air quality.

In order to evaluate the air quality benefits from additional, SCR-level control of the U.S. coal fleet, we retained Sonoma Technology, Inc. (Sonoma) to conduct air dispersion modeling using the Comprehensive Air Quality Model with extensions (CAMx). Sonoma used EPA's 2011 modeling platform, including acquiring 2011 emissions data from EPA, 2011 outputs from the Weather Research and Forecasting (WRF) meteorological model, and 2011 GEOS-Chem

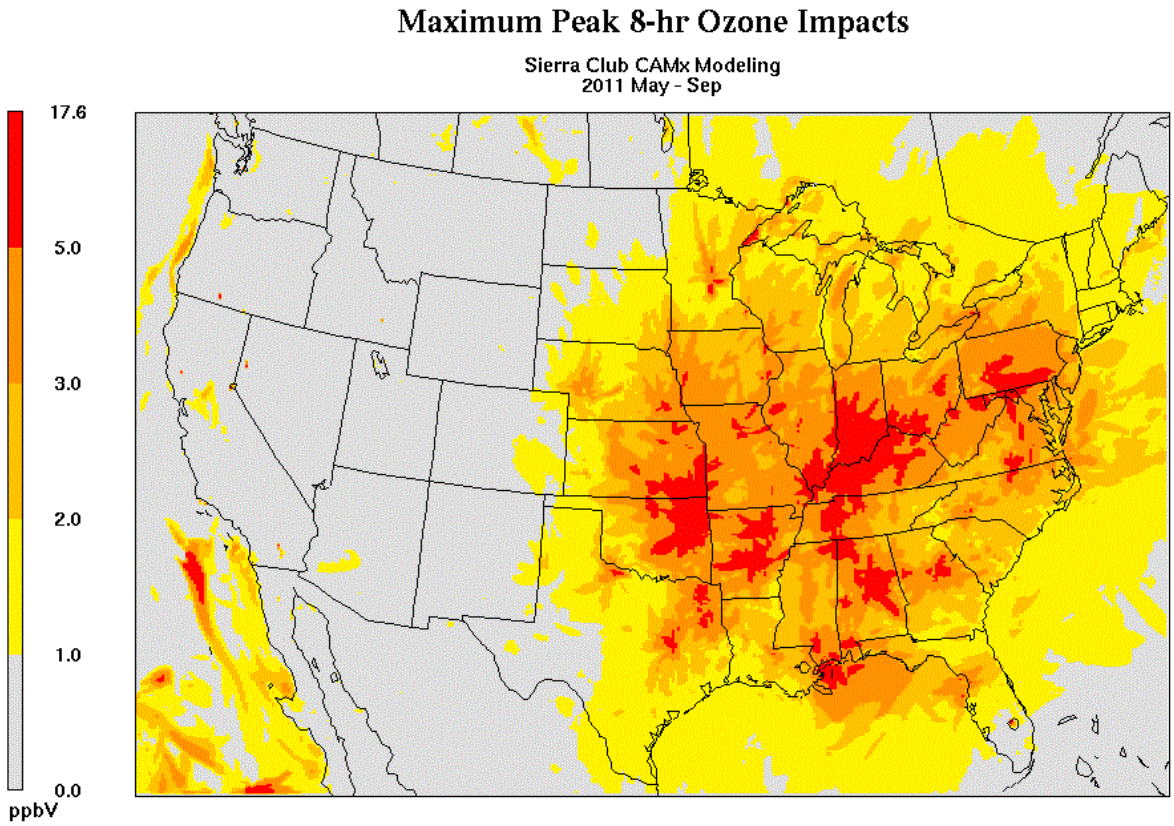
[728] Installation of SCR was simulated by scaling a plant's NOx emission rate down by the ratio of its actual average ozone season rate and 0.07 lb/MMBtu. The latter represents a rate that is consistent with recent entries in the RACT/BACT/LAER Clearinghouse and is demonstrated to be readily achievable in practice by coal units equipped with SCR. Indeed, according to data from EPA, during the 2013 ozone season 105 coal-fired units had NOx emissions at or below 0.07 lb/MMBtu. EPA, Air Markets Program Database, ampd.epa.gov/ampd/.

[729] Emissions from these sources comprise another more than 56,000 tons of 2011 NOx.

results to prepare initial conditions and boundary condition inputs. Emissions processing was conducted using the Sparse Matrix Kernel Emissions Modeling System (SMOKE).

In order to simulate the impacts of SCR-level control of the U.S. coal fleet, Sonoma completed two 2011 ozone season model runs using CAMx. First, Sonoma completed a base case run in which EPA's 2011 emission inventory was left unchanged. This base case run provided baseline hourly ozone concentrations at all 12 km x 12 km grid cells in the nationwide modeling domain for all hours of the 2011 ozone season. Second, Sonoma completed an SCR-level control run in which it scaled down the emissions of coal plants in the CSAPR region to levels reflecting installation and operation of SCR. Using the same methodology discussed above in Section VII.A.3, to simulate operation of SCR for units that had already installed this control, each unit's 2011 ozone season average NOx emission rate was compared to the unit's best historical 30-consecutive day 24-hour emission rate (calculated using the same methodology described above). 2011 ozone season hourly emissions were then scaled down by the ratio of the best historical rate to the actual 2011 ozone season rate. This resulted in a small degree of scaling for most SCR units, but a larger degree of scaling for units that chronically failed to use or optimize use of their existing SCR. For units without SCR, the 2011 ozone season average NOx emission rate for each unit was scaled down by the ratio of 0.07 lb/MMBtu and the unit's actual average rate to reflect the fact that any unit that installs an SCR today should be able to achieve an emission rate at least as good as 0.07 lb/MMBtu.

Figure 15 depicts peak 8-hour air quality improvements between the base case and SCR-level control runs. As the figure illustrates, most areas throughout the Eastern United States experience peak 8-hour air quality improvements of more than 2.0 ppb; a large percentage of areas in the East experience peak air quality improvements of more than 3.0 ppb; and significant areas in many states, including Pennsylvania, West Virginia, Virginia, Ohio, Indiana, Kentucky, Illinois, Georgia, Alabama, Tennessee, Arkansas, Oklahoma, Texas, and Kansas experience peak 8-hour air quality improvements of between 5.0 and 17.6 ppb.



Sonoma also looked at air quality improvements in current ozone nonattainment areas and other major metropolitan areas. Figures 16, 17, 18 and 19 provide the results of that analysis. Numerous major metropolitan areas, including numerous nonattainment areas, experience large reductions in modeled 4th highest 8-hour daily maximum ozone concentrations as a result of simulated SCR-level control of the coal fleet. As shown in Figure 16, 4th highest 8-hour daily maximum ozone concentrations in major urban areas and nonattainment areas are reduced by as much as nearly 5 ppb in Cincinnati, and more than 30 major urban areas and nonattainment areas experiencing reductions in 4th highest 8-hour daily maximum ozone concentration of more than 1 ppb. As shown in Figures 17, 18, and 19, the number of days during ozone season 2011 when modeled ozone levels exceeded key thresholds—60 ppb, 65 ppb, and 70 ppb—declined dramatically in many urban areas based on SCR-level control of the coal fleet. For example, eleven cities, including three nonattainment areas, experienced a reduction of 10 or more days during the ozone season when maximum 8-hour ozone concentrations exceeded 60 ppb. An additional 14 cities, including 11 nonattainment areas, experienced 6 or more days during the ozone season when maximum 8-hour ozone concentrations exceeded 60 ppb.

Reductions in exceedances of 65 and 70 ppb were also widespread in the SCR-level control run. Four metropolitan areas, including two nonattainment areas experienced a reduction of 10 more days during the ozone season when maximum 8-hour ozone concentrations exceeded 65 ppb. An additional 15 metro areas, including seven nonattainment areas, experienced 6 or more days during the ozone season when maximum 8-hour ozone concentrations exceeded 65

ppb. And 19 metro areas, including five nonattainment areas, experienced 4 or more days during the ozone season when maximum 8-hour ozone concentrations exceeded 70 ppb.

Figure 17

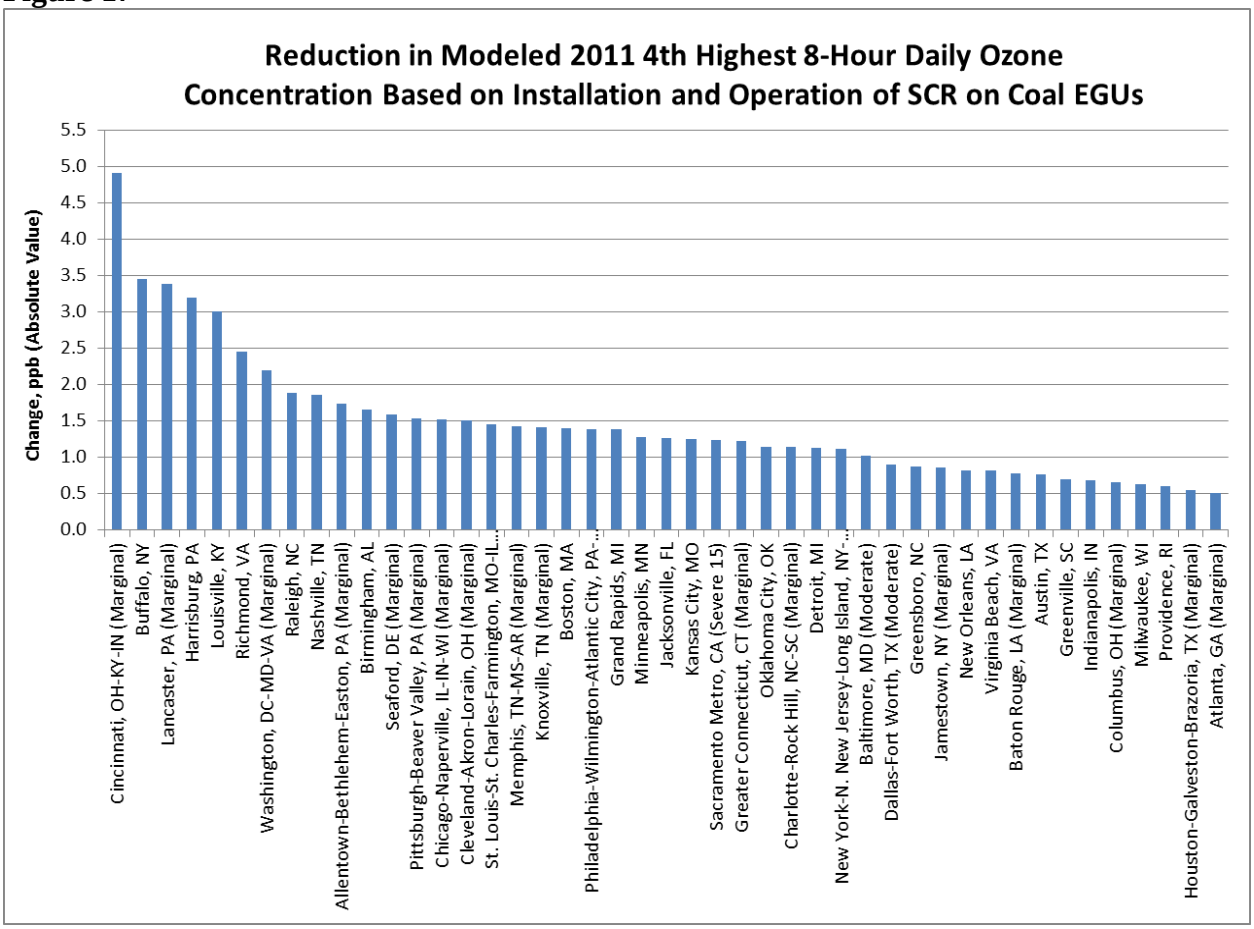


Figure 18

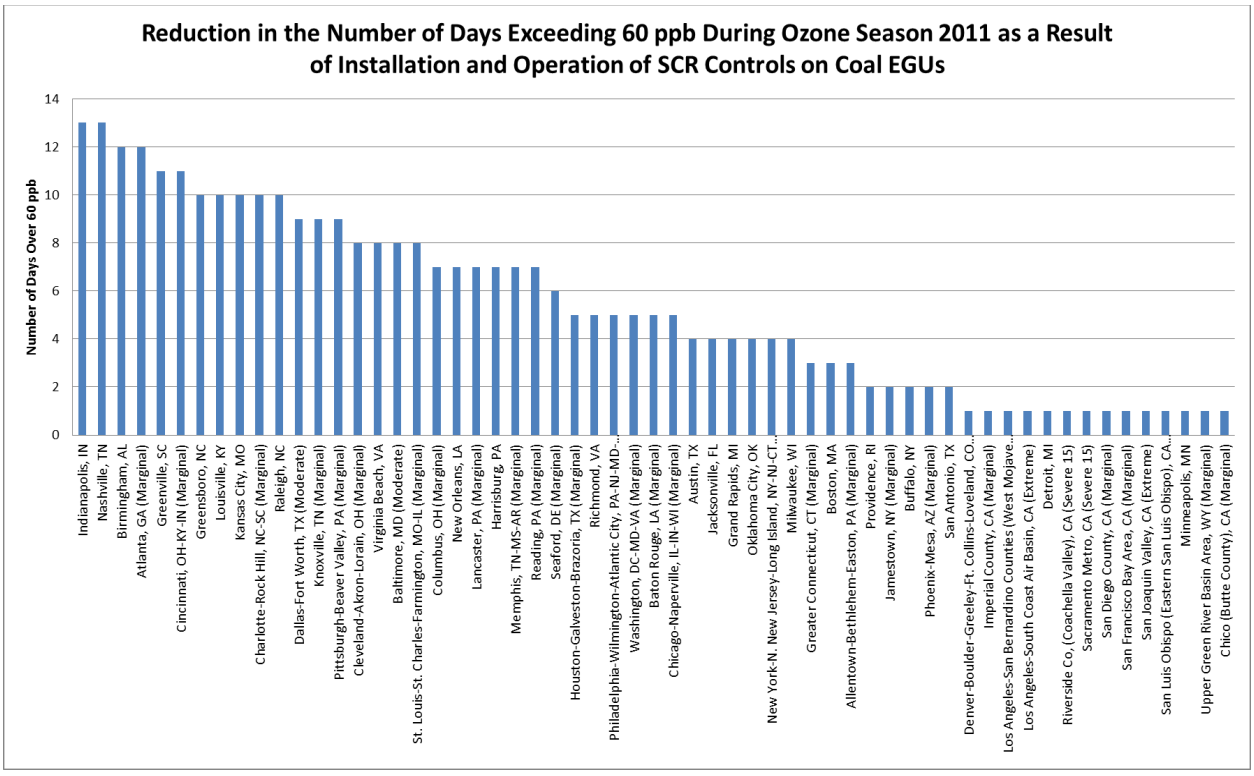
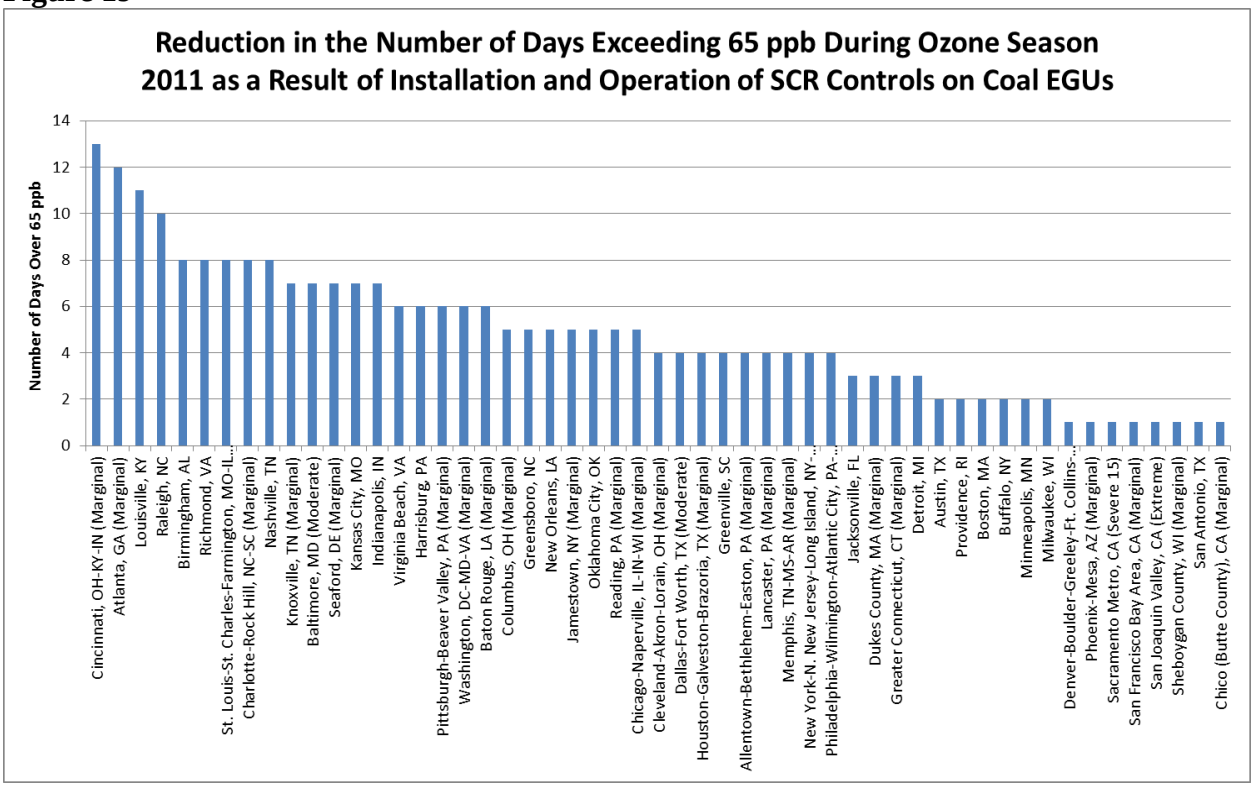

Reduction in the Number of Days Exceeding 60 ppb During Ozone Season 2011 as a Result of Installation and Operation of SCR Controls on Coal EGUs
Number of Days Over 60 ppb
0
2
4
6
8
10
12
14
Indianapolis, IN
Nashville, TN
Birmingham, AL
Atlanta, GA (Marginal)
Greenville, SC
Cincinnati, OH-KY-IN (Marginal)
Greensboro, NC
Louisville, KY
Kansas City, MO
Charlotte-Rock Hill, NC-SC (Marginal)
Raleigh, NC
Dallas-Fort Worth, TX (Moderate)
Knoxville, TN (Marginal)
Pittsburgh-Beaver Valley, PA (Marginal)
Cleveland-Akron-Lorain, OH (Marginal)
Virginia Beach, VA
Baltimore, MD (Moderate)
St. Louis-St. Charles-Farmington, MO-IL (Marginal)
Columbus, OH (Marginal)
New Orleans, LA
Lancaster, PA (Marginal)
Harrisburg, PA
Memphis, TN-MS-AR (Marginal)
Reading, PA (Marginal)
Seaford, DE (Marginal)
Houston-Galveston-Brazoria, TX (Marginal)
Richmond, VA
Philadelphia-Wilmington-Atlantic City, PA-NJ-MD-...
Washington, DC-MD-VA (Marginal)
Baton Rouge, LA (Marginal)
Chicago-Naperville, IL-IN-WI (Marginal)
Austin, TX
Jacksonville, FL
Grand Rapids, MI
Oklahoma City, OK
New York-N. New Jersey-Long Island, NY-NJ-CT...
Milwaukee, WI
Greater Connecticut, CT (Marginal)
Boston, MA
Allentown-Bethlehem-Easton, PA (Marginal)
Providence, RI
Jamestown, NY (Marginal)
Buffalo, NY
Phoenix-Mesa, AZ (Marginal)
San Antonio, TX
Denver-Boulder-Greeley-Ft. Collins-Loveland, CO...
Imperial County, CA (Marginal)
Los Angeles-San Bernardino Counties (West Mojave...
Los Angeles-South Coast Air Basin, CA (Extreme)
Detroit, MI
Riverside Co. (Coachella Valley), CA (Severe 15)
Sacramento Metro, CA (Severe 15)
San Diego County, CA (Marginal)
San Francisco Bay Area, CA (Marginal)
San Joaquin Valley, CA (Extreme)
San Luis Obispo (Eastern San Luis Obispo), CA...
Minneapolis, MN
Upper Green River Basin Area, WY (Marginal)
Chico (Butte County), CA (Marginal)

Figure 19


Reduction in the Number of Days Exceeding 65 ppb During Ozone Season 2011 as a Result of Installation and Operation of SCR Controls on Coal EGUs
Number of Days Over 65 ppb
0
2
4
6
8
10
12
14
Cincinnati, OH-KY-IN (Marginal)
Atlanta, GA (Marginal)
Louisville, KY
Raleigh, NC
Birmingham, AL
Richmond, VA
St. Louis-St. Charles-Farmington, MO-IL...
Charlotte-Rock Hill, NC-SC (Marginal)
Nashville, TN
Knoxville, TN (Marginal)
Baltimore, MD (Moderate)
Seaford, DE (Marginal)
Kansas City, MO
Indianapolis, IN
Virginia Beach, VA
Harrisburg, PA
Pittsburgh-Beaver Valley, PA (Marginal)
Washington, DC-MD-VA (Marginal)
Baton Rouge, LA (Marginal)
Columbus, OH (Marginal)
Greensboro, NC
New Orleans, LA
Jamestown, NY (Marginal)
Oklahoma City, OK
Reading, PA (Marginal)
Chicago-Naperville, IL-IN-WI (Marginal)
Cleveland-Akron-Lorain, OH (Marginal)
Dallas-Fort Worth, TX (Moderate)
Houston-Galveston-Brazoria, TX (Marginal)
Greenville, SC
Allentown-Bethlehem-Easton, PA (Marginal)
Lancaster, PA (Marginal)
Memphis, TN-MS-AR (Marginal)
New York-N. New Jersey-Long Island, NY-...
Philadelphia-Wilmington-Atlantic City, PA-...
Jacksonville, FL
Dukes County, MA (Marginal)
Greater Connecticut, CT (Marginal)
Detroit, MI
Austin, TX
Providence, RI
Boston, MA
Buffalo, NY
Minneapolis, MN
Milwaukee, WI
Denver-Boulder-Greeley-Ft. Collins-...
Phoenix-Mesa, AZ (Marginal)
Sacramento Metro, CA (Severe 15)
San Francisco Bay Area, CA (Marginal)
San Joaquin Valley, CA (Extreme)
Sheboygan County, WI (Marginal)
San Antonio, TX
Chico (Butte County), CA (Marginal)

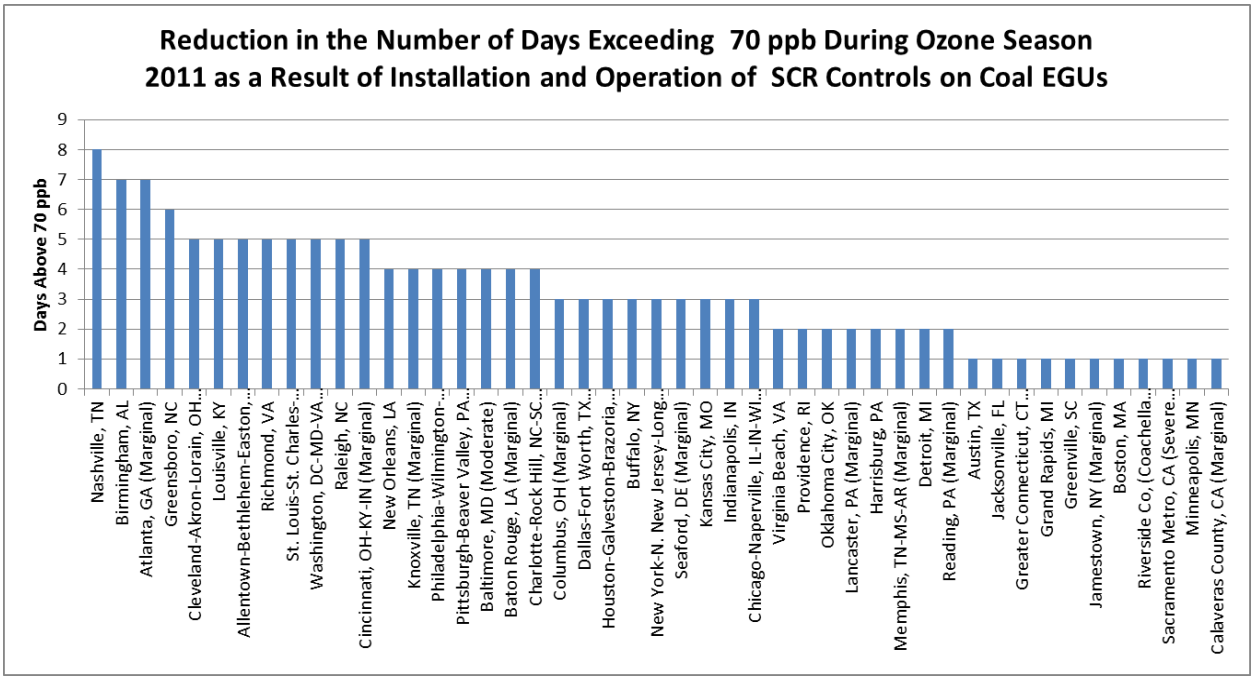
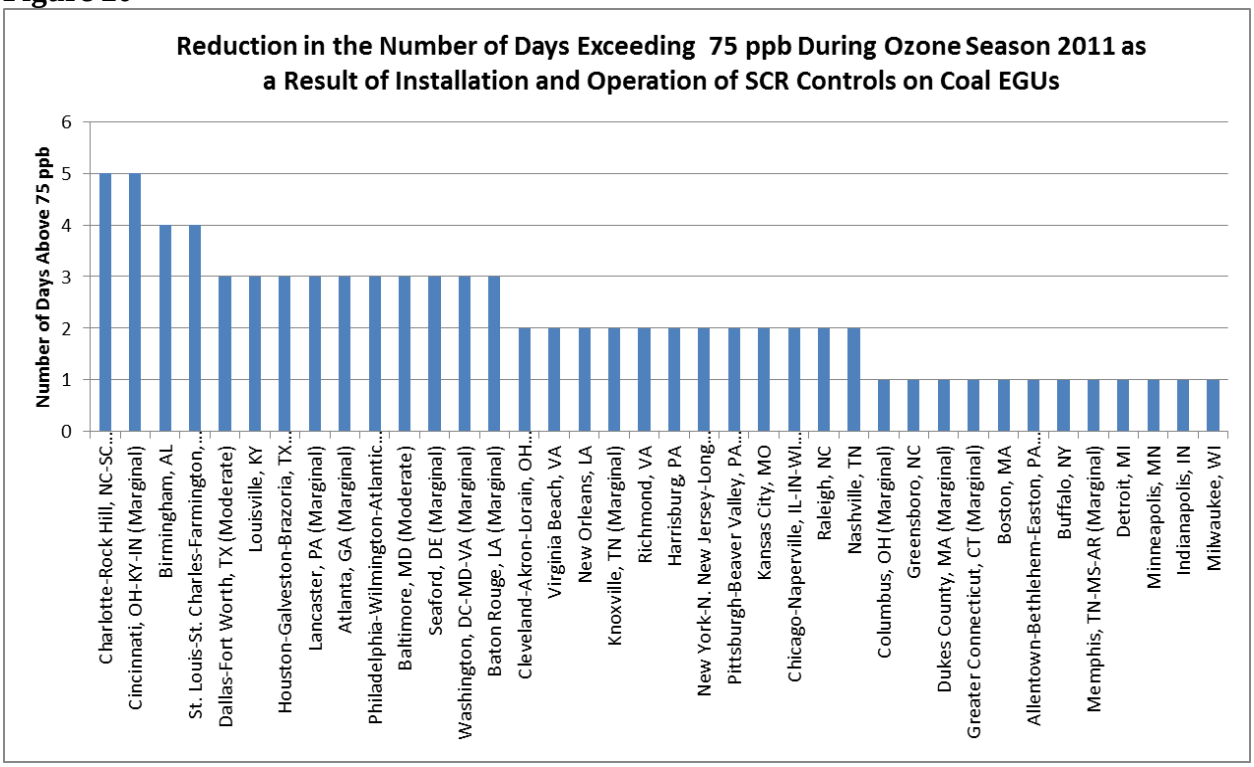


Figure 20



5. Tremendous NOx Reductions--from Coal Fired EGUs as well as from Other Sources--Can Be Achieved Cost-Effectively And Have Been Judged to Be Economically Reasonable Time and Again

Not only are significant additional NOx and VOC reductions available, but they can be achieved at a reasonable cost, far below recent industry estimates, which have repeatedly proven to be significantly exaggerated.

NOx is a precursor for ozone that originates from a wide variety of stationary and mobile sources. The vast majority of NOx comes from anthropogenic sources. According to data from EPA's National Emissions Inventory (NEI), the top U.S. sources of NOx are dominated by on-road light- and heavy-duty vehicles and coal-fired power generation. [730] For these and other categories, there are many cost-effective methods readily available for reducing NOx pollution.

Again, some of the most cost-effective reductions are achievable from the nation's coal-fired EGUs. Coal-fired power plants are the third largest source category of NOx emissions, emitting 11.5% of NOx nationwide. They are also the largest type of stationary source by a significant margin, emitting over twice as much NOx per year as the second largest stationary source category, natural gas-fired industrial boilers and internal combustion engines (ICEs). As discussed in Section VII.A above, simply ensuring that power plants that have already upgraded their emission controls actually operate them consistent with best demonstrated historical rates has the potential to reduce total coal EGU emissions by approximately 1/6th. Dr. Sahu in his analysis not only quantified the emission reductions achievable both annually and during the ozone season from coal plants with SCRs already installed fully operating these controls, but he also quantified the cost per ton of doing so. As detailed in Dr. Sahu's accompanying report, Dr. Sahu relied on industry estimates of variable operating and maintenance costs for additional reagent use and catalyst replacement and disposal developed by Sargent and Lundy for EPA's Integrated Planning Model.[731] Dr. Sahu concluded that by simply running already installed controls at proven levels it is possible to achieve 136,534 tons of additional ozone season NOx emission reductions each year at a total cost of $63.5 million and a cost/ton of only $465. And it is possible to achieve 296,160 tons of additional annual NOx reductions each year at a total cost of $129.5 million and a cost/ton of just $437.

Beyond these extremely cost-effective NOx reductions from simply operating installed controls, there are significant additional highly cost-effective reductions from the more poorly controlled remainder of the coal fleet. Control measures for coal-fired EGUs are low-hanging fruit offering cost-effective NOx reductions. EPA has determined that "there likely would be very large emissions reductions available from EGUs before costs reach the point for which non-EGU sources have available reductions."[732] By installing and operating SCR in combination with low-NOx burners (LNB) and over-fired air (OFA), existing plants can remove NOx at rates between $1,210 and $4,550 (2015$) and up to 89% control efficiency.[733] SCR plus sorbent

[730] U.S. EPA NEI version 2, *available at* http://www.epa.gov/ttnchie1/net/2011inventory.html (accessed Mar. 9, 2015).
[731] Sargent and Lundy, IPM Model – Updates to Cost and Performance for APC Technologies SCR Cost Development Methodology Final March 2013, *available at* (see also Exhibit 29, Exhibit 30, Exhibit 31) http://www.epa.gov/airmarkets/documents/ipm/attachment5_3.pdf. (See Exhibit 28).
[732] 76 Fed. Reg. 48208, 48249 (Aug. 2, 2011)
[733] *See e.g.*, Technical Support Document to Comments of Conservation Organizations, Proposed Federal Implementation Plan To Address Regional Haze Requirements for the Navajo Generating Station Units 1, 2 and 3 (Dec. 30, 2013); 77 Fed. Reg. 50,936 (Aug. 23, 2012); 78 Fed. Reg. 8,273–8,294 (Feb. 5, 2013); Enviroplan Consulting, Final Findings Report Golden Valley Electric Association (GVEA) Best Available Retrofit Technology

injection has control costs of $1,690 to $2,060 (2015$).[734] For SCR alone, existing coal EGUs control costs range from $2,835 to $3,430 (2015$).[735]

Significant cost-effective emission reductions are also available from mobile sources. According to EPA, most controls for mobile sources can be installed and operated at between $1,160 and $5,500 (2015$)[736] per ton of NOx or NOx+non-methane hydrocarbons, with average costs ranging from $3,500 to $7,000.[737] Moreover, many measures produce fuel savings, offsetting control costs and even resulting in net savings in some cases.

On-road diesel heavy-duty vehicles are responsible for over 19% of U.S. NOx emissions.[738] Retrofitting diesel vehicles with cleaner equipment – such as installation of SCR - can be performed at costs as low as $4,966 (2015$) per ton for class 8 vehicles.[739] Retrofits of class 6 and 7 heavy duty vehicles and commuter programs are also available, though they have higher costs per ton in part because they do not necessarily provide large emission reductions.[740] Technology to reduce long idling on heavy-duty diesel vehicles can reduce NOx and actually save money by reducing fuel costs.[741] For example, two measures to eliminate idling on heavy-duty diesel vehicles, such as advanced truck electrification--which provide trucks parked overnight with electrical power, heating, and cooling to avoid running the engines--and installing truck auxiliary power units--a small engine and generator that provides power to the truck with the main engine shut off--cost between $1,600 to $3,950 (2015$) per ton of NOx and VOC.[742] These measures are in various stages of build out in a number of states. Off-road diesel vehicles are also major NOx emitters, and are accompanied by a range of control costs, starting at around $2,120 for engine upgrades.[743]

For light-duty vehicles, which emit 16.5% of U.S. NOx, median costs for operating continuous inspection and maintenance programs are around $2,550 per ton of NOx and VOCs, although these programs, too, can actually pay for themselves and in fact save money in the

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(BART) Evaluation (April 27, 2009); 77 Fed. Reg. 76,871 (Dec. 31, 2012); 77 Fed. Reg. 76,871 (Dec. 31, 2012); 77 Fed. Reg. 72,511 (Dec. 5, 2012); 77 Fed. Reg. 72,511 (Dec. 5, 2012); U.S. EPA, RACT/BACT/LAER Clearinghouse, *available at* http://cfpub.epa.gov/RBLC; 77 Fed. Reg. 40,151 (July 6, 2012).

[734] *See e.g.*, 76 Fed. Reg. 52,388 (Aug. 22, 2011).

[735] *See e.g.*, 75 Fed. Reg. 64,221 (Oct. 19, 2010); 77 Fed. Reg. 51,915 (Aug. 28, 2012); 76 Fed. Reg. 80,754 (Dec. 27, 2011); 75 Fed. Reg. 64,221 (Oct. 19, 2010)..

[736] All costs converted to 2015 $ using the CPI inflation calculator. http://data.bls.gov/cgi-bin/cpicalc.pl.

[737] U.S. EPA, Chapter 3: Emissions Controls Analysis –Design and Analytical Result, Final Regulatory Impact Analysis (RIA) for the NO2 National Ambient Air Quality Standards (NAAQS) (Jan. 2010), *available at* http://www.epa.gov/ttnecas1/regdata/RIAs/FinalNO2RIACh31-20-10.pdf.

[738] *See* National Emissions Inventory, 2011.

[739] U.S. EPA, Menu of Control Measures. Last updated April 2012, *available at* http://www.epa.gov/air/criteria.html; U.S. EPA, Update of the Control Measures Database for Onroad Sources with MOVES, OAQPS (Contract No. EP-D-07-097, WA No. 4-09). Prepared by TranSystems (2011).

[740] U.S. EPA, Chapter 3, *supra* note 725.

[741] U.S. EPA 2012, Menu of Control Measures *supra* note 727; U.S. EPA, 2011: "Update of the Control Measures Database for Onroad Sources with MOVES". Prepared for the U.S. Environmental Protection Agency, OAQPS; http://www.epa.gov/ttnecas1/regdata/RIAs/FinalNO2RIACh31-20-10.pdf

[742] Per ton NOx and VOCs. Cost-effectiveness uses weighting ratio of 4:1 for NOx:VOC; U.S. EPA, The Cost-Effectiveness of Heavy-Duty Diesel Retrofits and Other Mobile Source Emission Reduction Projects and Programs, Transportation and Regional Programs Division Office of Transportation and Air Quality(May 2007).

[743] U.S. EPA. Lists of Potential Control Measures for PM 2.5 and Precursors (2007).

long-run.[744] Commuter programs offer incentives to reduce light-duty vehicle miles traveled and trips, with programs such as regional rideshares, vanpool programs, park-and-ride lots, regional transportation demand management, and employer trip reduction program. EPA reports that the costs for operating these programs start as low as $1,630 (2015$) per ton of NOx and VOCs[745], with median costs across all types of commuter programs running at $26,000 per ton.[746] On average, EPA has estimated that implementing more stringent requirements for aftermarket replacement catalytic converters could reduce NOx at a cost of $3,700 per ton. [747]

Simply retiring dirty vehicles can be a cost effective measure for reducing NOx pollution. Vehicle retirement programs for heavy-duty diesel vehicles can be cost-effective, as has been demonstrated by California's Carl Moyer Grant Program, which performed retirements at an overall cost of $3,040 (2015$) per ton.[748] California's Bay Area Air Quality Management District's retirement program estimated cost-effectiveness at $7,000 to $8,115 (2015$) per ton and was funded by a $4 increase in car registration fees.[749,750]

Many low cost reductions have already been made available through EPA's national vehicle standard. EPA programs tightening emissions standards for both on-road and non-road mobile sources and reducing the sulfur content of gasoline and on- and non-road diesel fuel, along with fleet turnover, have already caused mobile NOx emissions to begin to decline significantly.[751] EPA's Tier 3 Motor Vehicle Emission and Fuel Standards, which come into effect in 2017, are projected to reduce NOx at a cost of $4,484 a ton by 2030.[752]

Cost-effective NOx reduction measures are within reach. While EPA ultimately must set the primary standard at a level that protects public health, without consideration of cost, affordable application of existing technology can offer NOx reductions from major sources at a reasonable cost.

B. The Benefits of a 60 ppb Ozone Standard Outweigh the Costs

[744] *Per ton NOx and VOC. Cost-effectiveness uses weighting ratio of 4:1 for NOx:VOC.
U.S. EPA. The Cost-Effectiveness of Heavy-Duty Diesel Retrofits and Other Mobile Source Emission Reduction Projects and Programs. Transportation and Regional Programs Division Office of Transportation and Air Quality. May 2007.; http://www.epa.gov/ttnecas1/regdata/RIAs/FinalNO2RIACh31-20-10.pdf
[745] 1:4 weighted sum of VOC and NOx
[746] http://www.epa.gov/ttnecas1/regdata/RIAs/452_R_08_003.pdf
[747] http://www.epa.gov/ttnecas1/regdata/RIAs/FinalNO2RIACh31-20-10.pdf
[748] Wagner and Rutherford. Survey of Best Practices in Emission Control of In-Use Heavy-Duty Diesel Vehicles. ICCT. CCAC. August 2013.
[749] http://www.nj.gov/dep/baqp/rapt/wps/DI009_fin.pdf
[750] As a higher-end example for light-duty vehicles, costs for a small-scale vehicle retirement programs in Illinois ran around $76,350 a ton of NOx (2015$). See http://escholarship.org/uc/item/1h30m2r7. The National Association of Manufactures (NAM) has recently estimated the cost per ton of reducing NOx via "Cash for Clunkers" at a whopping $500,000 per ton. See - http://www.nam.org/Issues/Environment/Ozone-Regulations/NERA-NAM-Ozone-Full-Report-20140726.pdf. The 2009 "Cash for Clunkers" program was designed as an economic stimulus program, never as a cost-effective NOx reductions strategy. Even the high cost of $76,350 cited here is still seven times lower than NAM's baseless estimate used in analyses regarding the compliance costs of the new ozone NAAQS. See discussion in Section VII.B, below.
[751] U.S.EPA. *Non-EGU Emissions Reductions Cost and Potential.* Technical Support Document (TSD) for the Transport Rule. Docket ID No. EPA-HQ-OAR-2009-0491. Office of Air and Radiation. May 2010.
[752] U.S. EPA. Draft Regulatory Impact Analysis: Tier 3 Motor Vehicle Emission and Fuel Standards. March 2013.

Though, as explained in Section VII.A above, the cost-benefit balance is not relevant and EPA must not consider it, if it were relevant, it would strongly favor a 60 ppb ozone standard. By EPA's own calculation, for the states outside of California, the net benefits—even accounting for compliance costs—of setting the ozone standard level to 60 ppb is approximately $13 billion. And as EPA has recognized, compliance cost estimates often exceed actual costs in practice.[753]

Many of the emission reductions required to achieve a lower ozone standard have already been baked into final and proposed rules. As discussed above in Section VII.A, these rules will significantly ratchet down emissions of ozone precursors, dramatically reducing the cost of compliance with a new, lower ozone standard. As the Office of Management and Budget's (OMB) Office of Information and Regulatory Affairs recently confirmed, it is not appropriate to double count the costs associated with those rules in estimating the cost of compliance with a new ozone NAAQS.[754] Consistent with this principle, OMB excluded the benefits and costs associated with the 2006 PM2.5 NAAQS from its 10-year aggregate and year-by-year estimates to prevent double-counting due to implementation of CSAPR.[755] The NOx reduction co-benefits of the soon to be finalized Clean Power Plant will also reduce the cost of compliance with a new ozone standard.

As EPA well knows, industry has a consistent track record of grossly overstating the costs of proposed new rules.[756] Industry always argues that new regulations will be tremendously expensive and harm the economy, but industry's assertions have proven false. For example, in 1990, when strengthening amendments to the Clean Air Act were proposed, utilities forecasted that reducing sulfur dioxide would cost $1,000 to $15,000 per ton and that electric rates would increase by 10%.[757] In reality, the reductions cost only $100-$200 per ton, 5-75 times less than what industry predicted.[758] At the same time electric rates fell in most states.

The latest effort to impermissibly inject cost considerations into the NAAQS-setting process for ozone is no different. The NERA Economic Consulting analysis completed on

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[753] U.S. EPA, Regulatory Impact Analysis, 8-11 (2014) (finding "[s]tudies indicate that it is not uncommon for pre-regulatory cost estimates to be higher than later estimates, in part because of difficulty in predicting technological changes").

[754] White House Office of Management and Budget, Office of Information and Regulatory Affairs. 2014 Draft Report on the Benefits and Costs of Federal Regulations and Unfunded Mandates on State, Local, and Tribal Entities. (2014), at 14.

[755] *See id.*

[756] EPA Admin'r Lisa P. Jackson, Remarks on the 40th Anniversary of the Clean Air Act, As Prepared, yosemite.epa.gov/opa/admpress.nsf/12a744ff56dbff8585257590004750b6/7769a6b1f0a5bc9a8525779e005ade13!OpenDocument (Sept. 14, 2010) ("Today's forecasts of economic doom are nearly identical—almost word for word—to the doomsday predictions of the last 40 years. This 'broken-record' continues despite the fact that history has proven the doomsayers wrong again and again.").

[757] U.S. House of Representatives, Committee on Energy and Commerce, Democratic Staff, *The Clean Air Act's Track Record: Cleaner Air and Economic Growth* (2013), available at http://democrats.energycommerce.house.gov/sites/default/files/documents/Fact-Sheet-The-Clean-Air-Act-2013-6-25.pdf.

[758] *Id; see also* U.S. House of Representatives Committee on Energy and Commerce, *Industry Claims about the Costs of the Clean Air Act* (2009), available at: http://democrats.energycommerce.house.gov/Press_111/20090616/dc_industryjobs.pdf.

behalf of the National Association of Manufacturers (NAM)[759] both wholly disregards the myriad public health benefits associated with a more health-protective NAAQS and attendant avoided medical expenses associated with asthma-induced emergency department visits, asthma hospitalizations, and missed work and school days. Moreover, it also massively distorts the cost of compliance by basing all NOx reductions of unknown origin on an assumed cost/ton of $500,000.

The NAM study authors have admitted that the huge difference between their asserted $500,000 per ton compliance cost for unknown controls and EPA's far lower estimates, is due "almost entirely" to a cost methodology that is based upon the "Cash for Clunkers" economic stimulus program.[760] Cash for Clunkers was not a program intended to drive NOx reductions: it was a program that was intended to drive the purchase of new vehicles. Any NOx reductions were incidental benefits. Independent, academic experts have characterized this NAM cost methodology as "insane" and "unmoored from economic reality."[761] As discussed above, NAM's cost estimates are well over 1,000 times higher than the cost at which 1/6th of U.S. coal plant emissions could be reduced. It is approximately 100 or more times higher than the cost/ton at which SCR can and has been added to many coal plants. And it is orders of magnitude higher than the cost-effective emission reductions from the mobile source sector discussed above.

In 2011, EPA reviewed the costs and benefits of the steps taken under the Clean Air Act authority from 1990 to 2010 and projected to 2020. In a peer-reviewed study, they found that by 2020, the benefits of Clean Air Act will have outweighed the costs by 30:1.[762] Congress receives an annual report from the White House on the costs and benefits of federal programs. Under both Republican and Democratic Administrations, benefits have always been much higher than costs. Looking at the decade from 2002 to 2012, the OMB calculated the benefits of 21 EPA air pollution rules at up to $529.1 billion, compared to $35.3 billion in costs (in 2001 dollars).[763]

According to EPA, since 1970 industry and other regulated parties have been able to cut harmful air pollution by approximately seventy percent (70%) while the economy has grown by tripled. Since 1980, ozone levels have been reduced by thirty-three percent (33%) across the

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[759] NERA Economic Consulting, *Assessing Economic Impacts of a Stricter National Ambient Air Quality Standard for Ozone*, (July 2014) (Note: this report predated EPA's proposal by five months, and made numerous assumptions where information was unavailable) *available at* http://www.nam.org/Issues/Environment/Ozone-Regulations/NERA-NAM-Ozone-Full-Report-20140726.pdf

[760] See "Industry Report Identifies Higher Costs For Ozone Proposal Than EPA Estimates," Bloomberg BNA Daily Environment Report (Feb. 27, 2015) (Attachment __).

[761] See "Experts: Pro-Smog Pollution Report Is 'Unmoored From Reality,' http://mediamatters.org/research/2014/08/20/experts-pro-smog-pollution-report-is-unmoored-f/200490 (Aug. 20, 2014) (quoting Professor Michael Livermore, senior advisor at New York University's (NYU) Institute for Policy Integrity) (emphasis added) (Attachment __). For a thorough critique of the multitude of flaws and biases in the NAM cost study, see generally id. and "National Association of Manufacturers: Thin Air," Laurie Johnson, http://switchboard.nrdc.org/blogs/ljohnson/national_association_of_manufa.html (Sept. 16, 2014) (Attachment __).

[763] Executive Office of the President, Office of Management and Budget. 2013 Report to Congress on the Benefits and Costs of Federal Regulations and Unfunded Mandates on State, Local. And Tribal Entities. March, 2014. Available at http://www.whitehouse.gov/sites/default/files/omb/inforeg/2013_cb/2013_cost_benefit_report-updated.pdf.

country. For over 45 years, there has been little to no evidence of economic damage due to the abatement of air pollution.

C. The Phenomenon of NOx Scavenging Does Not Counsel a Higher Ozone Standard

Industry and other petitioners have sought to use the "ozone scavenging" phenomenon – whereby fresh NO_X emissions at ground level can "scavenge" ozone molecules and reduce ozone concentrations measured by surface monitors – as an argument against setting a lower ozone standard. According to these petitioners, lowering the ozone standard would require increasing relative NO_X concentrations, which, in turn, would lower the rate of ozone scavenging and therefore increase ozone levels in urban cores, where fresh NO emissions are typically found. This argument rests on multiple assumptions: (1) that ozone destruction is simply a function of increased NO_X levels in VOC-limited areas, (2) that ozone scavenging is an effective pollution control mechanism, and (3) that the increased strategic NO_X reductions accompanying a lower ozone NAAQS will result in harmful ozone increases in all urban cores. As shown below, these assumptions are false.

This is fundamentally an argument based on control considerations; as such, it is not legally relevant for the reasons discussed above in Section VII.A. Even if considered (which it should not be), the ozone scavenging argument against lowering ozone NAAQS should be rejected because it rests on an incomplete understanding of both the science of ozone formation and EPA modeling. More importantly, it should be rejected because, contrary to what its opponents claim, a lower ozone standard will result in significant health benefits for urban cores populations.

1. Ozone Formation is Complex and Non-Linear

The formation of ozone is a complex process involving nonlinear change in response to hundreds of precursors.[764] In particular, NO_X precursors can lead to both the formation and the destruction of ozone, depending on the local concentrations of NO_X, VOC, and radicals such as the hydroxyl (OH) and hydroperoxy (HO2) radicals, as well as local meteorological conditions.[765] NO_X emissions contribute to ozone formation in so-called "NO_X-limited" areas, which feature high VOC/NO_X ratios, where the production of ozone varies directly with NO_X concentrations (e.g., downwind of important NO_X sources, typically in rural or suburban areas). NO_X contributes to ozone destruction in so-called "VOC-limited" areas,[766] which feature low VOC/NO_X ratios, where ozone ozone is destroyed through NOx scavenging[767] (e.g. in downtown metropolitan areas, close to busy streets and roads, and in power plant fumes). However, while

[764] California Air Resources Board, *The Physics and Chemistry of Ozone*, Section 1.1. Available at: http://www.fraqmd.org/ozonechemistry.htm.
[765] DRAFT RULE, at 75242; HREA, at 2-5.
[766] Also called "radical limited" or "NOx-saturated." California Air Resources Board, *The Ozone Weekend Effect in California*, Staff Report, at 20 (June 30, 2003). Available at http://www.arb.ca.gov/research/weekendeffect/arb-final/web-executive-summary.pdf (hereinafter, "CARB Staff Report").
[767] NO_X scavenging refers to the process whereby O_3 is depleted by reaction with NO to form NO_2. Also referred to as "ozone titration."

NO_X can "destroy" ozone through titration in the immediate vicinity of emission sources, the resulting NO_2 will later lead to ozone formation downwind.

Though the VOC/NO_X-limited paradigm is often used to explain ozone formation, the relationship between ozone and its precursors "is actually much more complicated than implied by the simple description of VOC-limited v. NO_X-limited conditions."[768] Generalizations about the impact of control strategies on the amount of ozone in an area are therefore "difficult to make because of the fact that the relationship depends strongly on local conditions such as topography, and spatial distributions and types of emission sources, which vary from one city to another, and on meteorological conditions that vary from day-to-day" (noting that the interplay between NOx and VOC is affected by their ratio, the reactivity of the VOC mix, the role of biogenic hydrocarbons, the extent of photochemical aging; and the severity of an air pollution event).[769] Indeed, the VOC-limited and NO_X-limited labels do not neatly correspond to the urban/rural divide: some cities are strongly or primarily VOC-limited (e.g. Los Angeles, San Francisco, Phoenix, New York), others have mixed VOC-NO_x-limitations (e.g. Houston, Philadelphia), and still others are NO_X-limited (e.g., Atlanta, Nashville).[770] Finally, the VOC- NO_X-limited explanation is incomplete, as there are other chemical processes partially contributing to or causing ozone destruction in VOC-limited areas.[771]

Accordingly, ozone response to NO_X emissions reductions is complex and may include ozone decreases at some times and locations and ozone increases in other times and locations.[772] Generally, in NO_X-limited conditions, ozone concentrations are most effectively reduced by lowering NO_X rather than VOC emissions, while the reverse is true in VOC-limited conditions. However, a VOC-only reduction strategy is not robust enough to achieve desired ozone reductions in most VOC-limited areas, and further, even in VOC-limited areas, "very large decreases in NOx emissions can cause the O3 formation regime to become NO_X-limited. Consequently, reductions in NO_X emissions (when large), can make further emissions reductions more effective at reducing O_3."[773] Additionally, not all areas fall neatly into either a NO_X-limited or a VOC-limited category, both because there is a transitional region, where ozone is less sensitive to marginal changes in either NO_X or VOCs[774] and because the NO_X-VOC ratio of a certain area can vary significantly from day to day.[775] Accordingly, "the key question is whether controlling NO_X is beneficial or counterproductive for an area as a whole, not just at a single

[768] U.S. Congress, Office of Technology Assessment, (July 1989). *Catching Our Breath: Next Steps for Reducing Urban Ozone*, at 99 (hereinafter, "OTA Report")
[769] OTA Report, at 97; *see also* Sillman, S., *Overview: Tropospheric Ozone, Smog and Ozone-NO_x-VOC Sensitivity*. Available at: http://www-personal.umich.edu/~sillman/ozone.htm#OZO1.2.1.
[770] *See* Duncan B., et al. (2009). The Sensitivity of U.S. Surface Ozone Formation to NOx and VOCs as Viewed From Space, at 3-4. Available at https://www.cmascenter.org/conference/2009/abstracts/duncan_sensitivity_us_2009.pdf (hereinafter, "2009 Duncan"); *see also* ISA, at 3-7.
[771] CARB Staff Report, at 17 (suggesting "NOx reductions, different timing of emissions including NOx, different amounts and impacts of pollutants that persist overnight aloft, different amounts of light-absorbing particulate matter in the air, and ozone quenching by nitric oxide emissions" as equally plausible explanations for the weekend effect).
[772] HREA, Section 2.2.1.
[773] HREA, at 2-5.
[774] HREA, at 2-5.
[775] OTA Report, at 102.

location."[776]

2. Ozone Scavenging Is Not A Pollution Control Mechanism – It Is A Pollution-Moving Mechanism

Arguing that the EPA should not set a lower ozone standard because doing so would reduce NO_X scavenging in urban cores assumes that NO_X scavenging is an effective and acceptable pollution control mechanism. It is neither. NO_X scavenging is not a solution to the problem of ozone formation, because while "NOx can initially destroy O_3 near the emission sources, these same NO_X emissions eventually do react to form more O_3 downwind."[777] In other words, NO_X scavenging does not actually destroy ozone, it merely delays its formation, and shifts it downwind. NO_X scavenging is also an unacceptable pollution control mechanism because it results in the creation of NO_2, which is another harmful criteria pollutant. Finally, NO_X scavenging does not address the problem of NO_X itself, which is a key precursor not only of ozone, but also of particulate matter (PM) and other compounds with health and environmental concerns.[778] Therefore, reducing NO_X emissions is beneficial not only because it is necessary to meet a lower O_3 standard, but also because it will result in health benefits beyond those directly associated with reducing ambient O_3 concentrations.[779]

3. Even If Scavenging Were Relevant, Lowering the Ozone Standard Will Produce Health Benefits In All Areas, Including Urban Cores

a. The Ozone Scavenging Implications of Reduced NO_X Emissions Are Extremely Limited

The ozone scavenging implications of NO_X emissions reductions accompanying lower standards is extremely limited, both temporally and geographically. As the EPA's photochemical model simulation in the current revision of ozone NAAQS shows, adjusting NO_X emissions reductions to meet existing and alternative standards resulted in an overall narrowing of the range of ozone concentrations: as peak ozone concentrations (typically occurring in rural or suburban areas, during warm months, on days with higher ozone concentration) tended to decrease, concentrations in the lower part of the ozone distribution (typically occurring in urban areas, including urban cores, during cool months, on days with lower ozone concentration) tended to increase.[780] Additionally, NO_X emissions reductions accompanying lower ozone standards generally decreased ozone concentrations in the high and mid-range portions of the ozone distribution in rural, urban and suburban areas, as well as most urban cores.[781]

Further, the ozone scavenging implications of NO_X emissions reductions in urban cores was extremely restricted: NOx reductions in urban cores generally decreased peak concentrations

[776] OTA Report, at 102.
[777] DRAFT RULE, at 75242.
[778] These include nitrogen dioxide (NO2), nitric acid (HNO3), nitrous acid (HONO), peroxyacetylnitrate (PAN), nitro-polycyclic aromatic hydrocarbons (nitro-PAHs), regional haze, and nitrate deposition with subsequent fertilization and eutrophication of soils and surface waters. CARB Staff Report, at 20.
[779] DRAFT RULE, at 75285; CARB Staff Report, at 8.
[780] HREA, Sections 4.3.3.2 (Figures 4-9 and 4-10) and 9-7 and at 8-48; DRAFT RULE, at 75242.
[781] HREA, at 9-7.

and increased lower concentrations (as they did in the larger study areas) but they only increased concentrations near the center of the ozone distribution in a small subset of cities. Ozone responses near the center of the ozone distribution in urban cores followed one of the three patterns shown in the following table:

Table 9-3. General Patterns in Seasonal (May-Sept) Mean of Daily Maximum 8-hr O_3 Concentrations after Adjusting to Meet Existing and Alternative Standards.*

After Adjusting Air Quality to Just Meet Existing Standard	After Further Adjusting to Just Meet Lower Alternative Standards	Urban Study Areas Showing Pattern
Decreased	Continued to decrease	Atlanta, Sacramento Washington, D.C
Increased	Decreased	Baltimore, Cleveland Dallas, Detroit, Los Angeles. New York Philadelphia, St. Louis
Increased	Continued to increase or remained constant	Boston, Chicago, Denver, Houston

* These patterns refer to O_3 responses in the urban core of each urban study area based on analysis of the interpolated monitor values used as inputs to the exposure and lung function risk analyses.

HREA, at 9-7 (Table 9-3). As Table 9-3 shows, *no* seasonal mean increases were found in the majority of the cities studied (Atlanta, Sacramento, Washington D.C., Baltimore, Cleveland, Dallas, Detroit, Los Angeles, New York, Philadelphia, and St. Louis) at any of the proposed alternative standards, while increases in mean seasonal ozone at alternative standards were only found in four cities (Boston, Chicago, Denver and Houston).[782] Additionally, the extent and impact of any such ozone increases was further mitigated by the fact that increases were slight and occurred mostly on days with lower ozone concentrations, such as "during cool, cloudy weather and at night when photochemical activity is limited or nonexistent."[783] Accordingly, while reductions in NO_X precursors led to increases in ozone in certain locations, those increases were restricted to the middle and lower ends of the ozone distribution and a small number of urban cores, and they did not get progressively larger as alternative standards were lowered.

b. Any Health Risk Increases Resulting From Reduced Ozone Scavenging Are Limited at 70 ppb and Offset Entirely at 60 ppb

Ozone increases resulting from reduced ozone scavenging in urban cores do not translate into significant health risk increases. To begin with, urban core populations would be expected to experience important *reductions* in both ozone exposures and ozone -induced lung function risks when meeting alternate standards.[784] Further, while urban core areas "in some cases show[] overall increases in epidemiology-based mortality and morbidity risk", these increases are not substantial: only 3 of the 12 urban study areas (Boston, Detroit, and Houston) exhibited an increase in the percentage change in ozone-attributable risk after meeting the 70 ppb standard

[782] HREA, at 9-7 (Table 9-3).
[783] DRAFT RULE, at 75242; *see also* HREA, at 7-55—7-56 (Figures 7-2 and 7-3) (showing that increases in O_3 (and resulting estimated increases in risk) occur largely on days with initial O_3 concentrations in the range of 10 to 40 ppb).
[784] HREA, at 9-42 (emphasis added).

(compared to risk that meets the current standard) and only did so in the 2009 simulation year.[785] Importantly, those increased risks are offset *entirely* when adjusted to meet the lowest alternative standard (60 ppb).[786] *Id.* Furthermore, even those increases are likely to be mitigated through a concurrent VOCs/ NO_X reduction strategy. *See infra*, Section VII.C.3.iii.

c. The Effects of Reduced Ozone Scavenging Can Be Mitigated With a Combined VOCs/NOx Reduction Strategy

In assessing the ozone air quality response to reducing both NOX and VOC emissions for a subset of seven urban study areas, the EPA found that a concurrent VOCs/NOx reduction strategy mitigates median ozone level increases.[787] Specifically, the addition of VOC reductions generally resulted in larger decreases in mid-range ozone concentrations (25th to 75th percentiles), and reduced the low concentration ozone increases in all seven of the urban study areas evaluated.[788] Given that the EPA's reduction strategies and broad nationwide emission cuts are merely crude approximations of the actual strategies individual states can pursue, it is more than likely that state implementation plans will be even more efficient at mitigating the effects of O_3 scavenging.

Indeed, there is a long success record for concurrent reductions of VOCs and NO_X. NO_X scavenging is not a new phenomenon – also referred to as "the weekend effect", it has been a part of pollution control strategies for decades. CARB, facing the highest ozone levels and the most VOC-limited urban area in the country, has been pursuing a strategy of concurrent VOCs and NO_X reductions for more than thirty-five years, and has been successful at reducing the high ozone levels all over southern California.[789] While acknowledging temporary ozone increases through reduced NOx scavenging, CARB maintains that "NO_X reductions are not counter-productive for attaining ambient air quality standards" and that "ozone air quality has improved most when NO_X air quality has also improved."[790] In other words, NO_X scavenging demands an optimized reduction strategy, not a higher ozone standard.

VIII. CONCLUSION

For all of the reasons explained above, EPA should heed its duty to follow the science and establish the ozone NAAQS at a level that is truly protective of public health with and adequate margin of safety: 60 parts per billion.

Respectfully submitted,

Janice Nolan, Assistant Vice President, National Policy

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[785] HREA, at 7-69 (Table 7-7 and 7-8).
[786] The risk is increased in simulating estimated risk from moving from recent conditions to just meeting the existing standard. *See* HREA, Appendix 7b, Tables 7B-1 and 7B-2 (showing increases for Houston and Los Angeles for the 2007 year, and increases for Boston, Cleveland, Houston, Los Angeles, New York, Philadelphia for the 2009 year).
[787] DRAFT RULE, at 75278.
[788] DRAFT RULE, at 75278 (citing HREA, Appendix 4D, Section 4.7).
[789] CARB Staff Report, at 6.
[790] CARB Staff Report, at 9.

American Lung Association

Deborah Shprentz
Consultant to the American Lung Association

Georgia Murray, Staff Scientist
Appalachian Mountain Club

Kevin Bundy, Senior Attorney
Anna "Mickey" Moritz, Legal Fellow
Center for Biological Diversity

Joseph O. Minott, Esq., Executive Director
Ryan Knapick, Staff Attorney
Clean Air Council

David Baron
Seth Johnson
Attorneys
Earthjustice

Elena Craft, PhD, Senior Health Scientist
Environmental Defense Fund

Stephanie Kodish, Director & Counsel, Clean Air Program
National Parks Conservation Association

John Walke, Clean Air Director
Emily Davis, Staff Attorney
Natural Resources Defense Council

Josh Stebbins, Managing Attorney
Josh Berman, Staff Attorney
Sierra Club

Appendix of Additional Comments of Members of the Clean Air Scientific Advisory Committee

Dr. John Balmes, a clinician, pointed to critical controlled human exposure studies and a reanalysis of the Adams 2006 study published subsequent to the 2008 review, and now considered in the current review.

> *"Since the scientific evidence was reviewed for the preparation of the 2006 Criteria Document for Ozone, the results of the Adams et al. (2006) study have been carefully reanalyzed (Brown et al., 2008) and actually show a statistically significant group effect. In addition, two other studies have shown statistically significant decrements in FEV1 after 6.6-hour exposures to 0.070 ppm (Schelgele et al., 2009) and 0.060 ppm (Kim et al., 2011), respectively."*

Dr. Balmes also referenced newly published studies that are now part of the record of this review.

> *"The reanalysis shows a statistically significant group decline in FEV1 at 60 ppb in the Adams study, at 70 ppb in the Schelgele study, and at 60 ppb in the Kim study, after 6.6 hour exposures. These findings only reinforce the need for a standard of 60 ppb."*

He further explains the clinical significance of a ten percent decrement in FEV_1, and the fact that in the absence of a threshold, even a standard of 60 ppb would not be fully protective of all individuals.

> *"From a clinical perspective, a 10% decrement in FEV_1 is often associated with respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (i.e., decreased baseline FEV_1) such that a ≥ 10% decrement could be associated with moderate to severe respiratory symptoms. From a public health perspective, the exposure and risk assessment conducted for the last review of the ozone NAAQS clearly document that a substantial proportion of the U.S. population is exposed to levels of ozone at the various alternative standards considered. This means that even if a NAAQS of 0.060 ppm were to be selected, some sensitive individuals could still be exposed to concentrations that could cause them to have a clinically relevant decrement in lung function".*

> *"The cumulative evidence to date on the ozone exposure-lung function response relationship strongly suggests that it is linear with no threshold, at least through 0.060 ppm. Therefore, it is reasonable to assume a similar exposure-response relationship for exacerbations of asthma. Considering the patterns of change in the estimates of exposures at alternative standards, as well as the uncertainties and limitations of the estimates, it is likely that susceptible individuals would still be adversely affected at a*

NAAQS of 0.060 ppm, although the number of such individuals would be substantially lower than at higher alternate standards."

Commenters urge EPA to heed this advice and to set a standard at the most protective level now under consideration, that is 60 ppb. Committee member Dr. Joe Brain cited the importance of inflammatory responses:

"Chronic inflammation and the presence of increased neutrophils and neutrophil elastase raise concerns. Chronic inflammation and resulting increased levels of reactive oxygen species (ROS) may result in cumulative irreversible damage. These changes raise concerns about increases in morbidity and mortality caused by chronic exposure to ozone."

Dr. Brain's comment is relevant to the Kim et al 2011 study that found inflammation after 6.6 hour exposures to 60 ppb.

Dr. James Gauderman commented on the statistical soundness of the Brown 2007 reanalysis.

"In the re-analysis of Adams (2006) study of 30 subjects by EPA (Brown, 2007), a small but statistically significant decline in FEV1 was observed. Specifically, a 2.85% mean O3-induced decline in FEV1 was observed following 6.6 hr square wave exposure to 0.060 ppm O3 compared to 6.6 hr filtered air (FA) exposure. The statistical analysis by EPA was based on a straightforward paired comparison, and they conservatively used a nonparametric sign test to obtain a p-value of 0.002 for the 0.06 ppm vs. FA comparison. Alternative, more powerful analytic methods using either a Wilcoxon signed-rank test or a paired t-test yielded even lower p-values in the EPA analysis. The EPA comparison remained significant after a Bonferroni correction for multiple comparisons. The original analysis of the data by Adams did not find a significant difference in FEV1 between the 0.06 and FA exposure conditions. However, that analysis was based on a Scheffe correction for multiple comparisons, which is known to have very low power for the type of pairwise comparisons conducted by Adams compared to other well-known methods for multiple-testing correction (Kirk, 1982)."

"Thus, from my understanding of the statistical analyses that have been conducted, I would argue that the analysis by EPA should be preferred to that of Adams for the specific comparison of the FEV1 effects of 0.06 ppm exposure relative to FA exposure. Of the 30 study subjects in Adams, 24 showed some evidence for an O3-induced decline in FEV1, and 2 of the 30 (7%) experienced a decline greater than 10%. Although the sample size is relatively small, the consistency of effects across O3 exposure levels, as well as the consistency with effects observed by an earlier independent study (McDonnell, 2002), indicates that the observed deficits in FEV1 at the 0.060 ppm from the Adams study are not spurious. In other words, it is likely that prolonged exposure to 0.06 ppm O3 causes a general shift in the distribution of FEV1 towards lower values. The following plot of the Adams data, derived from Figure 8-2 of Volume I of the "Air Quality Criteria for Ozone and Related Photochemical Oxidants, 2006" document, shows an approximate normal distribution in the O3-induced changes in FEV1 with exposure to

0.06 ppm."

"Although the mean decrement is less than 3% and would not be considered clinically important, the shift to the right in this distribution pushes a fraction of subjects into the region that becomes clinically interesting (>10%). All of the Adams study subjects were healthy volunteers. From a public health standpoint, these results suggest that a large number of individuals in the general population (that are otherwise healthy), are likely to experience FEV1 deficits greater than 10% with prolonged exposure to 0.06 ppm O3. Although most healthy individuals can probably sustain a short-term 10-15% decline in FEV1 with little or no noticeable effect, it is not clear how they might be affected in the longer term if they experience repeated lung function deficits due to 0.06 ppm or greater O3 exposures over multiple days or weeks. Based on several other controlled exposure studies, we might expect that O3-induced FEV1 deficits in subjects with an existing respiratory condition (e.g. asthma) would be shifted even further to the right compared to the above figure. A 10-15% (or greater) pollution-related deficit in FEV1 in an individual with an existing respiratory condition is large enough that it could cause a clinically observable response."

Dr. Rogene Henderson indicated the scientific basis for ozone standard in the range of 0.060 to 0.070 ppm, based on the controlled human exposure studies, noting the increased sensitivity of people with asthma.

"Human exposure studies provide the most direct evidence of the health effects on humans and the studies clearly show that adverse effects occur in some healthy adults after exposure for 6.6 hr to 0.060 ppm ozone. This finding has recently been confirmed in clinical studies in 59 healthy young adults exposed to 0.060 ppm ozone for 6.6 hours (Kim et al., dol:10.1164/rccm.201011-18130C, Lung function and inflammatory responses in healthy young adults exposed to 0.060 ppm ozone for 6.6 hours.) Asthmatic persons are known to be more sensitive to ozone than are healthy persons. Therefore, to provide some margin of safety, the standard must take into consideration these sensitive subpopulations."

Dr. Mort Lippman pointed the large number of people that are impacted by ozone concentrations evaluated in the chamber studies:

"Since the numbers of subjects exposed in the each of the controlled chamber studies at each concentration have been small, extrapolation to the much larger general population indicates that a very large number of individuals would have substantial responses, even though they would constitute only about 10% of the population."

Dr. Frank Speizer's comments indicated that susceptible populations might respond more severely to ozone exposures tested in the chamber studies.

"Although the two Adams studies represent the only reported work at levels of exposure below 0.080 ppm of ozone, what has been pointed out and what is highly significant is that first the studies were done in normals and second that some 7-20% of the subjects

experienced what I would consider very significant lung function decreases (> 10%) and or moderate respiratory symptoms. These findings essential preclude, because of the ethics of carrying out clinical studies in diseased individuals, from extending these studies to what are likely to be an even more sensitive groups. Thus, without having specific studies among asthmatics and children at these levels of exposure it is most prudent that, in spite of the uncertainty – more later on this issue –that EPA is justified to select and exposure level below the 0.080 ppm (and I would say closer to the 0.060 ppm level) to 'protect public health with an adequate margin of safety including the need to protect susceptible populations….'"

"Because these results represent a continuum of effects and it is unlikely that there is a threshold I would argue that the results are informative and suggest that EPA in carrying out its obligation must suggest a standard in the range indicated. I would argue that because there is no threshold that the data are consistent with the lower end of the range being more protective than the upper end."

"These small numbers of up to one-fifth of normals of the studied populations having changes in lung function or symptoms of this magnitude strongly suggests that the susceptible population would respond even greater and could reach clinically significant responses that might result in emergency room visits and or hospitalizations."

Dr. Helen Suh also commented on the adversity of effects in sensitive populations:

"The scientific evidence from controlled human exposure and epidemiological studies and from the exposure and risk assessments supports a primary ozone standard (with a margin of safety) between 0.060 to 0.070 ppm. The controlled human exposure studies by Adams (2002, 2006) show statistically significant changes in lung function from a 6.6 hour exposure to 0.060 ppm ozone. While these studies were limited in number, they were well designed and results were consistent with those from previous studies, thus lending credibility to their findings. Of particular interest is the fact that a small but important fraction of the study subjects experienced lung function decrements greater than 10% at exposures to 0.060 ppm ozone. These findings suggest that the impacts of ozone exposures at these levels may be significant for individuals with pre-existing respiratory conditions and must be considered to ensure adequate margin of safety for sensitive subpopulations."

"It is reasonable to consider findings of sub-clinical adverse impacts, such as increased inflammation and airway responsiveness, when considering adverse health impacts to healthy adults at exposures levels from 0.060 to 0.070 ppm. These findings are certainly pertinent to margin of safety considerations."

"These results provide important evidence that exposures to 0.060 ppm of ozone are harmful and are consistent with previous observations of no safe level for ozone exposures. Findings from Adams studies (2002, 2006) must be considered, at the least as being central to margin of safety determinations."

"For individuals with pre-existing respiratory disease, a 10% decrement in FEV1 is significant."

"Although the sample sizes are small, the variability in the response observed for healthy adults in the controlled human studies can inform judgments on the effects of ozone in susceptible populations. For example, the 7-20% of healthy adults who were found to have large ozone-mediated responses in controlled exposure studies may provide an indication of the fraction of individuals in the general population who may also be large responders. Ozone-mediated response may comprise an even greater percentage of the susceptible population."

Dr. James Ultman commented on the strengths and weaknesses of the controlled human exposure studies.

"Clinical Studies. Has several strengths including accurate and precise administration of exposure gas mixtures and patterns of exposure. The methods of measuring lung function and biological responses are also accurate, and precise, and are generally standardized between different laboratories. The medical and physiological states of the subjects are well-defined. Weaknesses include the use of ozone exposure levels that are usually 0.08 ppm or above. Only two studies (Adams 2002,2006) were conducted in the range 0.06-0.07 ppm ozone being considered for the new standard. Also, due to ethical concerns, the large majority of all clinical studies are performed on healthy or young subjects or subjects with mild respiratory disease. Moreover, only a handful clinical studies elucidate the role of copollutants in the exposure gas mixture, and responses are observable only when exercise is superimposed on exposure."

"The coherence of a substantial amount of data at 0.08 ppm and above also appears to be similar between the two labs (table 5-3). This coherence of a substantial amount data at 0.08 ppm and above, together with the plausibility of the exposure-response curve that passes through the more limited data at 0.06 and 0.04 ppm gives us confidence that clinically importance FEV1 responses can occur in moderately exercising subjects at 0.06 ppm ozone exposure."

"Though it only occurs in 7-20% of the subjects, the observation of decrements in FEV1 >10% at 0.06 ppm ozone exposure is an important indicator of a possible health effect in sensitive individuals. The probabilistic exposure-response curve in the staff paper of January 2007 (Fig. 5.4) further supports the expectation that, even in a 'healthy' population, there will be some individuals whose lung function is adversely affected by a single 8 hour exposure that includes intermittent moderate exercise."