

Comments on the U.S. Environmental Protection Agency's Proposed Reconsideration of the National Ambient Air Quality Standards for Ozone



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Earthjustice
Environmental Defense Fund
Sierra Club
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Executive Summary

The American Lung Association, Earthjustice, Environmental Defense Fund, Natural Resources Defense Council, and Sierra Club submit these comments in support of science driven primary National Ambient Air Quality Standards for ozone in order to protect public health with an adequate margin of safety as required by the Clean Air Act.

We welcome the U.S. Environmental Protection Agency's proposal reconsider the 2008 standards and to set the ozone NAAQS in the range of 60 to 70 ppb.

The proposed primary standards follow the science and are consistent with the unanimous recommendations of the Clean Air Scientific Advisory Committee (CASAC).

There is abundant scientific evidence in the record of the 2008 rulemaking pointing to the need to set the 8-hour standard at 60 ppb to protect public health with an adequate margin of safety.

These comments make a compelling case for EPA to adopt the most protective final standards that have been proposed.

Specifically, we urge EPA to set an 8-hour average primary standard for ozone of 60 ppb or below.

The comments that follow outline the legal and scientific case for our positions, including a discussion of the strong consensus in the international scientific community that the ozone standard must be substantially strengthened to protect public health. We cite strong evidence from controlled human exposure studies and epidemiology studies, supported by the toxicology studies, and from EPA's risk assessment for an 8-hour average ozone standard of 0.060 ppm or below.

EPA's Statutory Obligations under the Clean Air Act

National Ambient Air Quality Standards (NAAQS) are the cornerstone of the Clean Air Act's approach to regulating air pollution. The Act requires EPA to set primary NAAQS at levels requisite to protect public health with an adequate margin of safety. In determining whether proposed NAAQS achieve this mandate, EPA must err on the side of protecting public health, consider health impacts that may be impossible to quantify or are as yet uncovered by science, and ensure that sensitive populations like children and the elderly are protected. EPA must set secondary NAAQS at levels requisite to protect public welfare from any known or anticipated adverse effects associated with the presence of ozone in the ambient air.

In addition, in setting any NAAQS, EPA must give due deference to the advice of an independent panel of scientific advisors, the Clean Air Scientific Advisory Committee (CASAC). Finally, the Supreme Court has held that the EPA cannot consider the cost or feasibility of meeting the standard in setting the NAAQS.

Legislative Framework for NAAQS

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

National Ambient Air Quality Standards still drive many of the Clean Air Act's key 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 demonstrate what the state will do to reduce air pollution emissions in order to reduce the ambient concentrations of regulated pollutants to levels compatible with the NAAQS (including how the state will initially attain the standards, and how it will maintain and enforce the NAAQS).

The Clean Air Act provides a clear process for establishing the NAAQS. The first step in establishing a NAAQS involves identifying those pollutants "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. . . ." 40 U.S.C. § 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).

Thus any standards that EPA promulgates under these provisions must be adequate to (1) protect public health and (2) provide an adequate margin of safety, and (3) to prevent any known or anticipated non 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 (1) must err on the side of protecting public health, (2) must base decisions on the latest scientific knowledge giving due deference to the recommendations of the Clean Air Science Advisory Committee, and (3) 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.). For primary standards, “[b]ased 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 Assn. v. EPA*, 134 F.3d 388, 389 (D.C. Cir. 1998) (citations omitted); *see also Whitman v. American Trucking Assn.*, 531 U.S. 457, 464-71 (2001). *See* H.Rep. 294, 95th Cong., 1st Sess. 49-51 (1977) (explaining amendments designed *inter alia* “[t]o emphasize the preventive or precautionary nature of the act, i.e., to assure that regulatory action can effectively prevent harm before it occurs”).

Prior Revisions of 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 (January 18, 2001). The initial predecessor to the current ozone NAAQS was promulgated in 1971 at 0.08 ppm, averaged over one hour. 36 Fed. Reg. 8187 (April 30, 1971). *See American Petroleum Institute 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, one hour average. 44 Fed. Reg. 8220 (February 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, CAA § 109(d)(1), EPA failed to consider the new evidence, or to revise the NAAQS to reflect it. 58 Fed. Reg. 13013 (March 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. 13008, 13013-14, 13016 (March 9, 1993). 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. In 2008, EPA lowered the standard to 0.075 ppm, which EPA has now proposed to reconsider and revise.

NAAQS Must Protect Public Health with an Adequate Margin of Safety

In setting or revising a primary NAAQS, Section 109 of the Clean Air Act requires that the EPA achieve one thing at minimum: protect public health with an adequate margin of safety. The following excerpt from an opinion of the U.S. Court of Appeals for the District of Columbia sums up EPA's "margin of safety" 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 Assn. v. EPA, 134 F.3d 388, 389 (D.C. Cir. 1998)

Likewise, "[s]tandards must be based on a judgment of a safe air quality level and not on an estimate of how many persons will intersect given concentration levels. EPA interprets the Clean Air Act as providing citizens the opportunity to pursue their normal activities in a healthy environment." 44 Fed. Reg. 8210 (February 8, 1979). Thus, EPA cannot deny protection from air pollution's effects by claiming that the people experiencing those effects are insufficiently numerous or that levels that are likely to cause adverse health effects occur only in areas that are infrequently visited. To the contrary, the NAAQS mandate "carries the promise that ambient air in all parts of the country shall have no adverse effects upon any American's health." 116 Cong. Rec. 42381 (December 18, 1970)(remarks of Senator Muskie, floor manager of the conference agreement).¹

¹ See also 116 Cong. Rec. at 32901 (September 21, 1970) (remarks of Senator Muskie) ("This bill states that all Americans in all parts of the Nation should have clean air to breathe, air that will have no adverse effects on their health."); *id.* at 33114 (September 22, 1970) (remarks of Senator Nelson) ("This bill before us is a firm congressional statement that all Americans in all parts of the Nation should have clean air to breathe, air which does not attack their health."); *id.* at 33116 (remarks of Senator Cooper) ("The committee modified the President's proposal somewhat so that the national ambient air quality standard for any pollution agent represents the level of air quality necessary to protect the health of persons."); *id.* at 42392 (December 18, 1970) (remarks of Senator Randolph) ("we have to insure the protection of the health of the citizens of this Nation, and we have to protect against environmental insults -- for when the health of the Nation is endangered, so is our welfare, and so is our economic prosperity"); *id.* at 42523 (remarks of Congressman Vanik) ("Human health and comfort has been placed in the priority in which it belongs -- first place.").

In implementing its NAAQS mandates, EPA cannot deny protection against adverse health and welfare effects merely because those effects are confined to subgroups of the population or to persons especially sensitive to air pollution. It is inherent in NAAQS-setting that adverse effects are experienced by less than the entire population, and that we do not know in advance precisely which individuals will experience a given effect. In light of these circumstances, opponents of protective NAAQS often argue that NAAQS-setting involves evaluating "risk" and setting a level of risk that is "acceptable." But where—as here—peer-reviewed science shows that adverse effects stem from a given pollutant concentration, EPA must set NAAQS that protect against those effects with an adequate margin of safety. It cannot, under the guise of risk management, set NAAQS that allow such effects to persist. Indeed, given the scientific evidence documenting the occurrence of adverse effects year after year in numerous individuals at levels allowed by both the current NAAQS and EPA's proposal, risks are by definition "significant" enough to require protection under the Act's protective and precautionary approach. *See* H.R. Rep. No. 95-294 at 43-51; *Ethyl Corp. v. EPA*, 541 F.2d 1 (D.C. Cir. 1976). That is all the more true where the effects involved include highly serious ones like death and hospitalization. *See Ethyl*, 541 F.2d at 18 ("the public health may properly be found endangered ... by a lesser risk of a greater harm").

EPA Must Err on the Side of Protecting Public Health

Quite clearly, the Act's mandate requires that in considering uncertainty EPA must err on the side of caution in terms of protecting human health. As the D.C. Circuit held in reviewing the last round of NAAQS revisions, "The Act requires EPA to promulgate protective primary NAAQS even where ... the pollutant's risks cannot be quantified or 'precisely identified as to nature or degree.'" *Am. Trucking Assoc. v. EPA*, 283 F.3d 355, 369 (D.C. Cir. 2002) (quoting Particulate Matter NAAQS, 62 Fed. Reg. 38653); *id.* (citing Ozone NAAQS, 62 Fed. Reg. 38857 (section 109(b)(1)'s "margin of safety requirement was intended to address uncertainties associated with inconclusive scientific and technical information ... as well as to provide a reasonable degree of protection against hazards that research has not yet identified")). *See* H.Rep. 294, 95th Cong., 1st Sess. 49-51 (1977) (explaining amendments designed *inter alia* "[t]o emphasize the preventive or precautionary nature of the act, i.e., to assure that regulatory action can effectively prevent harm before it occurs").

Courts have properly characterized the NAAQS as "preventative in nature." *Ethyl Corp. v. EPA*, 541 F.2d 1, 15 (D.C. Cir. 1976). That is all the more true where, as with ozone, the effects involved include highly serious ones like death and hospitalization. *See Ethyl*, 541 F.2d at 18 ("the public health may properly be found endangered ... by a lesser risk of a greater harm").

NAAQS Must Guard Against Potential Health Effects of Ozone

In keeping with the cautionary and preventative nature of NAAQS, EPA must set primary standards that protect against potential health effects—not just those impacts that have been well established by science.

In a 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 Industries Assn. 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.” *Lead Indus. Ass’n*, 647 F.2d at 1154-55.

In another case dealing with the “margin of safety” requirement of Section 109, the D.C. Circuit rejected industry’s argument that EPA was required to document “proof of actual harm” as a prerequisite to regulation, instead upholding EPA’s conclusion that the Act contemplates regulation where there is “a significant risk of harm.” *Ethyl Corp. v. EPA*, 541 F.2d 1, 12-13 (D.C. Cir. 1976). 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 statutes — and common sense — demand regulatory action to prevent harm, even if the regulator is less than certain that harm is otherwise inevitable. *Id.* at 25. *Accord, Industrial Union Dept. v. American Petroleum Institute*, 448 U.S. 607, 655-56 (1980) (plurality) (agency need not support finding of significant risk “with anything approaching scientific certainty,” but rather must have “some leeway where its findings must be made on the frontiers of scientific knowledge,” and “is free to use conservative assumptions in interpreting the data,” “risking error on the side of overprotection rather than underprotection”)

NAAQS Must Protect Vulnerable Subpopulations

Primary NAAQS must be set at levels that are not only adequate to protect the average member of the population, but also guard against adverse effects in vulnerable subpopulations, such as children, the elderly, and people with heart and lung disease. In fact, courts have repeatedly found that if a certain level of a pollutant “adversely affects the health of these sensitive individuals, EPA must strengthen the entire national standard.” *American Lung Assn. v. EPA*, 134 F.3d 388, 390 (D.C. Cir. 1998) (citations omitted).

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

As the U.S. Court of Appeals for the D.C. Circuit has stated: “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 Assn. v. EPA*, 134 F.3d 388, 390 (D.C. Cir. 1998) (citations omitted). Stated another way, NAAQS must “be set at a level at which there is ‘an absence of adverse effect’ on these sensitive individuals.” *Lead Industries Assn, Inc. v. EPA*, 647 F.2d 1130, 1153 (D.C. Cir. 1980). See also *Washington v. Glucksberg*, 521 U.S. 702 (1997) (people near death are of no less worth than other members of society).

Twenty-two million Americans have been diagnosed with heart disease, nine million with chronic bronchitis, three million with emphysema, while twenty million adults and twelve million children have chronic asthma. The standards must set at a level that protects these and other populations with an adequate margin of safety.

Background concentrations of ozone are irrelevant to the statutory determination of the level of the primary standard that is requisite to protect public health with an adequate margin of safety. The plain language of Clean Air Act section 109, as well as the court decisions cited above dictate that the level of the standard be determined solely by what is requisite to protect public health with a margin of safety, regardless of how that levels might compare to background ozone levels in a given community or nationally.

To the extent that Congress chose to allow consideration of matters such as background concentrations, it did so not in the standard-setting process, but in the Act’s implementation provisions. For example, Congress provided special implementation provisions for certain particulate matter nonattainment areas where anthropogenic sources do not contribute significantly to nonattainment. 42 U.S.C. 7513(f).

EPA Cannot Consider Economic Cost of Meeting NAAQS

In setting or revising primary and secondary NAAQS, EPA cannot consider the economic impact of the standard—only the impact on public health for primary standards and on public welfare for secondary standards.

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 v. American Trucking Assns.* 531 U.S. 457, 465 (2001).

In addition to the plain language of the Clean Air Act, the Court found that Congress had specifically instructed EPA to consider economic costs in other pollution regulations, and would have included similar instructions if it intended EPA to consider economic costs in setting NAAQS. *Id.* at 466-467.

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. §§ 7409(d)(2)(B), 7607(d)(3). *See also American Farm Bureau Federation v. EPA*, 559 F.3d 512 (D.C. Cir. 2009). 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). That is particularly true here, where the panel – composed of recognized health and air quality experts - unanimously recommended that the primary standard be set within the 0.060 to 0.070 ppm range, and recommended that a separate cumulative seasonal secondary standard be established in the range of 7 to 15 ppm/hrs.

Reconsideration of the Standards is Warranted by the Law and the Science

EPA's decision to publish a reconsideration proposal for the ozone NAAQS is justified on multiple grounds. First, reconsideration of the primary standard is warranted by the extensive evidence in the record for 2008 review showing that a 0.075 ppm standard allows adverse health effects affecting many thousands of Americans each year—including premature death and serious morbidity impacts such as hospitalization and asthma attacks. As further detailed below, numerous peer-reviewed studies show adverse health effects at 8-hour ozone levels well below 0.075 ppm, at levels down to and below 0.060 ppm. These include controlled human exposure studies showing adverse effects in healthy individuals at levels as low as 0.060 ppm, and numerous epidemiological studies showing morbidity and mortality effects at levels even below 0.060 ppm. The 0.075 ppm standard adopted in 2008 allows these documented adverse effects to persist, and therefore is not requisite to protect public health as mandated by the Act, let alone protect public health with an adequate margin of safety as the Act requires. The extensive record of evidence led CASAC to unanimously recommend a standard in the range of 0.060 to 0.070 ppm, finding “overwhelming” scientific evidence for this recommendation in the Final Ozone Air Quality Criteria Document and Final Ozone Staff Paper.

Second, EPA in 2008 failed to provide a rational justification for adopting a standard well above levels recommended by CASAC and above levels shown by the science to be associated with adverse health effects. EPA asserted that a 0.075 ppm standard would substantially reduce “exposures of concern,” but the Act requires that the NAAQS not merely “reduce” adverse effects, but that they be “set at a level at which there is ‘an absence of adverse effect’ on [] sensitive individuals.” *Lead Industries*, 647 F.2d at 1153. Moreover, because EPA arbitrarily selected the “exposures of concern,” and because such exposures do not represent the only exposures resulting in adverse effects, the degree of protection provided against such exposures hardly provides a rational basis for finding that a 0.075 ppm standard was requisite to protect public health, or that exposures below 0.075 ppm do not adversely affect public health. EPA also claimed discretion to set the standard at 0.075 ppm “because there is no bright line clearly directing the choice of level.” 73 Fed. Reg. 16477. Even if there is no bright line, however, that

is not a rational basis for allowing adverse health effects to occur at lower levels. The Act does not give EPA the option of setting the standard at a level that allows adverse health effects to persist. EPA further erred in 2008 in claiming that a standard below 0.070 would not be appropriate because the evidence from clinical studies at exposure levels below 0.080 ppm was “quite limited,” and because a few epidemiologically studies did not report positive ozone-related associations below 0.080 (even though many did report such associations). *Id.* This explanation arbitrarily rejected a large body of peer-reviewed work, as well as a key clinical study (the Adams study) that the agency failed to substantively or rationally refute. EPA failed to provide a rational explanation as to why the evidence was too limited below 0.075 in the face of peer-reviewed studies showing actual health effects at those levels, and corroboration of the likelihood of health effects at those levels by other evidence.

EPA also asserted that there were uncertainties associated with estimates of beneficial effects at 0.064 ppm (estimates relied on by CASAC), but nowhere did the agency provide a rational basis for concluding that whatever uncertainty that may exist as to health effects at lower ozone levels (e.g., at 0.064 ppm or lower) was so great as to render such health effects improbable. And nowhere did EPA in 2008 explain how a standard 0.075 ppm included a margin of safety, which requires setting the NAAQS below levels where health effects are certain. *American Lung Assn.*, 134 F.3d at 389. For all the foregoing reasons, EPA’s stated justifications in 2008 for discounting adverse health effects associated with ozone levels below 0.075 ppm and for rejecting CASAC’s recommendation were arbitrary and unlawful. *State Farm*, 463 U.S. at 43; *American Lung Assn.*, 134 F.3d at 392.

Third, CASAC took the unusual step of writing to EPA on April 7, 2008, to protest the agency’s ozone NAAQS decision as being flatly contrary to CASAC’s unanimous recommendation and “not sufficiently protective of public health.” CASAC further stated its opinion that the EPA’s failure to set the primary standard in the 0.060 to 0.070 ppm range failed to ensure an adequate margin of safety for all individuals, including sensitive populations. CASAC’s objections warrant EPA’s reconsideration of the standards.

Fourth, reconsideration of the 2008 standards is warranted by the intervening decision of the D.C. Circuit in *American Farm Bureau Federation v. EPA*, 559 F.3d 512 (D.C. Cir. 2009). There the Court rejected EPA’s rationales for refusing to adopt stronger NAAQS for particulate matter – rationales that in several cases were similar or identical to those relied on by EPA in 2008 in rejecting more protective ozone NAAQS. There, as here, EPA rejected CASAC’s recommendation on the level of the standard on the ground that EPA found it more “appropriate” to discount evidence supporting a more protective standard. The Court found such assertions did not amount to an adequate explanation of why the standard chosen was requisite to prevent adverse health effects. *Id.* 522-23. The *Farm Bureau* Court also found that EPA acted arbitrarily in discounting as too limited the evidence from a study showing adverse health effects in children at pollution levels allowed by EPA’s chosen standard. EPA’s approach was unreasonable, said the Court, “in light of the agency’s obligation to explain how the standard it set would protect ‘not only average healthy individuals, but also ‘sensitive citizens.’” *Id.* 524. So too here. The agency in 2008 rejected as too limited the evidence from the Adams study showing statistically significant lung decrements in healthy persons at ozone levels as low as 0.060 ppm, without explaining why this evidence was too limited, and without explaining why

even more serious health effects would not be expected at 0.060 ppm in more sensitive persons. Indeed, the 2008 ozone decision was particularly deficient in that it failed to incorporate impacts on infants, young children, active children, outdoor workers and other sensitive populations into the standard setting analysis at all, even in light of relevant available data. The *Farm Bureau* Court further held that EPA had failed to show that its chosen standard would provide an adequate margin of safety because, among other things, the agency provided no explanation of how the standard would adequately reduce risks to the elderly or those with certain heart or lung diseases. *Id.* 525-26. As noted above, EPA in 2008 likewise provided no reasoned explanation of how a 0.075 ozone standard would provide an adequate margin of safety generally, much less for sensitive individuals. In sum, the *Farm Bureau* decision establishes that EPA cannot rely on the sorts of conclusory assertions and generalizations it provided in the 2008 ozone NAAQS decision to reject more protective standards recommended by CASAC and supported by peer reviewed evidence. Thus reconsideration of the 2008 action to ensure that EPA's ozone NAAQS decision conforms with the ruling in *Farm Bureau* is plainly warranted.

Scientific Consensus Supports Stricter Standards

A broad scientific consensus has emerged that EPA's 2008 air quality standards for ozone are not sufficient to protect public health.

This consensus is evidenced by the strong, unanimous, and repeated comments of the Clean Air Scientific Advisory Committee (CASAC). The CASAC recommendations are backed up by the endorsement of over 100 leading independent air quality scientists and physicians, the comments of EPA's Children's Health Protection Advisory Committee (CHPAC), the recommendations of EPA's professional staff scientists, and the recommendations of major public health and medical organizations. Further, the State of California and a number of other countries have adopted more stringent standards for ozone than the United States, and the World Health Organization has recently updated its guidelines for air quality standards to recommend lower levels than proposed by EPA.

The recommendations of these prominent scientific and medical panels are more than just optional advisories: they represent repeated peer review and assessment of the scientific research by recognized authorities. The fact that they arrive at similar conclusions bears witness to the strength of the underlying science.

We urge EPA to adopt final standards that follow the strong recommendations of the scientific and medical community.

Clean Air Scientific Advisory Committee

The Clean Air Scientific Advisory Committee is chartered under the Clean Air Act to advise the EPA Administrator on the review of the official limits on the National Ambient Air Quality Standards. Section 109 of the Clean Air Act requires CASAC to recommend to the EPA

Administrator any new NAAQS and revision of existing criteria and standards as may be appropriate.

EPA's Clean Air Scientific Advisory Committee Ozone Review Panel consists of 23 distinguished scientists representing a broad range of disciplines and perspectives. This panel was comprised of the nation's leading experts in ozone air pollution science and health.² The committee conducted a very thorough review of the adequacy of EPA's scientific assessments. The panel met at least six times over the course of the review and submitted detailed oral comments and seven sets of written comments totaling 500 pages on the review plan, the exposure and risk assessments and the draft and final Criteria Document and Staff Paper.

It is remarkable for such a diverse group of scientists to agree upon anything, but in this case they achieved consensus on several key issues in the review.

After reviewing the at least two drafts of the Criteria Document and the Staff Paper, the 23-member CASAC ozone panel reported to EPA these unanimous recommendations:³

- The current standard fails to protect public health from the harmful effects of ozone, the nation's most widespread outdoor air pollutant.
- EPA should set the 8-hour ozone standard much lower—in the range of 0.060 to 0.070 parts per million (ppm)—to adequately protect public health.
- EPA should eliminate the “rounding” loophole that weakens the current standard and leaves millions of Americans unprotected.

CASAC Issued a Unanimous, Clarion Call for the Administrator to Adopt an Ozone Standard More Protective of Public Health

It is highly unusual—perhaps unprecedented—for the CASAC to make such strong and unanimous recommendations. In making a final decision not to lower the annual average PM_{2.5} standard, EPA argued that the CASAC though nearly unanimous, was not totally unanimous, and that “reasonable minds can differ.” However, in the case of ozone, these are absolutely unanimous consensus recommendations. With such strong unanimous scientific conclusions, EPA has no reasonable justification for any different interpretation of the science.

In making its case, the CASAC painstakingly restated its original recommendations in a follow up letter after reviewing the EPA's final Ozone Staff Paper and added an additional recommendation:

- EPA must explicitly account for a “margin of safety” in setting the ozone standards.⁴

² A listing of members of the panel and a description of their expertise is available at: http://www.epa.gov/sab/pdf/casac_ozone_review_panel_biosketches.pdf and is hereby referenced.

³ Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee to Stephen L. Johnson, Administrator, U.S. Environmental Protection Agency, re Clean Air Scientific Advisory Committee's (CASAC) Peer Review of the Agency's 2nd Draft Ozone Staff Paper, EPA-CASAC-07-001, October 24, 2006.

We elaborate on several of these points by highlighting excerpts from the CASAC letters to EPA.

CASAC: The 8-hour ozone standard should be set in the range of 0.060 to 0.070 ppm.

The CASAC explicitly weighted in on the appropriate level for the standard, and backed up their recommendations with scientific evidence drawn from the Staff Paper and the Criteria Document, both of which were extensively vetted in a public peer review process.

“Therefore, the CASAC unanimously recommends a range of 0.060 to 0.070 ppm for the primary ozone NAAQS.”

“Several new single-city and large multi-city studies designed specifically to examine the effects of ozone and other pollutants on both morbidity and mortality have provided more evidence for adverse health effects at concentrations lower than the current standard...These studies are backed-up by evidence from controlled human exposure studies that also suggest that the primary ozone NAAQS is not adequate to protect human health (Adams, 2002; McDonnell, 1996).”

“Furthermore, we have evidence from recently reported controlled clinical studies of healthy adult human volunteers exposed for 6.6 hours to 0.08, 0.06, or 0.04 ppm ozone, or to filtered air alone during moderate exercise (Adams, 2006). Statistically-significant decrements in lung function were observed at the 0.08 ppm exposure level. Importantly, adverse lung function effects were also observed in some individuals at 0.06 ppm (Adams, 2006). *These results indicate that the current ozone standard of 0.08 ppm is not sufficiently health-protective with an adequate margin of safety.* It should be noted that these findings were observed in healthy volunteers; similar studies in sensitive groups such as asthmatics have yet to be conducted. However, people with asthma, and 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 volunteers (Mortimer *et al.*, 2002).”

“Going beyond spirometric decrements, adverse health effects due to low-concentration exposure to ambient ozone (that is, below the current primary 8-hour NAAQS) found in the broad range of epidemiologic and controlled exposure studies cited above include: an increase in school absenteeism; increases in respiratory hospital emergency department visits among asthmatics and patients with other respiratory diseases; an increase in hospitalizations for respiratory illnesses; an increase in symptoms associated with adverse health effects, including chest tightness and medication usage; and an increase in mortality (non-accidental, cardiorespiratory deaths) reported at exposure levels well

⁴ Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee to Stephen L. Johnson, Administrator, U.S. Environmental Protection Agency, re Clean Air Scientific Advisory Committee’s (CASAC) Review of the Agency’s Final Ozone Staff Paper, EPA-CASAC-07-002, March 26, 2007.

below the current standard. The CASAC considers each of the findings to be an important indicator of adverse health effects.”

“Accordingly, the CASAC unanimously recommends that the current primary ozone NAAQS be revised and that the level that should be considered for the revised standard be from 0.060 to 0.070 ppm, with a range of concentration-based forms from the third- to the fifth-highest daily maximum 8-hr average concentration.”

After EPA published its final decision in 2008, CASAC sent a rare letter to the Administrator stating that they disagreed with the decision to set the standard at 75 ppb. These scientists notified the Administrator that they “*do not endorse the new primary ozone standard as being sufficiently protective of public health.*” (emphasis in the original). They urged that the Administrator or his successor “select a more health-protective” standard in the next review cycle, in the range of 60-70 ppb.⁵

In February 2010, CASAC fully endorsed the proposed range, stating:

“EPA has recognized the large body of data and risk analyses demonstrating that retention of the current standard would leave large numbers of individuals at risk for respiratory effects and/or other significant health impacts including asthma exacerbations, emergency room visits, hospital admissions and mortality.”⁶

We quote extensively from these CASAC comment letters because of the importance of these comments to the standard-setting process. CASAC is not just any public commenter. CASAC is not just any EPA advisory committee. CASAC is the Congressionally-chartered advisory committee specifically charged by the Clean Air Act with making recommendations to the Administrator on the revision of air quality standards.

The CASAC committee reviews all the science during the NAAQS review process. Revisions of the standards must by law be based solely on the science.

The current proposal is consistent with CASAC recommendations regarding the level of the primary standard for ozone.

⁵ Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee to Stephen L. Johnson, Administrator, U.S. Environmental Protection Agency, re Clean Air Scientific Advisory Committee Recommendations Concerning the Final Rule for the National Ambient Air Quality Standards for Ozone, EPA –CASAC 08-009, April 7, 2008.

⁶Letter from Dr. Jonathan M. Samet, Chair, Clean Air Scientific Advisory Committee to The Honorable Lisa P. Jackson. Review of EPA’s proposed Ozone National Ambient Air Quality Standard (*Federal Register*, Vol. 75, No. 11, January 19, 2010), EPA-CASAC-10-007, February 19, 2010.

EPA Children's Health Protection Advisory Committee

The EPA Children's Health Protection Advisory Committee (CHPAC) is a body of researchers, academicians, health care providers, environmentalists, children's advocates, professionals, government employees, and members of the public who advise EPA on regulations, research, and communication issues relevant to children.⁷

On March 23, 2007, the Committee wrote a letter to then EPA Administrator Stephen L. Johnson about the review of the ozone standards.⁸ The Committee made the following specific recommendations on the form and level of the standards, based upon concern about impacts of ozone on children's health. A follow up letter on September 4, 2007, after the proposal was issued reiterated the committee's concerns that the proposed standards will not adequately protect the 73.7 million children in the U.S.⁹

CHPAC: We urge that the lower- and more child protective- value of 0.060 ppm be selected from the range recommended by CASAC.

“As pediatricians, public health and environmental professionals drawn from academia, government, industry and public interest organizations, we would like to again express our unanimous opinion that the 8 hour ozone standard should be set at the lowest level offered by the Clean Air Scientific Advisory Committee (CASAC), 0.060 ppm, in order to adequately protect the health of children with an appropriate margin of safety (CHPAC letter, March 23, 2007). This opinion is based on the existing scientific studies of children, which demonstrate serious adverse health effects of ozone exposure, including exacerbation of asthma with attendant increases in medication use, hospitalization, and missed school days, and impairment of normal lung development. It is also based on consideration of the evidence that disruption of lung development may result in permanent health consequences in children exposed to ozone.”

CHPAC: Children experience a wide variety of health impacts from ozone exposure that should be recognized in considering benefits from lowering the 8-hour ozone standard.

“Children are especially susceptible to ozone exposures because they have higher levels of physical activity, higher ventilation rates, and more frequent outdoor activities on average than adults in the same setting. Furthermore, the lungs undergo extensive development during childhood and adolescence, making children especially vulnerable to permanent alteration in lung function and chronic lung disease later in life if their normal development is disturbed.”

⁷ http://yosemite.epa.gov/oehp/ochpweb.nsf/content/whatwe_advisory.htm

⁸ Letter from Melanie A. Marty, Ph.D., Chair, Children's Health Protection Advisory Committee, to Stephen L. Johnson, Administrator, U.S. EPA, re: Review of the NAAQS for Ozone: Policy Assessment of Scientific and Technical Information, March 23, 2007, p. 686.

⁹ Letter from Melanie A. Marty, Ph.D., Chair, Children's Health Protection Advisory Committee, to Stephen L. Johnson, Administrator, U.S. EPA, re: Proposed NAAQS for Ozone, 4 September, 2007.

EPA Staff Scientists

For the last twenty five years, an integral part of the NAAQS review process has been the preparation of a “Staff Paper” that bridges the gap between the science assessment in the Criteria Document, and the policy issues concerning the setting of air quality standards. Typically, the Staff Paper prepared by EPA staff scientists in the Office of Air Quality Planning and Standards frames policy issues based on the scientific review and makes recommendations for ranges from which the EPA Administrator can select proposed and final standards.

The Staff Paper reached some strong conclusions regarding the strength of the new evidence available in this review:

EPA Staff: Adverse health effects caused at levels below the current standard.

“...we conclude that there is important new evidence demonstrating that exposures to O₃ at levels below the level of the current standard cause or are clearly associated with a broad array of adverse health effects in sensitive populations. For example, we note new direct evidence of transient and reversible lung function effects and respiratory symptoms in some healthy individuals at exposure levels below the level of the current standard. In addition, there is now epidemiological evidence of statistically significant O₃-related associations with lung function and respiratory symptom effects, respiratory-related ED [emergency department] visits and hospital admissions, as well as possibly increased mortality, in areas that likely would have met the current standard. There are also many epidemiological studies done in areas that likely would not have met the current standard but which nonetheless report statistically significant associations that generally extend down to ambient O₃ concentrations that are well below the level of the current standard. Further, there are a few studies that have examined subsets of data that include only days with ambient O₃ concentrations below the level of the current standard, or below even much lower O₃ concentrations, and continue to report statistically significant associations. Our level of confidence in the findings from these studies is not related to whether they were done in areas that likely would or would not have met the current standard.” (SP p. 6-46).¹⁰

In considering this evidence, EPA Staff Scientists conclude that the current standard is clearly inadequate to protect public health.

EPA Staff Scientists: Evidence questions the adequacy of the existing standard

“We conclude that *the overall body of evidence clearly calls into question the adequacy of the current standard* and provides strong support for consideration of an O₃ standard that would provide increased health protection for sensitive

¹⁰ U.S. EPA, Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA-452/R-07-003, January 2007.

groups, including asthmatic children and other people with lung disease, as well as all children and older adults, especially those active outdoors, and outdoor workers, against an array of adverse health effects that range from decreased lung function and respiratory symptoms to serious indicators of respiratory morbidity including ED visits and hospital admissions for respiratory causes, and possibly cardiovascular-related effects and mortality. We also conclude that risks projected to remain upon meeting the current standard, based on the exposure and risk assessment, are indicative of risks to sensitive groups that can reasonably be judged to be important from a public health perspective, which reinforces our conclusion that consideration should be given to revising the level of the standard so as to provide increased public health protection.”

The Staff Paper goes on to recommend that:

“consideration be given to a standard level within the range of somewhat below 0.080 ppm to 0.060 ppm, reflecting our judgment that a standard set within this range could provide an appropriate degree of public health protection and would result in important improvements in protecting the health of sensitive groups.”

It is significant that the lower end of the staff recommended range is 0.060 ppm, consistent with the recommendations of CASAC.

We highlight this specific conclusion here, but note that it is borne out by the extensive interpretation of the scientific data and hundreds of pages of analyses undertaken by EPA staff scientists as part of the policy assessment process.

Medical Societies

A number of prominent medical and scientific organizations including the American Medical Association, the American Academy of Pediatrics, the International Society for Environmental Epidemiology¹¹ and the American Thoracic Society have gone on record in support of more stringent ozone standards.

American Academy of Pediatrics

The American Academy of Pediatrics (AAP) is an organization of 60,000 pediatricians committed to the attainment of optimum health for infants, children, adolescents and young adults. In late 2004, the American Academy of Pediatrics (AAP) published a major review of ambient air pollution and health hazards to children. The review concluded that the 1997 NAAQS for ozone may not adequately protect the health of infants and children. The paper cites studies showing declines in lung function, hospitalizations for respiratory tract illness in young children, emergency department visits for asthma, and asthma exacerbations at levels at or below the current standards. In addition, cumulative childhood exposure to ozone may affect

¹¹ Letter from Daniel Wartenberg, PhD to Administrator Johnson, RE: Docket ID No. EPA-HQ-2005-0172, October 5, 2007.

lung function when exposed children reach young adulthood. The AAP review suggests that ozone may be toxic to children at concentrations lower than the current standard.¹²

In a September 12, 2006 letter commenting on the second draft Staff Paper, AAP wrote to EPA Administrator Stephen Johnson and stated that the current ozone air quality standards do not protect children and must be strengthened.

“Children are especially susceptible to the adverse effects of ambient air pollution due to their extensive lung growth and development after birth. In fact, 80 percent of the alveoli, the smallest portion of the lungs where gas exchange occurs, are formed after a child is born, and the lungs continue to develop through adolescence. During the early post-neonatal period, the developing lung is highly susceptible to damage from exposure to environmental toxicants. Children also have increased exposure to many air pollutants compared with adults because of their higher minute ventilation (the amount of air breathed into or out of the lungs per minute) and higher levels of physical activity. Because children spend more time outdoors than do adults, they have increased exposure to outdoor air pollution.¹³

Ozone is a powerful oxidant gas and respiratory tract irritant in adults and children. Exposure to ozone is known to cause shortness of breath, chest pain when inhaling deeply, wheezing, coughing, and inflammation in the lungs at lower concentrations than other ambient gaseous pollutants. Summer camp studies and other epidemiological studies have found that children have decreases in lung function, increased respiratory tract symptoms and asthma exacerbations, increased emergency room visits, and increased school absences linked to days with high levels of ambient ozone.¹⁴ Hospitalizations and premature mortality have also been linked to increases in ozone.¹⁵

In addition to the increase in short-term respiratory symptoms, long-term exposure to ozone may have lifelong consequences for children. A prospective study in Southern California found children involved in high levels of team sports who grew up in communities with high ozone levels were at increased risk for developing asthma.¹⁶ Another study found that chronic, long-term exposure to ambient ozone was associated with decreased levels of small airways function in college students.¹⁷

¹² American Academy of Pediatrics Committee on Environmental Health. Ambient Air Pollution: Health Hazards to Children. *Pediatrics* 2004; 114: 1699 -1707.

¹³ Ibid.

¹⁴ Ibid.

¹⁵ Bates DV. Ambient ozone and mortality. *Epidemiology* 2005; 16: 427-429.

¹⁶ McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 2002; 359: 386-391.

¹⁷ Tager IB, Balmes J, Lurmann F, Ngo L, Alcorn S, Kunzli N. Chronic exposure to ambient ozone and lung function in young adults. *Epidemiology* 2005; 16: 751-759.

...The AAP strongly recommends a tighter 8-hour standard for ozone and supports adoption of a revised ozone standard of 0.070 ppm (8-hour average, not to be exceeded) or lower.”

American Thoracic Society

With more than 18,000 members, the American Thoracic Society is a leading medical association dedicated to advancing lung, critical care and sleep medicine. The Thoracic Society has participated extensively in the review of the draft Criteria Document and Staff Paper for ozone. In July 2007, the American Thoracic Society published an editorial in its peer-reviewed journal, the *American Journal of Respiratory and Critical Care Medicine*, endorsing an 8-hour average ozone standard of 0.060 ppm, based upon concerns about both child and adult health.

“Among sensitive populations, children may be more at risk of the adverse effects of air pollution than adults for several reasons. First, children have a higher level of activity and a higher minute ventilation compared with adults, which increases the effective dose of inhaled pollutant (reviewed in Reference [1](#)). Second, children spend more time outdoors than adults do, increasing exposure to ambient air pollutants ([2](#)). Third, lung development is a long-term process. Although the human lung needs to be sufficiently formed at birth to perform its primary function, gas exchange, lung growth continues for an extensive period (8–12 yr) after birth ([3](#)). During this time, there are multifold increases in overall lung size, active cellular differentiation, cell division, and alveolar formation. As a result, airways change in size and shape with maturation, altering deposition patterns. In addition, lung function also continues to change, increasing until late adolescence in both males and females, when it plateaus ([4–6](#)). This period of lung growth and development is a critical one in which a deficit in growth could be carried throughout life.

Increasing numbers of epidemiological studies suggest that ozone is detrimental to children's respiratory health, including increased hospitalizations, emergency room visits, and decreased pulmonary function ([7–9](#)). Current ozone levels in Canada's largest cities are associated with increased hospitalization for respiratory problems in neonates under 1 month of age ([10](#)). Ozone levels lower than current U.S. EPA standards have also been associated with difficulty breathing in infants (aged 3 mo to 1.5 yr), particularly in those with asthmatic mothers ([11](#)), and with increased use of rescue medication in children with asthma under 12 years of age using maintenance medication ([12](#)). 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 ([13](#)). The effects of childhood exposure may be long-lasting. Decrements in small airways function have been reported in college freshmen who have grown up in polluted areas of California's South Coast Air Basin ([14, 15](#)).

Growing concern is emerging regarding the relative risks of increased morbidity and mortality among adults as well. A series of recently published meta-analyses and primary national-scale epidemiological studies have documented consistent

associations between premature mortality and ozone exposures below the current 8-hour standard of 0.08 ppm (16). Controlled human exposure studies of healthy adults have demonstrated reduced lung function, increased respiratory symptoms, changes in airway responsiveness, and increased airway inflammation following 6.6-hour exposures to 0.08 ppm ozone (17, 18). Recent studies demonstrate that some of the individuals tested experience these adverse effects at concentrations of 0.06 ppm and below (19).”¹⁸

In a 2010 editorial published on behalf of the American Thoracic Society Environmental Health Policy Committee, the Society again urged EPA to adopt a protective NAAQS for ozone of 0.060 ppm per 8-hour average. “Second chances are rare and should not be wasted,” stated the article. “As a growing body of evidence shows, such a standard is needed to protect the public from the known adverse health effects of ozone.”¹⁹

State Governments

State of California

California completed a comprehensive review of its state ozone air quality standards in April 2005, under the Children’s Environmental Health Protection Act. The California Air Resources Board unanimously approved establishment of a new 8-hour standard for ozone of 0.070 ppm, not to be exceeded. This standard supplements the pre-existing 1-hour state standard of 0.09 ppm, which was retained.

The “not to be exceeded” form of the California 8-hour standard is more protective than the current or proposed form of the NAAQS, which allows multiple exceedances over a several year period before a violation of the standard is registered.

The California standard is based primarily on numerous controlled human exposure studies of healthy individuals which demonstrate reduced lung function, increased respiratory and ventilatory symptoms, increased airway hyperreactivity, and increased airway inflammation following 6.6 to 8-hour exposures to 0.08 ppm ozone.

Additionally, evidence from epidemiological studies of several health endpoints including premature death, hospitalization, respiratory symptoms, and restrictions in activity and lung functions indicate that concentrations below the current federal standard cause adverse health effects.²⁰

¹⁸ Pinkerton KE, Balmes JR, Fanucchi MV, Rom WN. Editorial: Ozone, A Malady for All Ages. *Am J Res Crit Care Med* 2007; 176: 107-108. Available at: <http://ajrccm.atsjournals.org/cgi/content/full/176/2/107>

¹⁹ Dey R, Van Winkle L, Ewart G, Balmes J, Pinkerton K. A Second Chance: Setting a Protective Ozone Standard. *Am J Respir Crit Care Med* 2010; 181: 297-299.

²⁰ ARB. Evidence on the Health Effects of Ozone Provided from Hundreds of Studies. Presentation available at: <http://arb.ca.gov/research/aaqs/ozone-rs/aqac/pres/staff-1.pdf>

Ozone Transport Commission

The Ozone Transport Commission, which represents the thirteen Eastern states from Virginia to Maine, has gone on record urging EPA to propose standards within the range recommended by CASAC. At their June 6, 2007 meeting, the Commissioners approved a statement on the EPA review of the ozone NAAQS. The statement says, in part:

“The CAA calls on EPA to rely heavily on the science and CASAC’s recommendations in setting both the primary and secondary NAAQS. OTC supports the work of the CASAC and urges EPA to give great weight to the recommendations of the CASAC for a revision of the ozone NAAQS as set forth in its March 26, 2007 letter to EPA Administrator Johnson.”²¹

This is a powerful consensus statement from the environmental commissioners of the Mid-Atlantic and Northeastern states.

National Health and Environmental Organizations

A broad range of public health, medical, and environmental organizations are on record in support of a substantially strengthened ozone standard of 0.060 ppm, 8-hour average. In addition to the commenters, over a dozen additional national health and environmental organizations sent a letter to EPA on April 16, 2007 advocating a standard of 0.060 ppm, and elimination of the rounding loophole.²² They include the American Lung Association, American Academy of Pediatrics, American Public Health Association, Alliance for Healthy Homes, Appalachian Mountain Club, Asthma and Allergy Foundation of America, Clean Air Task Force, Clean Air Watch, Environmental Defense, Environmental Integrity Project, Greenpeace, National Environmental Trust, National Refinery Reform Campaign, Natural Resources Defense Council, Physicians for Social Responsibility, Science and Environmental Health Network, Sierra Club, Smart Growth America, Trust for America’s Health, Union of Concerned Scientists, and U.S. Public Interest Research Group.

Additional comments supporting a stronger ozone standard of 0.060 ppm were filed by a coalition of health organizations including the American Heart Association, American Nurses Association, National Association of County and City Health Officials, Health Care Without Harm, Institute for Children’s Environmental Health, and others.²³

²¹ Statement of the Ozone Transport Commission Concerning Setting of a New National Ambient Air Quality Standard for Ozone, Adopted by the Commission on June 6, 2007, signed by David Paylor, Director, VA DEP, Chair, Ozone Transport Commission. Available at: <http://www.otcair.org/document.asp?fview=Formal#>

²² Letter to EPA Administrator Stephen L. Johnson, re: Science Compels Stricter NAAQS for Ozone, from the heads of the American Lung Association, American Academy of Pediatrics, American Public Health Association, Asthma and Allergy Foundation of America, and 16 national health and environmental organizations, April 16, 2007. Available at: <http://www.cleanairstandards.org/wp-content/uploads/2007/04/ltr-from-public-health-enviro-groups-on-ozone-naaqs-04-16-07.pdf>

²³ Letter to EPA Administrator Stephen L. Johnson re: Proposed National Ambient Air Quality Standards (NAAQS) for Ozone--Docket ID Number EPA-HQ-OAR-2005-0172, October 5, 2007 signed by American Heart Association and 9 other national health organizations.

Speaking out in support of a standard set at 0.060 ppm during this review are many of these same groups, plus additional patient advocacy groups. New supporters include the Asthma and Allergy Foundation of America, Children's Environmental Health Network, the Foundation for Sarcoidosis Research and The LAM Foundation.²⁴

International Reviews

World Health Organization

In October 2006, the World Health Organization (WHO) revised their international air quality guidelines for ozone.²⁵ The prior guideline for 8-hour average ozone concentrations of 120 $\mu\text{g}/\text{m}^3$ (0.061 ppm) was reduced to 100 $\mu\text{g}/\text{m}^3$ (0.051 ppm). The previous guideline and the new guideline are both substantially lower than 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, independent of the effects of particulate matter. Similar associations have been observed in both North America and Europe. These time-series studies have shown effects at ozone concentrations below the previous guideline, 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.

The WHO recommendations were developed by a work group of dozens of leading international air quality and health scientists. According to WHO, the previously recommended guideline value, "which was fixed at 120 $\mu\text{g}/\text{m}^3$ 8-hour mean [61 ppm], has been reduced to 100 $\mu\text{g}/\text{m}^3$ [51 ppm] based on recent conclusive associations between daily mortality and ozone levels occurring at ozone concentrations below 120 $\mu\text{g}/\text{m}^3$."²⁶

International Standards

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 1 that follows, numerous developed and developing countries have promulgated ozone standards that are more stringent than the current U.S. standard.

²⁴ Letter to The Honorable Lisa P. Jackson from the American Academy of Pediatrics, American Association of Cardiovascular and Pulmonary Rehabilitation, American College of Chest Physicians, American College of Preventive Medicine, American Heart Association, American Lung Association, American Medical Association, American Public Health Association, American Thoracic Society, Asthma and Allergy Foundation of America, Foundation for Sarcoidosis Research, The LAM Foundation, National Association for the Medical Direction of Respiratory Care, National Association of County and City Health Officials, and Physicians for Social Responsibility, March 22, 2010. Submitted to the Docket EPA-HQ-OAR-2005-0172.

²⁵ 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>

²⁶ <http://www.who.int/mediacentre/factsheets/fs313/en/index.html>

Country	1 hour	8 hour	Exceedances Allowed per year
WHO		51	
European Union -2010		61	25
Australia	100	80 (4-hr)	1
Cambodia	102		
Canada		65	3
Hong Kong	122		
Indonesia	120		
(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

Table 1: Comparison of Ozone Standards Worldwide (ppb)²⁷

Individual Scientists

Over 100 leading air pollution scientists and physicians wrote to EPA on April 4, 2007 to express strong support for a revised primary eight-hour ozone ambient air quality standard between 60 and 70 ppb, consistent with the CASAC recommendation.

²⁷ Compiled from online sources: http://www.cleanairnet.org/caiasia/1412/articles-71889_Ozone_standards.pdf; www.airquality.co.uk/archive/standards.php; www.epa.ie/whatwedo/monitoring/airstandards/; www.epa.gov/ttncaat1/cica/airq_e.html; www.mfe.govt.nz/publications/rma/user-guide-draft-oct05/html/page3.html; www.environment.gov.au/atmosphere/airquality/standards.html; http://www2.dmu.dk/AtmosphericEnvironment/Expost/database/docs/AQ_limit_values.pdf

Arguments for Retaining the Existing Standard are Flawed

The Supreme Court decision in *American Trucking* in 2001 closed the door firmly on basing the NAAQS on anything other than the protection of public health with an adequate margin of safety. The Clean Air Act's approach to setting air quality standards provides American families with a transparent and unmitigated science-grounded benchmark for determining whether the air in their neighborhood or community is safe to breathe.

We have heard many arguments from opponents for not revising the standard. Here are some of the most common. Following each is a brief rebuttal. However, we repeat that even if these were true, the only acceptable basis for the standard is the protection of public health.

Flawed argument #1: EPA is “moving the goal post”

The argument claims that EPA is “moving the goal post” before the work on the 1997 NAAQS is complete.

The Clean Air Act requires EPA to protect the public from air pollution and clean up the air so that pollution no longer harms health. The Act requires EPA to review the science and the standard every five years so that protection can be maintained. The statute does not give EPA the option of withholding a standards revision where warranted by the science merely to allow more time for states to comply with the pre-existing standard.

The Clean Air Act is designed to have EPA base its decisions on the most current, best available information. Congress built into the law the requirement to review the science and the standard every five years, knowing that new research could warrant revisions to the standard. The mandate for review every five years hardly precludes EPA from correcting an erroneous decision in the most recent review, where the decision was unlawful and arbitrary at the time it was made.

Flawed argument #2: “A tighter standard will hurt local economies”

As discussed extensively above, this argument is legally irrelevant to EPA's decision on the ozone NAAQS, which must be based exclusively on the protection of public health -- not economics. Even if it were relevant, however, the claim that stronger standards will harm local economies fails to recognize the evidence of the last 40 years that show that reduced emissions and reduced ozone levels have not harmed the economy. Almost every major city in the U.S. has been in nonattainment during the previous 40 years, including cities such as Los Angeles, Houston, and Washington, DC, and economic growth has continued.

Flawed argument #3: Tighter standard would cripple the U.S. economy

Similar in theme to the previous argument and equally flawed, these Cassandras warn of devastation for the entire U.S. economy if new, tighter standards are adopted. This quote from the National Association of Manufacturers' website on June 19, 2007, argues:

“Does crippling U.S. manufacturing with higher energy costs -- the unavoidable result of regulatory overreach -- serve the public interest when any reduction in smog is marginal, at best?”

Again, these assertions are completely irrelevant to EPA's NAAQS decision, which must be grounded exclusively in protection of public health. Even if they were relevant, however, EPA's own graph, Figure A below, tracking the growth of the population and the gross domestic product since the passage of the Clean Air Act Amendments of 1990 shows that stronger standards do not harm the economy:

“The combined emissions of the six common pollutants and their precursors (PM_{2.5} and PM₁₀, SO₂, NO_x, VOCs, CO, and lead) dropped 41 percent on average since 1990, as shown in Figure 3. This progress has occurred while the U.S. economy continued to grow, Americans drove more miles, and population and energy use increased.”²⁸

The health costs—the human toll of air pollution—are huge: illness, emergency room visits, asthma attacks and even premature death. The benefits of cleaning up air pollution have proven time and time again to be overwhelmingly greater than the costs. Indeed, in addition to extraordinary public health improvements, the ancillary benefits of stricter ozone standards may include shifts towards improved public transit and urban planning to help reduce mobile source emissions and towards cleaner, more efficient power sources to reduce stationary source emissions. In other words, the ozone standards will generally help push the country towards a productive clean energy economy, creating jobs and improving quality of life in the process.

Each year the White House Office of Management and Budget analyzes the costs and benefits of such regulatory requirements as part of its annual report to Congress. The most recent estimate of the last ten years of EPA's air pollution regulations concludes that total benefits outweigh the costs by as much as 18 to 1.²⁹ What isn't usually discussed by opponents, but present in these OMB analyses are the huge costs associated with having people breathe polluted air, costs that are especially borne by children and teens, seniors, and people with chronic lung disease. We have 40 years of experience to show that cleaning up air pollution does not hurt economic growth.

²⁸ U.S. EPA. Our Nation's Air: Status and Trends Through 2008. February 2010, EPA 454/R-09-002. Available at <http://www.epa.gov/air/airtrends/2010/report/fullreport.pdf>.

²⁹ White House Office of Management and Budget, Office of Information and Regulatory Affairs. 2009 Report on the Costs and Benefits of Federal Regulations. January 2010. Posted at http://www.whitehouse.gov/omb/assets/legislative_reports/2009_final_BC_Report_01272010.pdf.

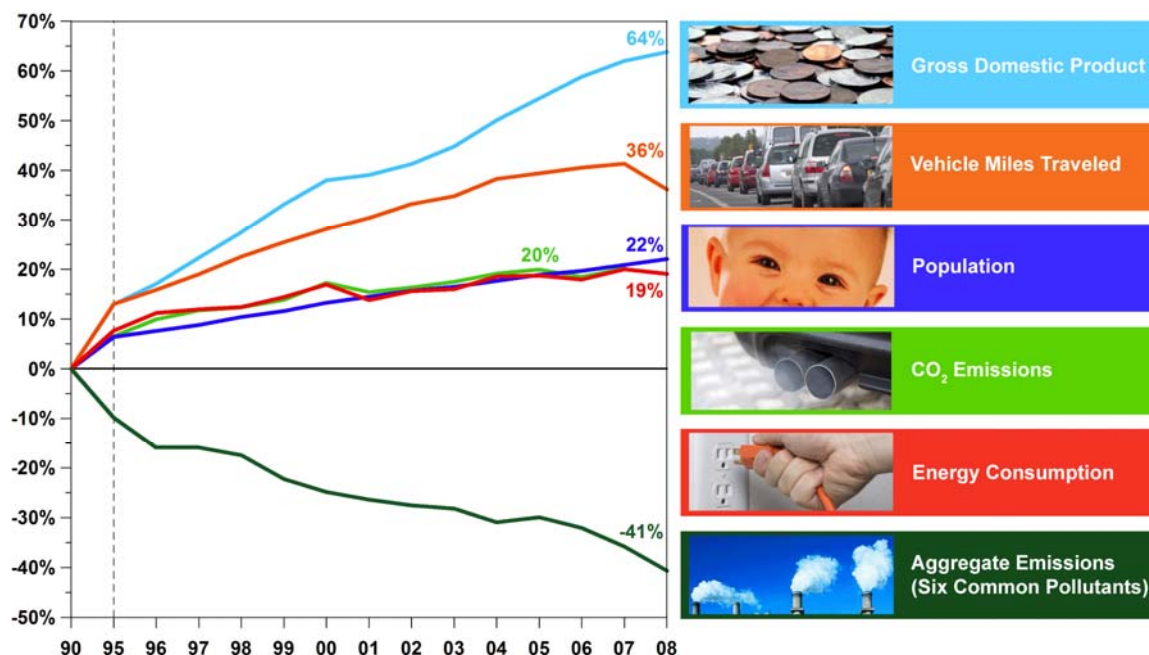


Figure A: EPA Chart showing the relative change in four measures of population and economic growth, in carbon dioxide emissions growth and in criteria pollutant emission reductions from 1970 to 2008. Source: EPA. Our Nation's Air: Status and Trends Through 2008. February 2010, EPA 454/R-09-002.

Flawed argument #4: Standard is impossible to meet / We don't have the technology to meet it

This argument is recycled during every major review of the NAAQS. It sounds like these quotes from the *Fort Worth Star Telegram*, June 16, 2007:

"You're going to have a whole lot of people spending a lot of money endlessly chasing their tail to meet a standard they can never meet."

"That's not us trying to get out of what we might need to do; it just gets down to the fact there's not much more we can squeeze out of the thing...It would be very, very tough."

Once again, this argument is legally irrelevant: The Act requires EPA to set the NAAQS based solely on what is requisite to protect public health, not only someone's notion of what level of air quality is achievable.

The argument that stronger ozone standards are not achievable is belied by the record. America has faced this challenge and met it since Congress strengthened the Clean Air Act in 1970.

Technological breakthroughs like the catalytic converter or cleaner filters for diesel school buses, equipment to clean up emissions from factories all happened because tighter standards pushed us and American ingenuity stepped up to solve problems. The U.S. leads the world in pollution control innovation.

In February 2010, EPA identified 113 communities across the nation with in nonattainment for the ozone health standard adopted in 1997. Based on preliminary air quality data, EPA estimates that all but 31 of those areas have ozone concentrations that meet that health standard. Since 1980, peak ozone concentrations monitored at some 275 sites across the country have declined by 25 percent.³⁰ See Figure B below. These pollution reductions have prevented hospital admissions and school absences for respiratory illnesses, and have saved lives.

An updated analysis of progress in ozone reduction demonstrates that most communities experienced significant decreases in ozone concentrations. A comparison of ozone concentrations, 2001-2003 with 2006-2008 reveals that almost 500 locations experienced decreases of at least 0.006 ppm.³¹ Another 385 sites experienced little change (+/- 0.005 ppm) and only 23 locations experienced significant increases in ozone. Once again, this demonstrates the feasibility of achieving significant decreases in ozone concentration across the United States.

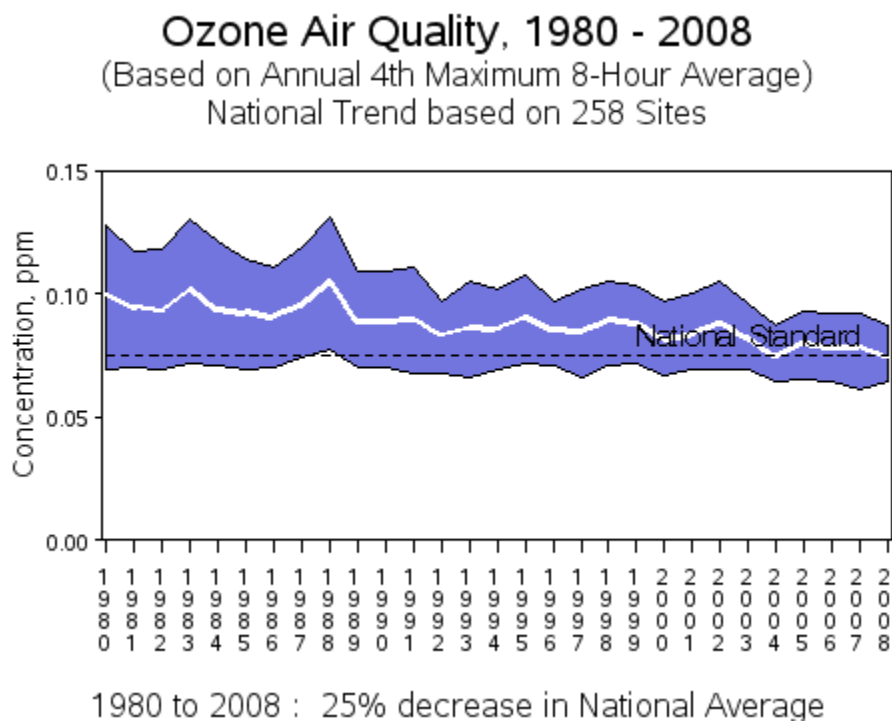


Figure B: Ozone Air Quality, 1980 - 2008. Source U.S. EPA, Air Trends—Ozone. National Trends in Ozone Levels. Available at <http://www.epa.gov/air/airtrends/ozone.html>.

³⁰ U.S. EPA. Our Nation's Air: Status and Trends through 2008. Contract No. EP-D-05-004, Work Assignment No. 5-07. February 2010. <http://www.epa.gov/airtrends/2010/index.html>

³¹ U.S. EPA, Air Trends—Ozone. National Trends in Ozone Levels. Available at <http://www.epa.gov/air/airtrends/ozone.html>. Accessed March 22, 2010.

It will take additional efforts in many communities to meet the new standard, but we can do it with new cleaner technology and public input. States will have time to plan and adopt new tools to accomplish this. EPA needs to do more, too, including adopting new rules to put tighter controls on coal-fired power plants and industrial boilers.

Flawed Argument #5: It will be impossible to meet these standards because they approach natural background concentrations.

As discussed above, the ease or difficulty of meeting a particular standard level is legally irrelevant to setting a NAAQS. That is true regardless of whether the claim of difficulty in compliance is due to background levels or other factors. In any event, such claims are not well-grounded with respect to ozone.

EPA defines policy relevant background as those concentrations that would exist in the absence of North American emissions. Some have argued that frequent occurrences of ozone concentrations above 50–60 ppbv at remote northern U.S. sites in spring are of stratospheric origin, implying that the proposed ozone standard may be unattainable.

We contend that the GEOS-CHEM model is the best tool available to derive estimates of background concentrations, should EPA continue to pursue this approach. PRB ozone and ozone precursors include photochemical interactions of natural sources of VOCs, CO and NO_x; long range transport of O₃ and O₃ precursors from outside of North America, and exchange of ozone between the stratosphere and troposphere.³² Computer modeling is required and the state-of-the-art global photochemical transport model GEOS-CHEM is appropriate to estimate these concentrations. This model has been peer-reviewed.³³ This model finds that background ozone concentrations are generally 15-35 ppb, lower than the 40 ppb assumed by EPA in prior reviews. The CASAC favorably reviewed the GEOS-CHEM model when reviewing the Criteria Document, and concurred that it represented a major advance in characterizing background concentrations in North America.³⁴

While the GEOS-CHEM model has received generally high marks from both EPA and the CASAC, evidence shows that overestimates PRB ozone in the southeastern U.S. That overestimation minimizes risk estimates for Atlanta, one of the cities modeled in the risk assessment. According to the EPA, several papers have evaluated the accuracy of the GEOS-

³² U.S. EPA. 2007. Staff Paper , p. 2-54.

³³ Fiore A, Jacob DJ, Liu H, Yantosca RM, Fairlie TD, Li Q. Variability in surface ozone background over the United States: Implications for air quality policy. *J Geophys Res* 2003; 108: 4787, DOI 10.1029/2003JD003855. Correction published 21 February 2004; and Fiore AM, Jacob DJ, Bey I, Yantosca RM, Field BD, Fusco AC, Wilkinson JG. Background ozone over the United States in summer: Origin, trend, and contribution to pollution episodes. *J Geophys Res* 2002; 107: 4275, DOI 10.1029/2001JD000982.

³⁴ CASAC letter to EPA Administrator Stephen L. Johnson, EPA-SAB-CASAC-05-010, re: Clean Air Scientific Advisory Committee (CASAC) Ozone Review Panel's Peer Review of the Agency's *Air Quality Criteria for Ozone and Related Photochemical Oxidants (First External Review Draft)*, Volumes I, II, and III, (EPA/600/R-05/004aA, bA, and cA, January 2005); Comments of Ted Russell, p. C-98, June 22, 2005.

CHEM simulation and demonstrated that PRB ozone values are inflated in the southeastern U.S. by as much as 10 ppb.³⁵

Fiore et al. 2003 has shown that a 3-D global model of tropospheric chemistry reproduces much of the observed variability in U.S. surface ozone concentrations, including the springtime high-ozone events, with only a minor stratospheric contribution (always <20 ppbv). They conclude that stratospheric intrusions might occasionally elevate surface ozone at high-altitude sites, but that these events are rare and would not compromise the ozone air quality standard. The Criteria Document concludes that ozone background is generally 15–35 ppbv. It declines from spring to summer and further decreases during ozone pollution episodes. These concentrations are well below the proposed standards.

More recently Wang et al (2009) have applied a newer version of the GEOS-Chem model used by Fiore et al (2003).³⁶ They have estimated background ozone levels throughout the U.S. and also have examined ozone levels that would result if emissions from Canada and Mexico were included.

Their analysis simulated ozone levels in the summer of 2001 and eliminated anthropogenic emissions in North America compared to those only in the U.S. The findings indicated that the 2001 mean North American and U.S. background concentrations were 26±8 ppb and 30±8 ppb. As seen in the figures below, even with emissions from Canada and Mexico included, ozone levels were still below 50ppb.

³⁵ U.S. EPA. 2007. Staff Paper, p. 2-54.

³⁶ Wang H, Jacob DJ, Le Sager P, Streets DG, Park RJ, Gilliland AB, van Donkelaar A. Surface ozone background in the United States: Canadian and Mexican pollution influences. *Atmospheric Environment* 2009; 43: 1310–1319.

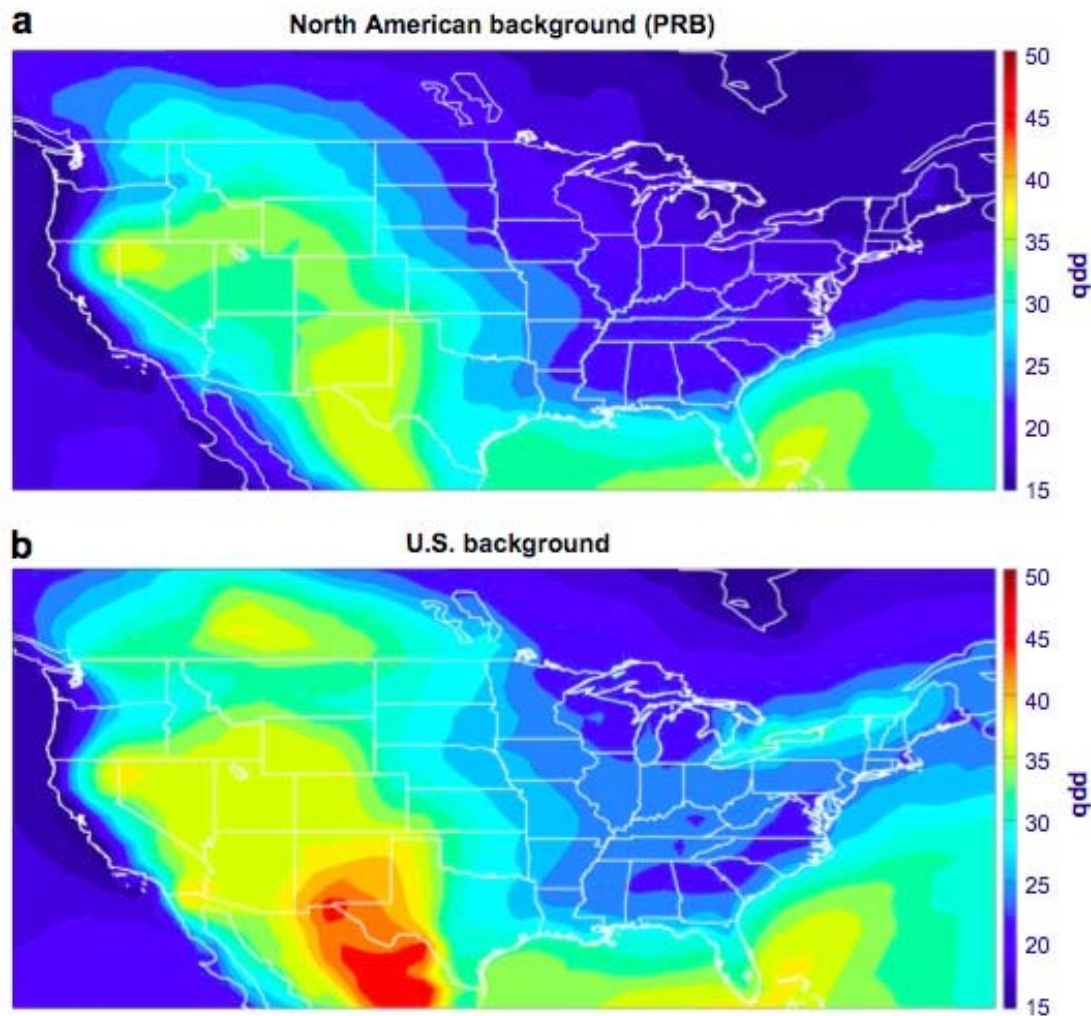


Figure C: Jun–Aug mean daily-8 h-max ozone concentrations in surface air for (a) North American background, (b) US background

Source: Wang H, Jacob DJ, Le Sager P, Streets DG, Park RJ, Gilliland AB, van Donkelaar A. Surface ozone background in the United States: Canadian and Mexican pollution influences. *Atmospheric Environment* 2009; 43: 1310–1319.

International controls to reduce the hemispheric pollution background would facilitate compliance with ozone standards in the United States.

According to the 2007 HTAP report,³⁷ for ground-level ozone, the hemispheric background concentration of 20–40 ppbv (parts per billion by volume) includes a large anthropogenic and intercontinental component. As part of the HTAP model intercomparison, a set of emission perturbation experiments were conducted to compare model estimates of how emission changes

³⁷ Hemispheric Transport Of Air Pollution 2007; Air Pollution Studies No. 16; Interim report prepared by the Task Force on Hemispheric Transport of Air Pollution acting within the framework of the Convention on Long-range Transboundary Air Pollution

in one region of the world impact air quality in other regions. The preliminary results of these experiments suggest that, under current conditions, local and regional emission changes have the greatest impact on surface air quality, but that changes in intercontinental transport can have small yet significant effects on surface concentrations. The benefits of measures to decrease intercontinental transport would be distributed across the Northern Hemisphere (HTAP, 2007). The final HTAP assessment is currently being completed; this work should provide substantive foundations for ongoing international deliberations on air quality.

Scientific Evidence Exists of Adverse Effects at Low Concentrations

EPA has proposed to set an 8-hour average primary ozone standard within the range of 0.060 ppm to 0.070 ppm. This range is consistent with the recommendations of CASAC, EPA staff scientists, and the broader public health, medical and scientific community. These comments will review the scientific evidence for setting a final air quality standard at the lower end of the proposed range, that is, a 0.060 ppm 8-hour standard. The evidence underscores the EPA's decision that the 0.075 ppm standard set in 2008 fails to provide protection required by the Clean Air Act.

Chamber Studies Show Need for More Protective Standards

Studies where human volunteers are exposed to known concentrations of ozone in an experimental chamber are the gold standard in ozone research. Several controlled human exposure studies provide evidence of harm down to 0.06 ppm concentrations of ozone. The evidence also makes it clear that the existing standard of 0.075 ppm fails to provide enough protection to prevent effects evident in chamber studies at 6.6- to 8-hour exposures to 0.08 ppm ozone.

A series of clinical chamber studies in the early 1990's demonstrated that a host of adverse health effects -- decrements in pulmonary function, increased respiratory symptoms such as cough and shortness of breath, heightened airway responsiveness, and inflammation of the airways-- occurred with at 6.6- to 8-hour exposures to 0.08 ppm ozone.

Chamber studies from the late-1980s to mid-1990s demonstrated a string of adverse health effects including³⁸:

- reduced lung function
- respiratory symptoms
- airway responsiveness
- inflammation
- increased susceptibility to respiratory infection

³⁸ Devlin RB, McDonnell WF, Mann R, Becker S, House DE, Schreinemachers D, Koren HS. Exposure of humans to ambient levels of ozone for 6.6 hours causes cellular and biochemical changes in the lung. *Am J Respir Cell Mol Biol* 1991; 4: 72-81.

These respiratory effects were all evident in healthy young adults exposed to 6.6 hour exposures of ozone of 0.08 ppm and higher, while exercising.^{39,40} The fact that a variety of adverse effects were evident in this study population indicates that a standard set at 0.075 ppm will not be adequate to protect against effects in more susceptible populations. (For ethical reasons, children and those with serious lung disease are not selected to participate in human exposure studies.) Standards must be set below the level shown to cause effects in healthy subjects, in order to protect sensitive populations with an adequate margin of safety.

Commenters concur with EPA that chamber studies of adult human volunteers exposed to known concentrations of ozone in a chamber provide powerful evidence to support the setting of standards more stringent than the 2008 standards. Because exposures are to known concentrations of ozone in a laboratory setting, the potential confounding effects other factors such as temperature or other pollutants are eliminated. Additionally, health responses may be precisely measured in the laboratory. Such studies leave no room for debate that adverse effects are occurring at ambient concentrations commonly encountered throughout the U.S.

In addition to the special sensitivity of those with asthma, COPD, and other respiratory diseases, which we will discuss in some detail, several additional factors suggest that the chamber studies justify a more stringent standard:

- First, exposures in these studies were for 6.6 hours, not 8 hours. Ozone harm clearly increases with the cumulative dose. A standard with a longer exposure time than the study period demands a lower level than that shown to induce adverse respiratory effects. In other words, if the study protocol is eliciting adverse effects at 0.08 ppm or 0.06 ppm after 6.6 hour exposures, a standard set for an 8-hour period must somewhat lower than the level at which effects are observed because of the longer averaging time and greater accumulated dose of ozone. This factor was cited by some members of the California Air Quality Advisory Committee in reviewing the draft staff report on revision of the California air quality standards for ozone.⁴¹
- Second, individuals tested in chamber studies are generally healthy, not people with severe respiratory diseases. By law, standards must be set at levels that will protect sensitive subpopulations.
- Third, subjects in controlled exposure studies are adults, not infants or children, who experience greater exposures due to their higher breathing rates.
- Fourth, the full range of human responses cannot be detected in studies with a small number of subjects.

³⁹ Hortstman DH, Follinsbee LJ, Ives PJ, Abdul-Salaam S, McDonnell WF. 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 Rev Respir Dis* 1990; 142: 1158-1163.

⁴⁰ McDonnell WF, Kehrl HR, Abdul-Salaam S, Ives PJ, Follinsbee LJ. Respiratory response of humans exposed to low levels of ozone for 6.6 hours. *Arch Environ Health* 1991; 46: 145-150.

⁴¹ <http://www.arb.ca.gov/research/aaqs/ozone-rs/aqac/pres/aqac-o3.pdf>

With ozone, it is well-established that some people are relatively insensitive, while other individuals—the so-called “responders”—experience enhanced responses. Because of the expense of a clinical chamber study, these studies use a small number of subjects and the inter-subject variability is less than for the general population. For that reason, in evaluating these chamber studies, it is important to recognize that a substantial fraction of subjects in these studies exhibited particularly marked responses in lung function and symptoms. Standards must be set to protect the more sensitive subjects, not just to protect against responses evident in the group mean effects.

For example, the Staff Paper discusses a 1996 study by McDonnell that provides additional evidence of differential responses to ozone.⁴² When combining data from a number of chamber studies of 6.6 hour exposures, the analysis shows that average FEV₁ responses to 0.08 ppm ozone were between 5 and 10 percent; however, 18 percent of exposed subjects had moderate functional decrements of between 10-20 percent; and about 8 percent experienced large decrements, greater than 20 percent.⁴³ Given that only 60 subjects were exposed at this level, it follows that individual responses in the general population would be much more variable, and that some individuals could experience more severe effects that could be clinically significant, as noted by the Staff Paper.

The findings of the earlier human exposure studies are reinforced by a recent 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.⁴⁴

This principle is also relevant to the evaluation of more recent chambers studies of effects of 0.06 ppm ozone, and below. In the last several years, several controlled human exposure studies have been conducted that evaluated the effect on lung function -- forced expiratory volume in one second (FEV₁) -- of various exposure regimes to concentrations of ozone of 0.08 ppm, 0.06 ppm and sometimes 0.04 ppm, for 6.6. hours.^{45,46} These studies by Adams were funded by the American Petroleum Institute and were intended to address the effect of various exposure regimes on lung function responses to ozone.

⁴² U.S. EPA. Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information. OAQPS Staff Paper, July 2007. pp 3-6.

⁴³ McDonnell WF. Individual variability in human lung function responses to ozone exposure. *Environmental Toxicology and Pharmacology* 1996; 2: 171-175.

⁴⁴ Mudway IS, Kelly FJ. An Investigation of Inhaled Ozone Dose and the Magnitude of Airway Inflammation in Healthy Adults. *Am J Respir Crit Care Med* 2004; 169: 1089-1095.

⁴⁵ Adams WC. Comparison of chamber and face-mask 6.6 hour exposures to ozone on pulmonary function and symptoms responses. *Inhalation Toxicol* 2002; 14: 745-764.

⁴⁶ Adams WC. Comparison of chamber 6.6 h exposures to 0.04-0.08 PPM ozone via square-wave and triangular profiles on pulmonary responses. *Inhalation Toxicol* 2006; 18: 127-136.

The Adams (2002) study reports that “some sensitive subjects experience notable effects at 0.06 ppm.” According to the Staff Paper (p. 3-9), this is based on the observation that 20% of the subjects exposed to 0.06 ppm ozone had a greater than 10 percent decrement in FEV₁ 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.

The CD (p. 8-42) indicates that in the Adams (2006) study, even group mean FEV₁ responses during the 0.06 ppm ozone exposures diverge from filtered-air and 0.04 ppm ozone exposures. The EPA Staff Paper presents a comparison of pre- to post- exposure effects using data from the Adams 2006 publication, which indicates a significant effect on FEV₁ of 0.06 ppm ozone compared to filtered air. (SP p. 3-8). This relationship is illustrated in Figure D below.

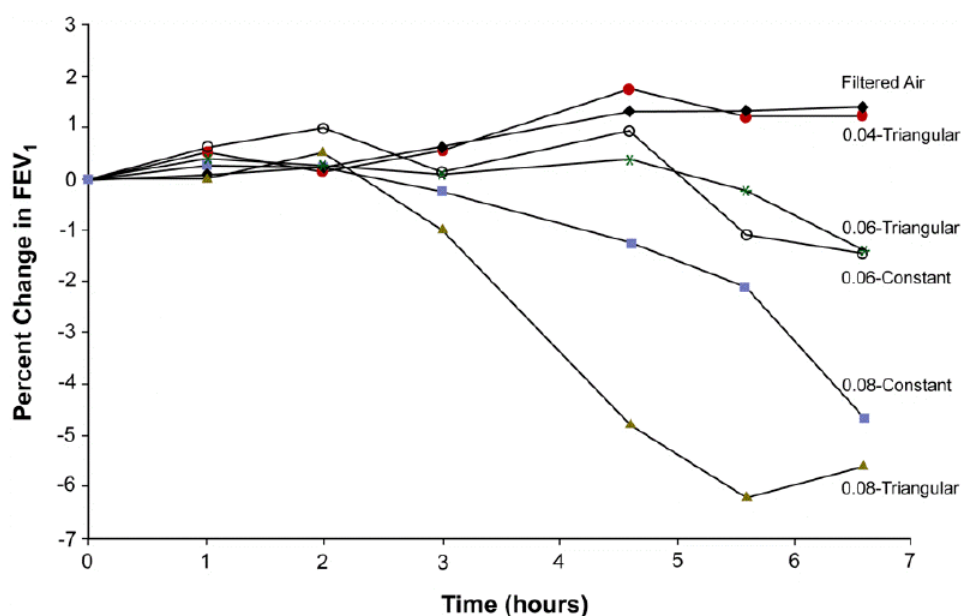


Figure 1. Hour by hour changes in FEV₁ (% change relative to preexposure) adapted from Adams (2006). Data are group mean (error bars were not provided in the published paper) responses of 30 healthy adults exposed to O₃ for 6.6 hours during quasi continuous exercise. The O₃ concentrations were either held constant for the entire 6.6 hour exposure or gradually increased to the lunch hour and then decreased to give a triangular exposure profile of an average concentration noted in the figure.

Figure D: From U.S. EPA Memorandum from James S. Brown, EPA, NCEA-RTP Environmental Media Assessment Group, Thru Mary Ross, EPA, NCEA-RTP, EMAG Branch Chief and Ila Cote, EPA, NCEA-RTP, Director, To Ozone NAAQS Review Docket (OAR-2005-0172), The Effects of Ozone on Lung Function at 0.06 ppm in Healthy Adults, June 14, 2007.

Additionally, the Adams 2006 paper reported that total subjective symptom scores reached statistical significance (relative to pre-exposure) at 5.6 and 6.6 hours, with the triangular exposure scenario. The article states that the pain on deep inspiration values followed a similar

pattern to total subjective symptom scores. The Staff Paper reports that the evaluation of pre- to post-exposure effects on both total subjective symptoms and pain on deep inspiration are suggestive of significant respiratory symptom effects at 0.06 ppm ozone. (SP p. 3-9).

EPA has undertaken a careful reanalysis of the underlying data in the Adams (2002, 2006) studies to assess the change in FEV₁ following exposure to 0.06 ppm ozone and filtered air.⁴⁷ The purpose of the analysis was to note differences in statistical methods between studies, and to analyze FEV₁ responses to low ozone exposure concentrations from the Adams studies in the same manner as the earlier chamber studies conducted by U.S. EPA. The reanalysis addresses criticisms raised to the conclusions presented in the Staff Paper by a consultant to the American Petroleum Institute.

The EPA reanalysis concludes that although appropriate for the design and intent of the Adams studies, the statistical techniques used were overly conservative for the evaluation of pre- to post-exposure changes in FEV₁ between filtered air and ozone exposure. Thus, the reanalysis employs the standard approach used by other researchers, and supported by CASAC.

The reanalysis concludes that the pre- to post-exposure analysis shows that exposure to 0.06 ppm causes a small but statistically significant decrease in group mean FEV₁ responses compared to filtered air, as illustrated in following Figure E.

⁴⁷ U.S. EPA Memorandum from James S. Brown, EPA, NCEA-RTP Environmental Media Assessment Group, Thru Mary Ross, EPA, NCEA-RTP, EMAG Branch Chief and Ila Cote, EPA, NCEA-RTP, Director, To Ozone NAAQS Review Docket (OAR-2005-0172), The Effects of Ozone on Lung Function at 0.06 ppm in Healthy Adults, June 14, 2007.

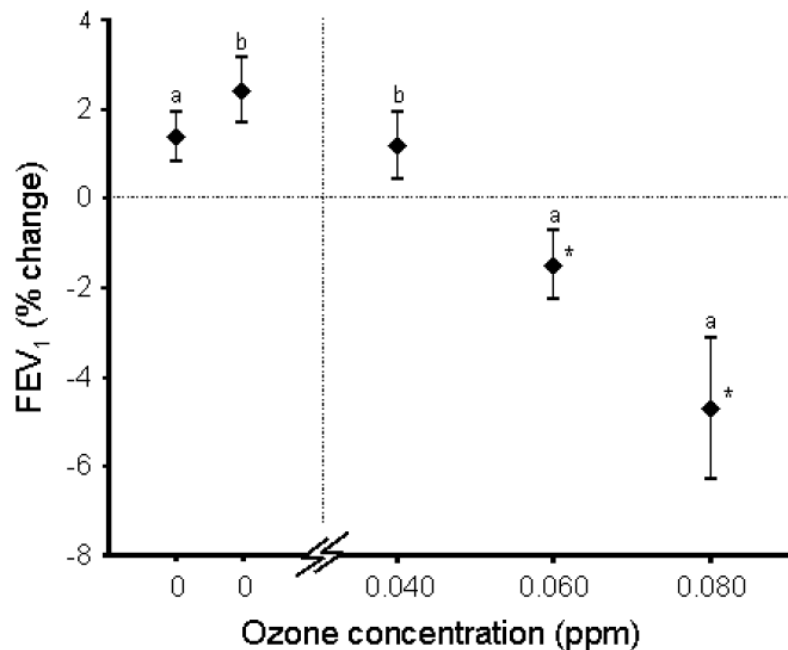


Figure 2. Effects of ozone on FEV₁ in healthy young adults exposed for 6.6 h during quasi continuous exercise to a constant (square-wave) O₃ concentration. Data are from a) Adams (2006) and b) Adams (2002). *Significantly different from responses to air exposure ($p \leq 0.001$, two-tail paired t test).

Figure E: from U.S. EPA Memorandum from James S. Brown, EPA, NCEA-RTP Environmental Media Assessment Group, Thru Mary Ross, EPA, NCEA-RTP, EMAG Branch Chief and Ila Cote, EPA, NCEA-RTP, Director, To Ozone NAAQS Review Docket (OAR-2005-0172), The Effects of Ozone on Lung Function at 0.06 ppm in Healthy Adults, June 14, 2007.

As the Brown memo indicates, while the average response is relatively small, it is important because this is an average response in healthy young adults. The data show considerable variability in lung function responses between similarly exposed subjects, with some individuals experiencing distinctly larger effects (> 10 percent decrements) even when the group mean responses are small.

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 at ozone exposures of 0.04 ppm, 7 percent at 0.06 ppm, and 23 percent at 0.08 ppm as shown in Figure F taken from the EPA Staff Paper (p. 3-7).

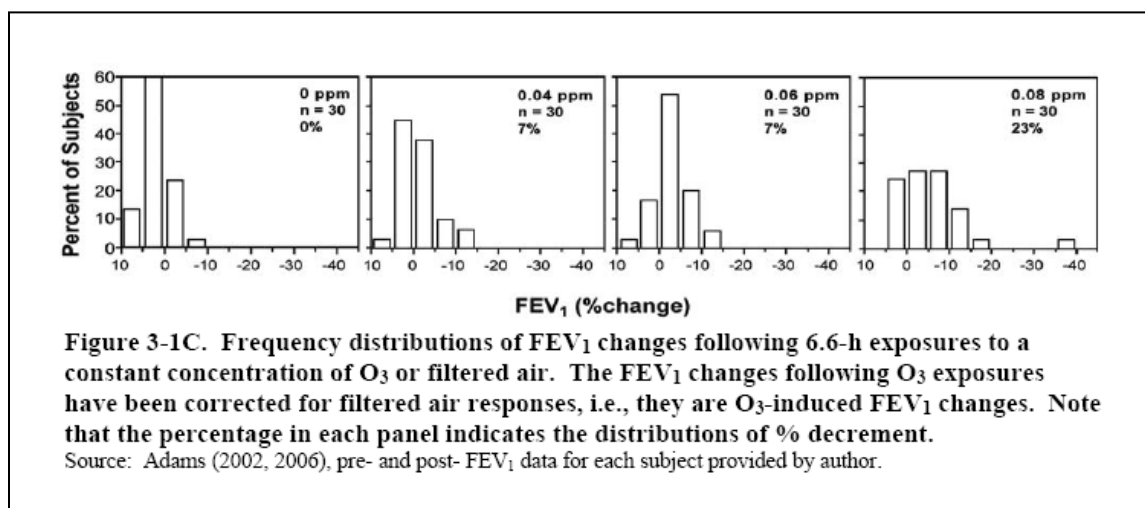


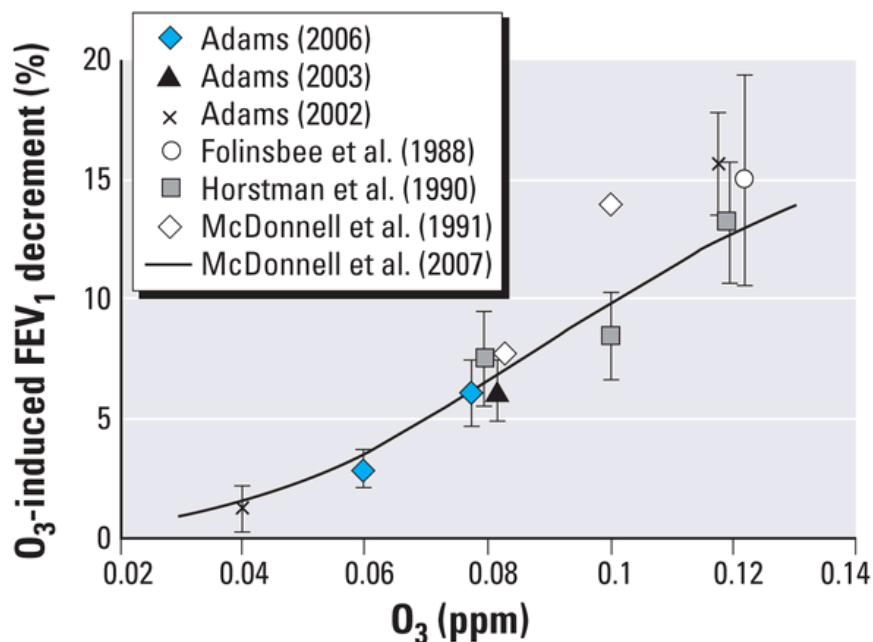
Figure F: from U.S. EPA Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA-452/R-07-003, January 2007.

While only 2 of 30 tested subjects responding at the 0.06 ppm level may seem like a small number, a 7 percent response rate is far from trivial. Consider a population of 300 million Americans. Seven percent is 21 million people.

We concur with the conclusion of the EPA staff reanalysis that larger decrements in FEV₁ would be expected in more susceptible populations.

Brown et al. subsequently published these findings in a peer-reviewed journal. A reanalysis of two clinical studies of ozone conducted by Adams reported that compared to breathing clean air, 60 ppb ozone causes a small statistically significant decline in mean lung function responses of healthy young adults.⁴⁸

⁴⁸ Brown JS, Bateson TF, McDonnell WF. Effects of exposure to 0.06 ppm ozone on FEV₁ in humans: A secondary analysis of existing data. *Env Health Perspect* 2008; 116: 1023-1026.



Cross-study comparison of mean O₃-induced FEV₁ decrements due to 6.6 hr of constant, S-W exposure to varied O₃ concentrations. All exposures were conducted in a chamber, except for a face-mask exposure to 0.04 ppm O₃ in the [Adams \(2002\)](#) study. All studies used a 6.6-hr exposure protocol in which volunteers alternated between 50 min of exercise ($V_E \approx 20$ L/min/m² BSA) and 10 min of rest with an additional 35 min of rest after the third hour. For this exposure protocol, the [McDonnell et al. \(2007\)](#) curve illustrates the predicted FEV₁ decrement at 6.6 hr as a function of O₃ concentration for a 23-year-old. Error bars (where available) are the SE of responses. The data at 0.08 and 0.12 ppm have been offset for illustrative purposes.

Figure G: Brown JS, Bateson TF, McDonnell WF. Effects of exposure to 0.06 ppm ozone on FEV₁ in humans: A secondary analysis of existing data. *Env Health Perspect* 2008; 116: 1023-1026.

A more recent study funded by the American Petroleum Institute investigated the effect of 6.6 hour inhalation of ozone concentrations from 60 to 87 ppb in 31 healthy young adults. This study reported statistically significant effects on respiratory symptoms and pulmonary function in healthy individuals at 70 ppb, below the level of the current 75 ppb standard. The study also found decrements in lung function at 60 ppb, of about 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, confirming that some healthy individuals are more sensitive to ozone than average.⁴⁹

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

⁴⁹ Schelegle ES, Morales CA, Walby WF, Marion S, Allen RP. 6.6 hour inhalation of ozone concentrations from 60 to 87 parts per billion in healthy humans. *Am J Respir Crit Care Med* 2009; 180: 265-272; Brown, JS. Acute effects of exposure to ozone in humans: How low can levels be and still produce effects? *Am J Respir Crit Care Med* 2009; 180: 200-201.

“There are at least three important findings from this study that have public health implications. First, statistically significant changes in FEV₁ and symptoms occurred in healthy individuals at 70 ppb. Second, the magnitude of the mean FEV₁ 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 O₃. Third, consistent with numerous studies, there is considerable intersubject variability in response to O₃. The distribution of response to O₃ becomes skewed with increasing concentration, with a few individuals exhibiting large FEV₁ decrements. Schelegle and colleagues found 16% of individuals to have greater than 10% FEV₁ decrements at 60 ppb, and this proportion increased to 19, 29, and 42% at 70, 80, and 87 ppb, respectively.”⁵⁰

Taken together, the chamber studies provide powerful evidence of the need to lower the 8-hour ozone standard to 60 ppb or below.

Epidemiological Studies Document Effects at Low Concentrations

Epidemiological studies provide further evidence of the need to lower the 8-hour ozone standard to 60 ppb or lower.

The conclusions in the Criteria Document, which were vetted by CASAC, were that the effects of ozone on respiratory symptoms, lung function changes, emergency department visits for respiratory and cardiovascular effects, and hospital admissions can be considered causal at the low concentrations reported in these studies. These effects are well supported by the Hill criteria of judging causality: strength of association, consistency between studies, coherence amongst studies, and biologic plausibility.

It is said that a picture is worth a thousand words. The second draft EPA Staff Paper (p. 3-53) presents a diagram indicating the results of epidemiological studies for associations between short-term ozone exposure and respiratory health outcomes. We copy that figure here for its value in summarizing the array of positive effect estimates and health endpoints observed in multiple locations in Canada and the United States. Figure H summarizes nine studies of various respiratory symptoms including asthma symptoms, wheeze, shortness of breath, medication use, and lower respiratory symptoms; thirteen studies of emergency department visits for respiratory causes including asthma, COPD, pneumonia, and respiratory infection; 21 studies of respiratory hospital admissions, and five studies of mortality from respiratory causes. As Figure F clearly shows, although not all the studies are positive, most are statistically significant.

⁵⁰ Brown JS. Acute Effects of Exposure to Ozone in Humans: How Low Can Levels Be and Still Produce Effects? *Am J Resp Crit Care Med* 2009; 180: 200-201.

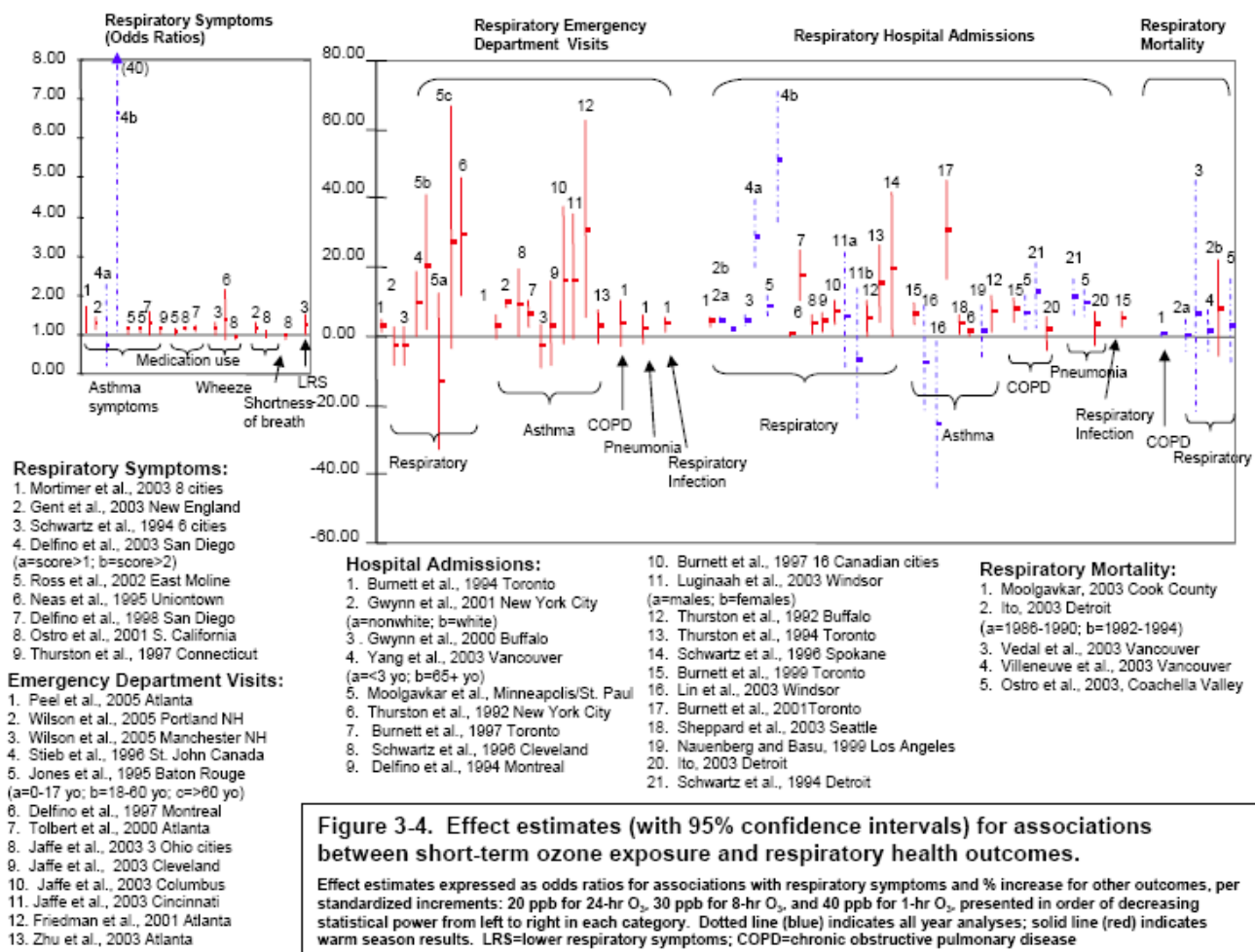


Figure H: From EPA's Second Draft Staff Paper, page 3-53.

In addition to the numerous studies discussed above, a number of other epidemiological and field studies have reported effects of ozone at concentrations less than 0.060 ppm.

Annex 7.1 of the Criteria Document indexes relevant details of epidemiologic studies of human health effects associated with ambient ozone exposure. This annex includes tables of dozens of studies of effects of acute ozone exposure on lung function and respiratory symptoms in field studies, effects of acute ozone exposure on cardiovascular outcomes in field studies, effects of ozone on daily emergency department visits, effects of ozone on daily hospital admissions, effects of acute ozone exposure on mortality, effects of chronic ozone exposure on respiratory health, and effects of chronic ozone exposure on mortality and incidence of cancer. All told, over 250 new epidemiologic studies published from 1996-2005 are included in this table. Our comments highlight just a few of the studies of special interest because they reported effects at

very low concentrations, or they are studies published since the completion of the Criteria Document.⁵¹

The studies discussed in the text of these comments provide statistics drawn from the studies themselves of mean and sometimes maximum ozone concentrations. This information 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. Investigators may make their own ozone measurements, or use publicly available databases of air quality measurements.

Obtaining accurate characterization of exposures is a major issue in carrying out epidemiological studies. Study authors select the most appropriate monitoring data and metrics for their study objectives. These analytical choices are subject to scrutiny during the peer review process, prior to publication in a peer-reviewed journal. Studies that find positive effects after excluding days above a certain concentration are especially pertinent to the setting of air quality standards.

During the NAAQS review process, EPA prepared a memo which described alternate air quality statistics for published studies included in the Criteria Document.⁵² These alternate metrics were an attempt to characterize exposures relative to the way EPA calculates nonattainment and defines nonattainment areas in the regulatory milieu. For example, if study authors had averaged all air quality monitors in a particular county to characterize exposure, the EPA memo reported alternate statistics based on the analysis of all air quality monitors in a metropolitan statistical area.

The EPA memo confuses the issue of the regulatory enforcement of the standards and scientific study of concentrations at which effects are observed. Nonattainment areas for ozone are defined in terms of metropolitan statistical areas in order to develop effective regional control strategies. The original metrics provided by the studies gave the best information about exposure levels and associated responses. These issues must be treated separately in the standard setting process.⁵³

EPA has carried this approach forward and expanded it in the final Staff Paper with the inclusion of Appendix 3B. As EPA states, it is difficult to consistently characterize relevant air quality statistics (SP p. 6-9) and the 98th percentile values are not necessarily equivalent to nonattainment “design values.”

Despite these concerns, useful information can still be gleaned from EPA’s analysis. Table 2 below, drawn from Appendix 3B of the Staff Paper, arrays a dozen North American studies which reported positive, statistically significant results for various health endpoints, for which

⁵¹ U.S. EPA, Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600/R-05/004bF, February 2006.

⁵² McCluney L, Rizzo M, Ross R. 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), August 23, 2006.

⁵³ McCluney L, Rizzo M, Ross R. 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), August 23, 2006.

EPA derived 98th percentile 8-hour daily maximum concentrations of about 70 ppb or lower.⁵⁴ The data demonstrate that even after taking a broader view of the air quality statistics than the study authors, and after looking at different air quality metrics, adverse health effects are observed at concentrations at and well below the current standards.

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 3 provide additional evidence for a standard of 0.060 ppm.

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. Furthermore, the Criteria Document and Staff Paper include discussion of numerous additional epidemiological studies that are positive, though not statistically significant, which add weight to the overall findings of effects that are evident at low concentrations.

EPA's Appendix 3B Table, Ozone Epidemiological Study Results, also reports the effect estimate and lower and upper confidence intervals for each health endpoint in the studies. Figure F above graphically shows the width of the confidence intervals across a range of U.S. and Canadian studies.

The width of the confidence interval can be a function of the sample size. For some studies and health endpoints with low mean and 98th percentile concentrations, there are small confidence intervals indicating lesser uncertainty. The width of the confidence interval is not necessarily a function of the concentration. In this universe of studies, there are both wide and narrow confidence intervals across a range of concentrations. This indicates that statistical uncertainty is not always greater in studies performed at lower concentrations. EPA has not done a systematic analysis to support its claim that the confidence intervals and related uncertainty are always wider at lower concentrations.

Study Endpoints	98 th 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
Brauer et al., 1996	55

⁵⁴ Results may not be statistically significant for all endpoints examined.

Study Endpoints	98 th percentile 8-hr daily max (ppb)
Emergency Department Visits: Respiratory Diseases	
Delfino et al., 1997	57.5
Hospital Admissions: Cardiovascular Diseases	
Koken et al., 2003	64.5
Hospital Admissions: Respiratory Diseases	
Delfino et al., 1994	69
Burnett et al., 1997	62
Yang et al., 2003	42.7
Burnett et al., 1999	68.4
Mortality:	
Vedal et al., 2003	53.3

**Table 2: Ozone Epidemiological Studies Showing Effects at Low Concentrations:
EPA Derived 98th Percentile Statistics Near or Below the Current Standard⁵⁵**

American Lung Association, 2010, 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.

With respect to ozone and short-term mortality, which we discuss in a separate section, the CD's conclusion is overly conservative. Sufficient evidence exists to consider the effect as causal. The late Dr. David Bates addressed the plausibility of low concentrations causing premature mortality in his comments on three meta-analyses of ozone and daily mortality:

“The 3 new meta-analyses ... along with the recent European study, each have unique features and appear to resolve the question of whether ambient ozone levels are associated with increased mortality. It seems unlikely that PM_{2.5} is an important confounder, and the effect of ozone appears to be independent of temperature. A final question — that of biologic plausibility — is in some ways the easiest to answer. Ozone is capable of causing inflammation in the lung at lower concentrations than any other gas. Such an effect would be a hazard to

⁵⁵ 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.

anyone with heart failure and pulmonary congestion, and would worsen the function of anyone with advanced lung disease.”⁵⁶

Indeed, a National Academy of Sciences study concluded in 2008 that the health-based evidence demonstrates that short-term exposure to ambient ozone is likely to contribute to premature deaths and recommends that ozone-related mortality be included in future estimates of the health benefits of reducing ozone exposure.⁵⁷

Additional Epidemiological Studies Show Need for 0.060 ppm Standard

Many additional studies document evidence of harm at levels well below both the existing standard and the proposed standard. Not surprisingly, most provided 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. They provide powerful evidence of effects of low level exposures to ozone in the real world that compel adoption of a final 8-hour average standard of 0.060 ppm.

Studies of Outdoor Workers and Exercisers

A recent study by Chan and Wu reported acute lung function decline in mail carriers exposed to ozone concentrations below the current ambient air quality standard.⁵⁸ The 8-hour average concentration of ozone in this study was 36 ± 12 ppb (mean $\pm$ SD), and the maximum 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% for a 0-day lag, 0.69% for a 1-day lag, and 0.52% for a 2-day lag. 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 PEF_R 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 PEF_R 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 FEV_{1.0} and FVC,

⁵⁶ Bates DV. Ambient Ozone and Mortality. *Epidemiology* 2005; 16: 427-429.

⁵⁷ National Research Council. 2008. Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution. Committee on Estimating Mortality Risk Reduction Benefits from Decreasing Tropospheric Ozone Exposure, National Research Council.
<http://www.nap.edu/catalog/12198.html>

⁵⁸ Chan C-C, Wu T-H. Effects of Ambient Ozone Exposure on Mail Carriers' Peak Expiratory Flow Rates. *Environ Health Perspect* 2005; 113: 735-738.

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. An important such study of the effect ozone exposure on lung function of outdoor farm workers was undertaken in the Fraser Valley of British Columbia. The mean work shift concentrations were low, just 26 ppb, with a maximum of 54 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 concentrations below 85 ppb 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.⁵⁹ The Staff Paper (p. 6-12) appears to dismiss the significance of this study by claiming that the exposure patterns of the outdoor workers would not be typical of the general population. 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.

Another study examined effects of ozone on a cohort of healthy young men who exercise outdoors -- in this case, a group of amateur cyclists in Netherlands. 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 43 ppb. 8-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, in relation to ozone exposure. The effect persisted, even after removing all observations with hourly ozone greater than 60 ppb. Studies like this provide vital evidence of the need for a 0.060 ppm standard.⁶⁰

In a study of hikers at Mount Washington in New Hampshire, researchers evaluated the effects of acute ozone, PM_{2.5}, and strong aerosol acidity on the pulmonary function of exercising adults. The mean 8-hour ozone concentration in this study was 0.04 ppm, and the maximum was 0.074 ppm. 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.⁶¹ A standard of 0.060 ppm is needed to protect hikers and others who exercise outdoors.

⁵⁹ Brauer M, Blair J, Vedal S. Effect of Ambient Ozone Exposure on Lung Function in Farm Workers. *Am J Respir Crit Care Med* 1996; 154: 981-987.

⁶⁰ Brunekreef B, Hoek G, Breugelmans O, Leentvaar M. Respiratory Effects of Low-level Photochemical Air Pollution in Amateur Cyclists. *Am J Respir Crit Care Med* 1994; 150: 962-966.

⁶¹ Korrick SA, Neas LM, Dockery DW, Gold DR, Allen GA, Hill LB, Kimball KD, Rosner BA, Speizer FE. Effects of Ozone and Other Pollutants on the Pulmonary Function of Adult Hikers. *Env Health Perspect* 1998; 106: 93-99.

A study of effects of ozone in ambient air on respiratory function in healthy adult nonsmokers engaged in a daily outdoor exercise program was undertaken in Tuxedo, New York in the summer of 1985. The authors concluded that ambient cofactors can potentiate the responses to ozone and that the results of chamber studies may underestimate responses to ozone.

“Our data indicate that respiratory function responses to inhaled O₃ occur at concentrations below 80 ppb. This is consistent with the results of our study of children at a summer camp that indicated significant effects, even with data sets limited to values below 80 and 60 ppb. The data are also consistent with the results of a study by Kinney and colleagues of school children in Kingston and Harriman, Tennessee whose lung function was measured in school on up to six occasions during a 2-month period in the late winter and early spring...Since the highest O₃ concentration in the study by Kinney and colleagues was 78 ppb, the threshold for responses to O₃ in ambient air for adults and children engaged in normal activities appear to be well below 80 ppb.”⁶²

Another study 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. 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 these adverse effects at concentrations of 0.06 ppm and below.⁶³

Studies of Infants, Children and Seniors

Recent 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.

An important study examined respiratory effects of ozone in 700 infants living in nonsmoking households in southwestern Virginia. The authors concluded: “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.” In this study there were no days when the 1-hour standard was exceeded, and only two days when the 8-hour ozone standard was exceeded. As shown in Table 3 and Figure I below, the mean 8-hour maximum ozone concentration was 54.5 ppb, with a standard deviation ± 13.0 .⁶⁴

⁶² Spektor DM, Lippmann M, Thurston GD, Liroy PJ, Stecko J, O’Connor G, Garshick, E, Speizer FE, Hayes C. Effects of Ambient Ozone on Respiratory Function in Healthy Adults Exercising Outdoors. *Am Rev Respir Dis* 1988; 138: 821-828.

⁶³ Kinney PL, Nilsen DM, Lippmann M, Brescia M, Gordon T, McGovern T, Fawal HE, Devlin RB, Rom WN. Biomarkers of lung inflammation in recreational joggers exposed to ozone. *Am J Respir Crit Care Med* 1996; 154: 1430–1435.

⁶⁴ Triche EW, Gent JF, Holford TR, Belanger K, Bracken MB, Beckett WS, Naeher L, McSharry J-E, Leaderer BP. Low-Level Ozone Exposure and Respiratory Symptoms in Infants. *Environ Health Perspect* 2006; 114: 911-916.

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

Pollutant	Mean $\pm$ 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
$PM_{2.5}$ ($\mu g/m^3$)	23.2 ± 10.3	22.3	3.5–59.6	15.7–29.4	13.7
Coarse ($\mu g/m^3$)	6.2 ± 3.2	5.9	0.0–19.8	4.2–7.8	3.6

Table 3: from Triche et al. 2006.

In Figure 1 of the article by Triche et al. [Environ Health Perspect 114:911–916 (2006)], the 24-hr average and the 8-hr maximum average were labeled incorrectly. The corrected figure appears below:

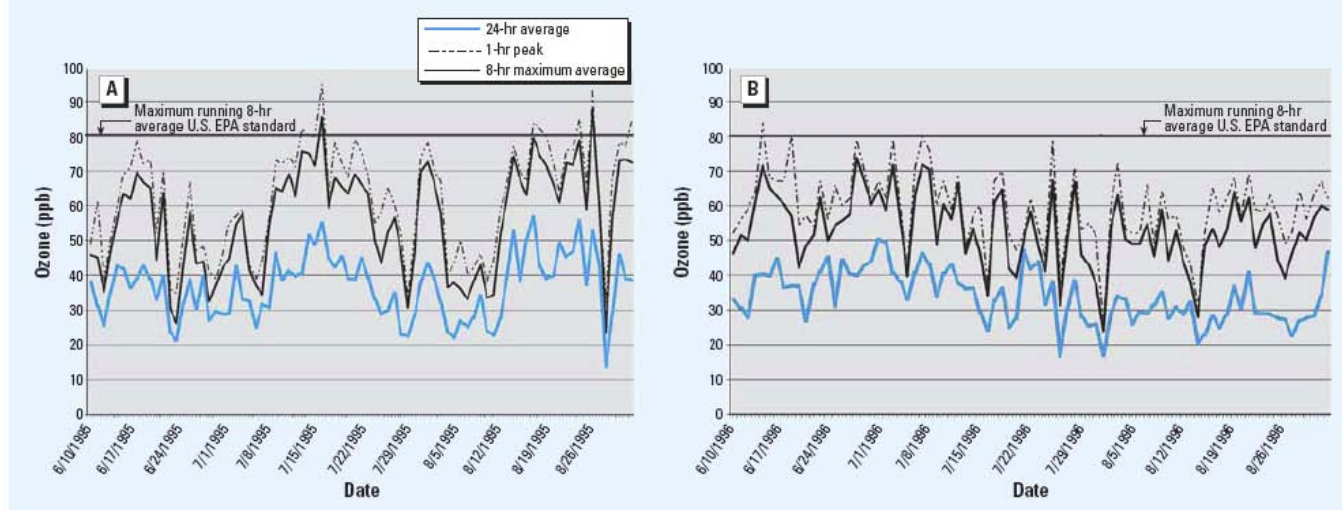


Figure I: from Triche et al. 2006.

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.⁶⁵ 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 Table 4, 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 at the bottom of the CASAC recommended range, or below.

⁶⁵ Dales RE, Cakmak S, Doiron MS. Gaseous Air Pollutants and Hospitalization for Respiratory Disease in the Neonatal Period. *Environ Health Perspect* 2006; 114: 1751–1754.

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

Table 4: from Dales et al., 2006.

Although hospital admissions for respiratory disease are relatively uncommon in newborns compared with adults, this study found a significant association with gaseous air pollutants. In fact, if the association was proven to be causal, air pollution at ambient levels seen in Canada could account for 15 percent of hospital admissions in neonates. The two strongest effects were with NO₂ and O₃.

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 for copollutants and socioeconomic status. The 24-hour average ozone concentrations in this study were very low, at 13.41 ppb.⁶⁶

New Evidence of Increased Sensitivity of People with Asthma

New studies provide extensive further evidence that people with respiratory disease are at increased risk, above that faced by the general population. In addition, substantial new toxicological evidence provides plausible biological mechanisms for the adverse impacts of ozone observed in epidemiological studies.

New evidence since the 1996 review correlates exposure to ozone with respiratory symptoms, increased airway responsiveness, school absenteeism and increased medication use in people with asthma.

In 2003, Höppe et al. documented large differences in the sensitivity of individuals to ozone. Those that are particularly sensitive are known as “responders.” A recent 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. The study found higher rates of ozone responders in asthmatics (21%) and children (18%), as compared to the elderly and athletes (both 5%). This means that children and asthmatics have a higher risk of being ozone sensitive and experiencing more acute lung function decrements than

⁶⁶ Yang Q, Chen Y, Shi Y, Burnett RT, McGrail KM, Krewski D. Association between ozone and respiratory admissions among children and the elderly in Vancouver, Canada. *Inhal Toxicol* 2003; 15: 1297-1308.

these other population groups.⁶⁷ This reinforces the findings of an earlier study, where, Höppe et al. reported that pulmonary decrements of juvenile asthmatics on high ozone days, with daily average concentrations of 0.070 ppm, were larger than those documented for healthy children.⁶⁸ These studies indicate 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 also comes from two studies by Mortimer et al. The effect of daily ambient air pollution was 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 DC. 8-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 (see Figure G below).⁶⁹ 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 0.060 ppm 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. 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 Figure J. 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."⁷⁰

⁶⁷ Höppe P, Peters A, Rabe G, Praml G, Lindner J, Jakobi G, Fruhmann G, Nowak D. Environmental Ozone Effects in Different Population Subgroups. *Int J Hyg Environ Health* 2003; 206: 505-516.

⁶⁸ Höppe P, Praml G, Lindner J, Fruhmann G, Kessel R. Environmental ozone field study on pulmonary and subjective responses of assumed risk groups. *Environ Res* 1995; 71: 109-121.

⁶⁹ Mortimer, KM, Neas LM, Dockery DW, Redline S, Tager IB. The effect of air pollution on inner-city children with asthma. *Eur Respir J* 2002; 19: 699-705.

⁷⁰ Mortimer KM, Tager IB, Dockery DW, Neas LM, Redline S. The Effect of Ozone on Inner-City Children with Asthma: Identification of Susceptible Subgroups. *Am J Respir Crit Care Med* 2000; 162: 1838-1845.

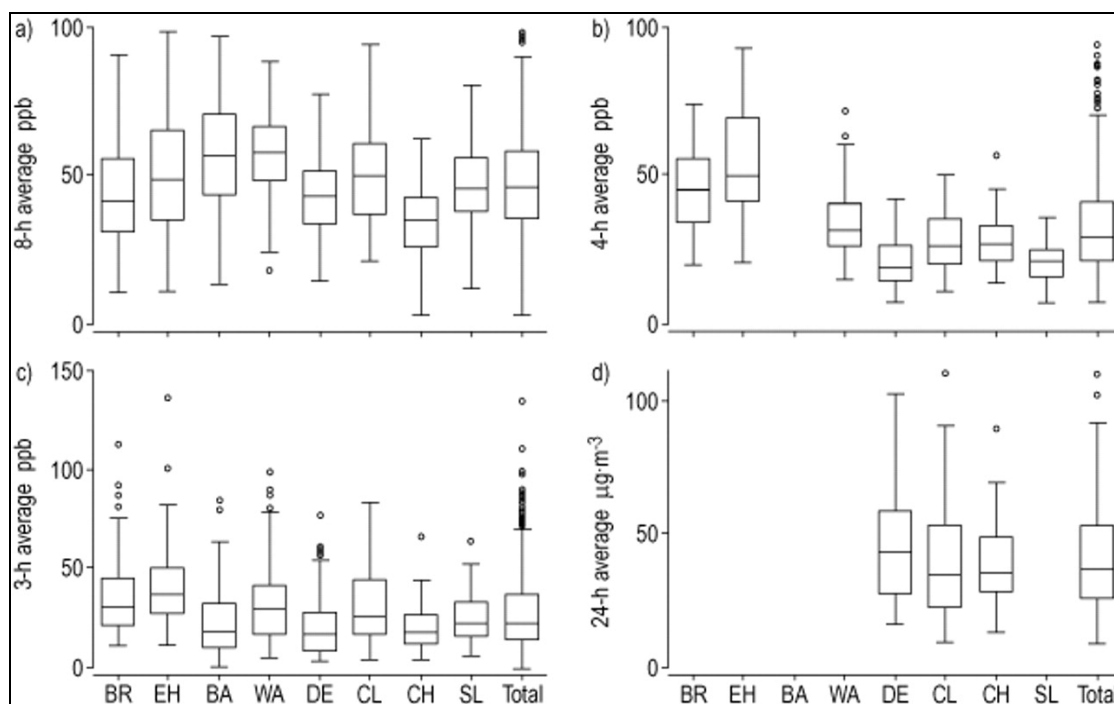


Figure J: from Mortimer et al., 2000.

Additional evidence of the increased sensitivity of asthmatic children is provided by the study of 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. 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 Table 5, mean 8-hour ozone concentrations in this study were 51.3 ppb, with a standard deviation of 15.5.⁷¹

⁷¹ Gent JF, Triche EW, Holford TR, Belanger K, Bracken MB, Beckett WS, Leaderer BP. Association of Low-Level Ozone and Fine Particles with Respiratory Symptoms in Children with Asthma. *JAMA* 2003; 290: 1859-1867.

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

	Mean (SD)	Range	Percentile				
			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
$\text{PM}_{2.5}$, 24-hour total, $\mu\text{g}/\text{m}^3$	13.1 (7.9)	3.7-44.2	6.9	9.0	10.3	12.1	19.0
Temperature, 24-hour maximum, $^{\circ}\text{C}$	23.5 (6.0)	4.89-36.2	17.6	23.7	25.0	26.1	28.4

Table 5: from Gent et al., 2003.

Asthmatics who already experience increased airway reactivity and inflammation may find their symptoms worsened or prolonged by exposure to ozone. In a study comparing airway inflammation and responsiveness to ozone in normal and asthmatic subjects, Balmes et al. reported that the ozone-induced increases in percentage of neutrophils and total protein concentration in bronchoalveolar lavage fluid were significantly greater for the asthmatic subjects than for the non-asthmatic subjects. These data suggest that the inflammatory response of the asthmatic lung may be more intense, indicating the need for tighter standards than proposed in order to protect the health of asthmatics.⁷²

A 2007 study used a passive ozone sampler to investigate the effects of personal ozone exposures on the pulmonary function and symptoms of 20 moderate to severe asthmatics. While there was no correlation with peak expiratory flow, the degree of asthma symptoms was influenced by the ozone level, even at concentrations less than 80 ppb. The average ozone exposure level in this study was 28.2 ppb. According to the authors, the results suggest that asthma symptoms are provoked or aggravated, even at ozone concentrations below 80 ppb in patients with moderate to severe persistent asthma, providing further evidence for a standard well below this concentration.⁷³

The reduction in traffic congestion in Atlanta during the summer Olympic Games resulted in a decline in peak daily (1-hour) ozone pollution from 83.1 to 53.6 ppb that was associated with reduced acute asthmatic events in children. Researchers concluded: "Our results ... indicate that reductions in ozone and PM_{10} pollution at levels considerably below EPA's National Ambient Air Quality Standards can reduce asthma morbidity in children." This intervention study suggests that ozone reductions will provide concrete public health benefits to children.⁷⁴

⁷² Balmes JR, Aris RM, Chen, LL, Scannell C, Tager IB, Finkbeiner W, Christian D, Kelly T, Hearne PQ, Ferrando R, Welch B. Effects of ozone on normal and potentially sensitive human subjects. Part I: Airway inflammation and responsiveness to ozone in normal and asthmatic subjects. *Res Rep Health Eff Inst.* 1997; 78: 1-37.

⁷³ Kim DH, Kim YS, Park JS, Kwon HJ, Lee KY, Lee S-R, Jee YK. The Effects of On-site Measured Ozone Concentration on Pulmonary Function and Symptoms of Asthmatics. *J Korean Med Sci* 2007; 222: 30-36.

⁷⁴ Friedman MS, Powell KE, Hutwagner L, Graham LM, Teague WG. Impact of changes in transportation and commuting behaviors during the 1996 summer Olympic Games in Atlanta on air quality and childhood asthma. *JAMA* 2001; 285: 897-905.

A new European study illustrates that ozone exerts a profound influence on patients with persistent asthma. A study of patients with persistent asthma who were taking maintenance medications concluded that these patients were more vulnerable to ozone, and that increased ozone levels resulted in sharp increases in coughing in children with persistent asthma. This study found that repeated exposure to ozone at peak ambient air levels (4 x 125 ppb) can enhance both the functional and inflammatory responses in inhaled allergen in subjects with preexisting allergic airway diseases, and that these effects might reach a clinically relevant magnitude.⁷⁵

New evidence of the special sensitivity of those with respiratory disease is also provided by epidemiological studies correlating increases in ozone with emergency department visits and hospital admissions for asthma and other respiratory diseases.

In a study funded by the Electric Power Research Institute, Tolbert et al. examined pediatric emergency room visits for asthma in relation to air quality. As shown in Table 6, mean 8-hour ozone concentrations in this study were 59.3 ppb. 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 data suggested an exposure-response trend, with the risk ratios consistently elevated for 70-79 ppb, and above. The authors conclude that both ozone and PM₁₀ 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 0.060 ppm.⁷⁶

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

* $p < 0.05$.

† PM₁₀, particulate matter $\leq 10 \mu\text{m}$ in aerodynamic diameter; NO_x, total oxides of nitrogen.

‡ Numbers in parentheses, standard deviation.

Table 6: from Tolbert et al., 2000.

⁷⁵ Holz O, Mucke M, Pashach K, Bohme S, Timm P, Richter K, Magnussen H, Jorres RA. Repeated ozone exposures enhance bronchial allergen responses in subjects with rhinitis or asthma. *Clin Exp Allergy* 2002; 32: 681-689.

⁷⁶ Tolbert PE, Mulholland JA, MacIntosh DL, Xu F, Daniels D, Devine OJ, Carlin BP, Klein M, Dorley J, Butler AJ, Nordernberg DF, Frumkin H, Ryan PB, White MC. Air Quality and Pediatric Emergency Room Visits for Asthma in Atlanta, Georgia. *Am J Epidemiol* 2000; 151: 798-810.

In a larger study of respiratory emergency department visits to 31 hospitals in Atlanta, visits for asthma, COPD, upper respiratory infection, and pneumonia were assessed in relation to air pollutants. Ozone was associated with visits for all respiratory disease, and for upper respiratory infection in particular, and this association persisted in multipollutant models. Again, effects are evident well below the current standard. During warm months a 25 ppb increase in ozone was associated with a 2.6 percent increase in pediatric asthma visits to the emergency room. As indicated in Table 7, mean 8-hour ozone concentrations in this study were 55.6 ppb, and the 90th percentile concentration was 87.6 ppb.⁷⁷

A study just out in 2010 reports examined the association between air pollution and people with asthma in the San Joaquin Valley, California. The study found that ozone, PM₁₀ and PM_{2.5} were associated with frequent asthma symptoms and asthma-related emergency department visits or hospitalization, while controlling for socioeconomic factors. The median annual average ozone concentration in this highly polluted region was 30.3 ppb.⁷⁸

TABLE 1. Mean, Standard Deviation, and Selected Percentiles of Daily Ambient Air Quality Measurements for 5 Criteria Pollutants From the AQS and for Pollutants From the ARIES Monitoring Station

	% Missing	Mean ± SD	10%	90%
24-h PM ₁₀ (μg/m ³)*‡	3	27.9 ± 12.3	13.2	44.7
8-h Ozone (ppb)*‡§	32	55.6 ± 23.8	26.8	87.6
1-h NO ₂ (ppb)*‡	1	45.9 ± 17.3	25.0	68.0
1-h CO (ppm)*‡	2	1.8 ± 1.2	0.5	3.4
1-h SO ₂ (ppb)*‡	1	16.5 ± 17.1	2.0	39.0
24-h PM _{2.5} (μg/m ³)†	2	19.2 ± 8.9	8.9	32.3
24-h coarse PM (μg/m ³)†	11	9.7 ± 4.7	4.4	16.2
24-h 10–100 nm particle count (#/cm ³)†	44	38000 ± 40700	11500	74600
24-h PM _{2.5} water-soluble metals (μg/m ³)†	9	0.028 ± 0.025	0.006	0.061
24-h PM _{2.5} sulfate (μg/m ³)†	10	5.5 ± 3.7	1.9	10.7
24-h PM _{2.5} acidity (μ · equ/m ³)†	15	0.018 ± 0.023	–0.001	0.045
24-h PM _{2.5} organic carbon (μg/m ³)†	6	4.5 ± 2.2	2.2	7.1
24-h PM _{2.5} elemental carbon (μg/m ³)†	6	2.0 ± 1.4	0.8	3.7
24-h oxygenated hydrocarbons (ppb)†	22	32.1 ± 15.3	15.0	53.1
Average temperature (°C)	0	17.5 ± 8.3	6.1	27.2
Average dew point (°C)	0	10.5 ± 8.9	–2.2	20.8

*Measurements available from AQS from 1 January 1993 to 31 August 2000.

†Measurements available from the ARIES monitoring station from 1 August 1998 to 31 August 2000.

‡Data were imputed for 17% (458 of 2703) of PM₁₀ values, 2% (46 of 1892) of ozone values, 14% (398 of 2775) of NO₂ values, 6% (161 of 2758) of CO values, and 9% (237 of 2775) of SO₂ values.

§Ozone was measured for 1896 days: 1 March 1993 to 30 November 1993, 1 March 1994 to 30 November 1994, 1 March 1995 to 30 November 1995, 1 March 1996 to 31 October 1996, 1 April 1997 to 31 October 1997, 1 April 1998 to 31 October 1998, 1 April 1999 to 31 October 1999, 1 March 2000 to 31 August 2000.

||Acidity reported in units of μ · equ/m³, a measure of pH level, accounting for the negative values. If converted into units of nmol/m³, the mean is 18 and standard deviation is 23.

PPB, parts per billion; PPM, parts per million

Table 7: from Peel et al., 2005.

⁷⁷ Peel JL, Tolbert PE, Klein M, Metzger KB, Flanders WD, Todd K, Mulholland JA, Ryan PB and Frumkin H. Ambient Air Pollution and Respiratory Emergency Department Visits. *Epidemiology* 2005; 16: 164-174.

⁷⁸ Meng Y-Y, Rull RP, Wilhelm M, Lombardi C, Balmes J, Ritz B. Outdoor air pollution and uncontrolled asthma in the San Joaquin Valley, California. *J Epidemiol & Comm Health* 2010; 64: 142-147. doi: 10.1136/jech.2009.083576,

Similarly, a study in New England reported that ozone increases 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).⁷⁹

A 2007 study reports 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 emergency admissions for asthma exacerbations. The 8-hour daily maximum ozone concentrations reached Code Red levels on only five days during the study period.⁸⁰

The evidence is overwhelming in demonstrating the correlation between high ozone days and hospital admissions for asthma. Silverman and Ito (2010), for example, demonstrate a 19% 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.⁸¹

⁷⁹ Wilson AM, Wake CP, Kelly T, Salloway JC. Air Pollution, Weather, and Respiratory Emergency Room Visits in Two Northern New England Cities: an Ecological Time-Series Study. *Environ Res* 2005; 97: 312 - 321.

⁸⁰ Babin SM, Burkom HS, Holtry RS, Taberner NR, Stokes LD, Davies-Cole JO, DeHaan K, Lee DH. 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* 2007; 21: 6-9.

⁸¹ Silverman RA, Ito K. Age-related association of fine particles and ozone with severe acute asthma in New York City. *J Allergy Clin Immun* 2010; 125: 367-373.

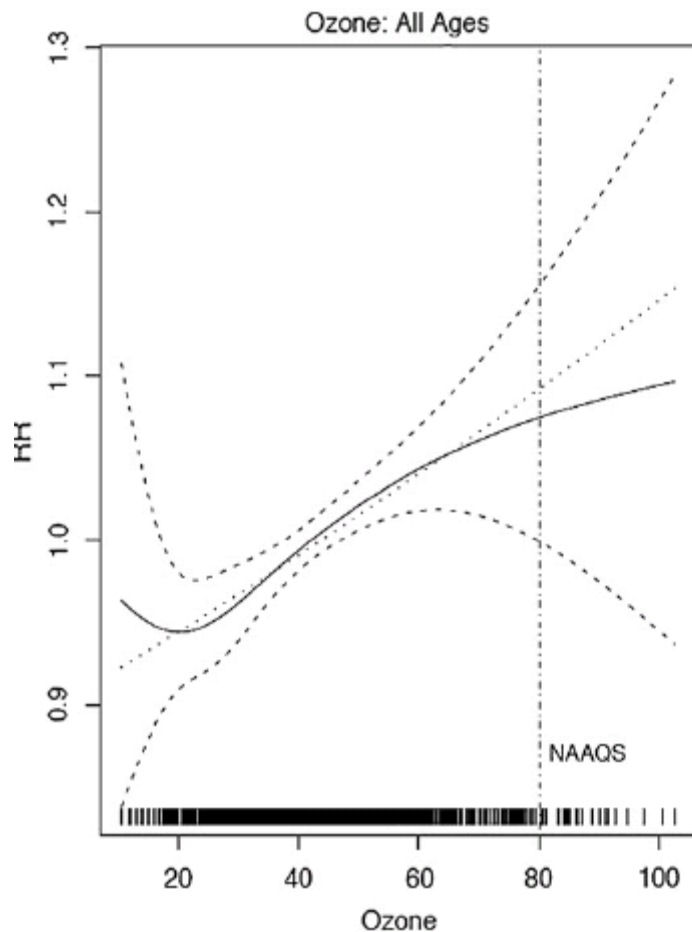


FIG 3. Estimated relative risks (*RRs*) of asthma hospitalization allowing for possible nonlinear relationship using natural splines. The average of 0-day and 1-day lagged $PM_{2.5}$ and ozone were used in a 2-pollutant model, adjusting for temporal trends, day of the week, and immediate and delayed weather effects. The *solid lines* are smoothed fit data, with *long broken lines* indicating 95% confidence bands. The *short broken lines* are linear fitted lines. The *vertical dotted lines* are the current NAAQS. The density of lines at the bottom of the figure indicates sample size.

Figure K: Source: Silverman et al 2010.

New Evidence of Harm to People with COPD

New studies also show that people with chronic obstructive pulmonary disease (COPD) and other diseases are especially impacted by ozone.

A recent very large case-crossover study of Medicare recipients in of 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 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 Table 8 below, concentrations in most cities in the 40-55 ppb range.⁸² This study provides powerful evidence for a standard of 0.060 ppm 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 (µg/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; PM₁₀, particulate matter with an aerodynamic diameter of ≤10 µm; COPD, chronic obstructive pulmonary disease.

Table 8: From Medina-Ramón, et al., 2006.

⁸² Medina-Ramón M, Zanobetti A, Schwartz J. The Effect of Ozone and PM₁₀ on Hospital Admissions for Pneumonia and Chronic Obstructive Pulmonary Disease: A National Multicity Study. *American Journal of Epidemiology* 2006; 163: 579-588.

Another recent study using the APHEA approach 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 evidence of the special susceptibility of people with COPD to ozone.⁸³

A study in Taipei, Taiwan also reported positive associations between ozone and hospital admissions for COPD in single- and two-pollutant models. Mean ozone concentrations were 20.52 ppb, and maximum ozone concentrations were 62.79 ppb in this study.⁸⁴

A French study reported that ozone exacerbates symptoms in COPD patients. Thirty-nine senior adults with severe chronic obstructive pulmonary disease (COPD) were followed by their physicians in Paris, France, during a 14-month period. Daily levels of PM₁₀, ozone, sulfur dioxide and nitrogen dioxide were monitored. No evidence of symptom exacerbation and PM₁₀, SO₂, or NO₂ was observed. However, the 8-hour average ozone concentration was associated with exacerbation of COPD symptoms. According to the researchers, "our results are consistent with those of toxicological studies that have shown the inflammatory mechanisms of O₃. 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."⁸⁵

According to the 2005 survey by the National Center for Health Statistics, roughly 32.6 million Americans have been diagnosed with asthma at some point in their lives. Some 12.3 million of them are children under age 18 and another 3.4 million are over 65.⁸⁶ This is a substantial segment of the overall population that is not adequately protected by the current air quality standards.

In summary, commenters concur with EPA that the new data on the sensitivity of asthmatics and people with allergic rhinitis to ozone indicate that the clinical studies that evaluate only healthy subjects will underestimate the effects of ozone on asthmatics and other susceptible groups, and provides convincing evidence of the need to lower the standards substantially in order to protect the health of these groups. Some 1,700 new studies have been considered in this latest review. The mounting evidence of the sensitivity of people with respiratory disease to react to lower concentrations of ozone than the general population, combined with new information about effects at low concentrations, discussed above and below, compels EPA to establish an 8-hour average ozone standard at 0.060 ppm.

⁸³ Ko FWS, Tam W, Chan DPS, Wong TW, Tung AH, Lai CKW, Hui DSC. The temporal relationship between air pollutants and hospital admissions for chronic obstructive pulmonary disease in Hong Kong. *Thorax*, Published Online First: 20 February 2007. doi:10.1136/thx.2006.076166.

⁸⁴ Yang CY, Chen CJ. Air pollution and hospital admissions for chronic obstructive pulmonary disease in a subtropical city: Taipei, Taiwan. *J Toxicol Environ Health A* 2007; 70: 1214-1219.

⁸⁵ Desqueyroux, H., Pujet, J.C., Prosper, M., Le Moulec, Y., Momas, I. Effects of Air Pollution on Adults With Chronic Obstructive Pulmonary Disease. *Archives of Environmental Health* 2002; 57: 554-560.

⁸⁶ American Lung Association. *Trends in Asthma a Morbidity and Mortality*. August 2007.

Effects in Healthy Women

Naeher et al. studied the relationship between ambient air pollution and daily change in peak expiratory flow in a sample of 473 nonsmoking women in Roanoke, Virginia over the summers of 1995-1996. A 30 ppb increment in 24-hour average ozone was associated with a decrease of 2.49 L/min in evening peak expiratory flow (PEF). A 5-day cumulative lag exposure showed the greatest effect of ozone, 7.65 L/min decrease per 30 ppb ozone increase. According to the authors, these results are consistent earlier studies. Notably, ozone concentrations in this study were well below the current 8-hour ozone standard. The mean daily maximum 8-hour ozone concentration was 53.69 ppb, and the maximum was 87.63. As illustrated by Figure L, ozone concentrations were generally well below the level of the 8-hour average standard, providing critical support for a standard at the low end of the range recommended by CASAC and EPA Staff Scientists.⁸⁷

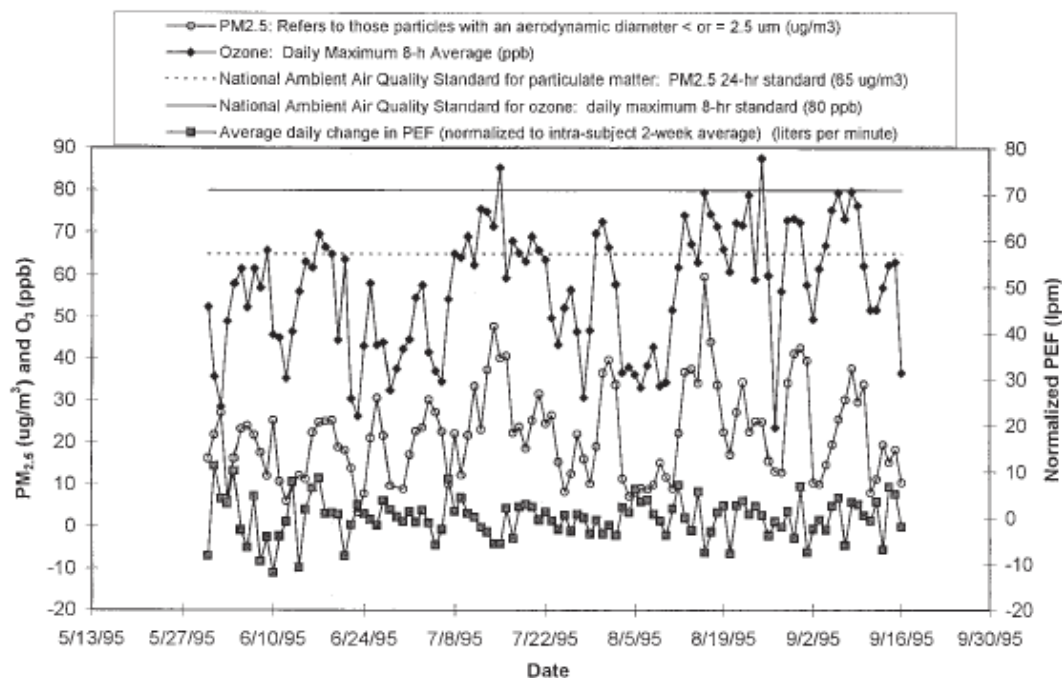


Figure 1. 24-h average PM_{2.5} and daily maximal 8-h average O₃ at the Vinton stationary ambient monitoring site, and daily variations in morning PEF: summer 1995.

Figure L: From Naeher et al., 1999.

Additional Evidence from International Studies

We disagree that U.S. and Canadian studies are the only studies relevant to standard-setting. Unlike particulate matter, ozone is a distinct substance that can be measured in ambient air with recognized monitoring devices. There is no rational basis for excluding from consideration

⁸⁷ Naeher LP, Holford TR, Beckett WS, Belanger K, Triche EW, Bracken MB, Leaderer BP. Healthy Women's PEF Variations with Ambient Summer Concentrations of PM₁₀, PM_{2.5}, SO₄²⁻, H⁺, and O₃. *Am J Respir Crit Care Med* 1999; 160: 117-125.

foreign studies that have been appropriately performed and evaluated. The mere fact that a study was conducted outside of the U.S. or Canada does not provide a reasoned basis for disregarding it.

A study in Seoul, Korea examined the associations of ozone with childhood asthma hospitalizations as stratified by socioeconomic status. The study found that the number of children who were hospitalized for asthma increased as the socioeconomic status decreased, suggesting that air pollution had a disproportionate impact on the poorer children, and that socioeconomic status should be considered as a potential confounding factor.⁸⁸

Australian researchers investigated the effects of ambient air pollution on 13,000 hospital admissions in Brisbane. The authors used the Air Pollution on Health: European Approach (APHEA) protocol to examine the effects of particles, ozone, sulfur dioxide, and nitrogen dioxide on daily hospital admissions for asthma and respiratory, cardiovascular, and digestive disorders (control diagnosis) that occurred during the period 1987-1994. Ozone was consistently associated with admissions for asthma and respiratory disease-with little evidence of a threshold. In two-pollutant models, the ozone effect was relatively unaffected by the control for high levels of other pollutants. In Brisbane, ozone levels are relatively constant year round, and aerosol sulfates were not present so the effect was due to ambient ozone alone.⁸⁹

Another study in Taiwan investigated the relationship of air pollution and weather to asthma prevalence and attack rate in adolescents, specifically junior high school students. After controlling for a variety of potential confounding factors, ozone concentrations at the level of the current U.S. 8-hour average standard were found to be proportional to asthma prevalence in males. Various air pollutants, including ozone, were significantly related to asthma attacks.⁹⁰

Additional evidence of the special sensitivity of asthmatics to ozone air pollution comes from studies exploring genetic susceptibility to asthma. A comprehensive review article reports that asthmatics with the null genotype for the antioxidant, GST, seem more at risk of the pulmonary effects of air pollution.⁹¹ Children in Mexico City with the GSTM1 null genotype demonstrate significant ozone-related decrements in lung function.⁹² Animal models have also identified factors which endow susceptibility to ozone response. Children with certain genotypes had greater increases in breathing difficulty in relation to ozone than other children. Ozone-related pulmonary impairment may be greater in individuals with certain genetic factors that make them more susceptible to oxidative stress.⁹³

⁸⁸ Son JY, Kim H, Lee JT, Kim SY. Relationship Between the Exposure to Ozone in Seoul and the Childhood Asthma-Related Hospital Admissions According to the Socioeconomic Status. *J Prev Med Pub Health* 2006; 39: 81-86.

⁸⁹ Petroschevsky A, Simpson RW, Thalib L, Rutherford S. Associations between outdoor air pollution and hospital admissions in Brisbane, Australia. *Arch Environ Health* 2001; 56: 37-52.

⁹⁰ Ho W-C, Hartley WR, Myers L, Lin M-H, Lin Y-S, Lien C-H, Lin R-S. Air pollution, weather, and associated risk factors related to asthma prevalence and attack rate. *Environ Res* 2007; 104: 402-409.

⁹¹ McCunney RJ. Asthma, genes, and air pollution. *J Occup Environ Med* 2005; 47: 1285-1291.

⁹² Romieu I, Ramirez-Aguilar M, Sienra-Monge JJ, Moreno-Macias H, del Rio-Navarro BE, David G, Marzec J, Hernandez-Avila M, London S. GSTM1 and GSTP1 and respiratory health in asthmatic children exposed to ozone. *Eur Respir J* 2006; 28: 953-959.

⁹³ London SJ. Gene-Air Pollution Interactions with Asthma. *Proc Am Thorac Soc* 2007; 4: 217-220.

Ozone Increases Risk of Mortality at Concentrations below the Current Standard

As EPA acknowledges in its proposal, one of the important new scientific developments that has emerged since the last review is the well-documented relationship between short-term exposures to ozone and premature mortality. Some studies considered in the last review of the ozone standard in 1997 raised the question of the link between ozone and short-term mortality, but EPA did not consider the evidence to be persuasive.

Now a decade later, the evidence is much stronger. 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.

EPA's peer-reviewed science assessment concludes that the overall evidence is highly suggestive that short-term exposure to ozone increases the risk of early death.⁹⁴ The Criteria Document reports that several newer multi-city studies, single-city studies, and several meta-analyses of these studies have provided strong evidence for associations between short-term ozone exposure and total mortality, even after adjustment for the influence of season and PM. In addition, consistently positive associations have been reported for ozone-related cardiovascular mortality across approximately 30 studies.

Further, the Criteria Document concludes that newly available experimental data from both animal and human studies provide evidence suggestive of plausible pathways by which risk of respiratory or cardiovascular morbidity and mortality could be increased by ambient ozone either acting alone or in combination with copollutants. (CD p. 8-78).

According to the Criteria Document, the recent multi-city and single-city studies generally show consistent positive and significant associations between acute ozone exposure and all-cause mortality in studies with 98th percentile 8-hour maximum ozone values of 80 to 85 ppb and above. (CD p. 8-38).

The evidence cited in the Criteria Document provides strong evidence, not only that ozone exposure causes premature death, but that increased risk of mortality is evident at levels well below the standard EPA proposes. The study designs have taken a variety of approaches including single- and multi-city time series and case-crossover approaches. They have explored the possible confounding by temperature, and particulate matter. The discussion below explores 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.

⁹⁴ U.S. Environmental Protection Agency. Air Quality Criteria for Ozone and Related Photochemical Oxidants (Final). Washington, DC, EPA/600/R-05/004aB-cB, 2006.

Multi-city studies

Two critical multi-city studies published the same week in 2004 showed clear evidence of the risk to life. 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.⁹⁵ A large 23-city European study by Gryparis et al. reported a positive association between one- and eight-hour concentrations of ozone air pollution and daily mortality, especially respiratory mortality, during the warm season.⁹⁶

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. 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 show that any threshold would exist at very low concentrations, far below current U.S. standards.

The authors concluded:

“our nationwide study provides strong and consistent evidence that daily changes in ambient O₃ exposure are linked to premature mortality, even at very low pollution levels, including an idealized scenario of complete adherence to current O₃ regulations.”

Importantly even when days exceeding 0.060 were excluded from the analysis, the mortality effect was little changed. As indicated in Figure M below, the relationship between mortality and ozone was evident even on days when pollution levels were below the 0.06 ppm. The ozone and mortality results do not appear to be confounded by temperature or PM₁₀.⁹⁷

⁹⁵ Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. Ozone and short-term mortality in 95 US urban communities, 1987-2000. *JAMA* 2004; 292: 2372-2378.

⁹⁶ Gryparis A, Forsberg B, Katsouyanni K, Analitis A, Touloumi G, Schwartz J, Samoli E, Medina S, Anderson HR, Niciu EM, Wichmann E, Kriz B, Kosnik M, Skorkovsky J, Vonk JM, Dortbudak Z. Acute effects of ozone on mortality from the “Air Pollution and Health: A European Approach” project. *Am J Respir Crit Care Med* 2004; 170: 1080-1087.

⁹⁷ Bell ML, Peng RD, McDermott A, Zeger SL, Samet JM, Dominici F. The Exposure-Response Curve for Ozone and Risk of Mortality and Adequacy of Current Ozone Regulations. *Environ Health Perspect* 2006; 114:532-536. Ozone and short-term mortality in 95 US urban communities, 1987-2000. *JAMA* 2004; 292: 2372-2378.

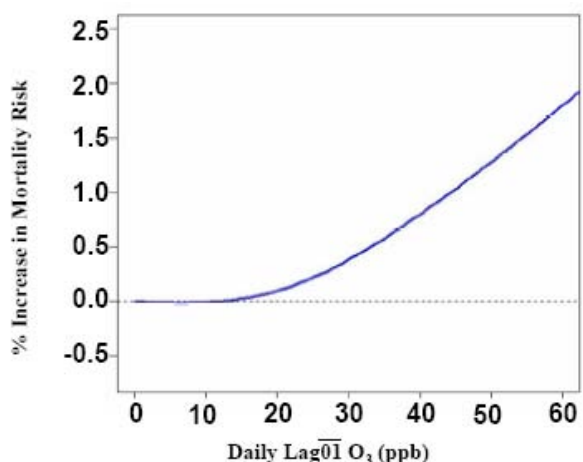


Figure M: Exposure Response Curve for ozone and mortality using the spline approach: percentage increase in daily nonaccidental mortality at various ozone concentrations.

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.

Bell et al. (2008) 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.⁹⁸

Another large multicity study of 48 U.S. cities reported a positive association between ozone and all-cause mortality during the summer months. In addition, 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.⁹⁹

A major study of 18 U.S. communities reported an association between summertime ozone levels and non-accidental mortality. This association was robust to the inclusion of PM_{2.5} in the analysis, strengthening confidence in the ozone-mortality link. 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. Mean daily ozone concentrations in the study ranged by community from 21.4 to 48.7 ppb.¹⁰⁰

⁹⁸ Bell ML, Dominici F. Effect modification by community characteristics on the short-term effects of ozone exposure and mortality in 98 US communities. *Am J Epidemiol* 2008; 167: 086-997.

⁹⁹ Zanobetti A, Schwartz J. Mortality displacement in the association of ozone with mortality: an analysis of 48 cities in the United States. *Am J Respir Crit Care Med* 2008a; 177: 184-189.

¹⁰⁰ Franklin M, Schwartz J. The impact of secondary particles on the association between ambient ozone and mortality. *Environ Health Perspect* 2008; 116: 453-458.

Meta-analyses

Meta-analyses offer compelling evidence that these ozone-mortality findings are consistent. Four meta-analyses completed between 2001 and 2004 reported evidence that ozone contributes to early death.¹⁰¹ 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-analysis at the request of EPA, using their own methods and study selection criteria. All three meta-analyses reported a remarkably consistent link between daily ozone levels and total mortality.^{102,103,104} The results of these meta-analyses are summarized in Figure N below, which illustrates the remarkable consistency in the findings.

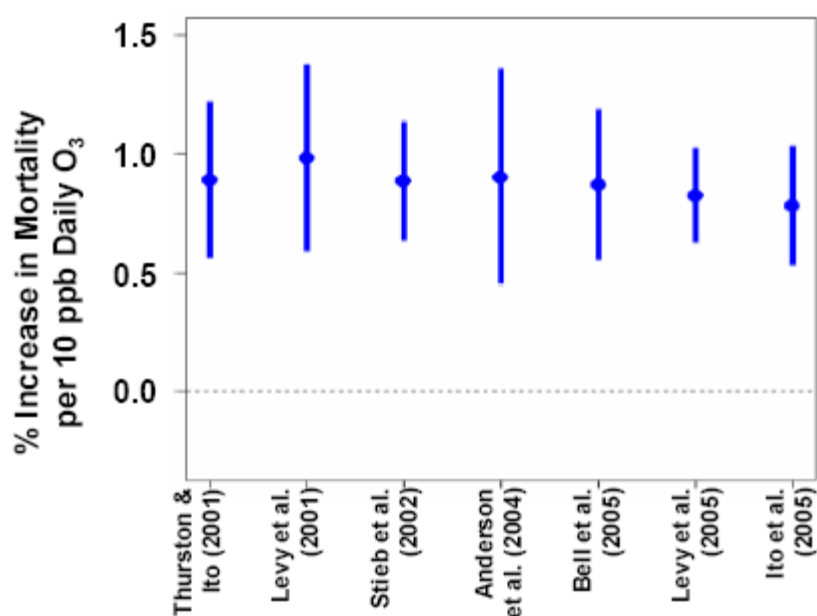


Figure N: Results of the Meta-Analyses studies. From Bell, ML. “Recent Evidence on the Relationship between Ozone and Mortality,” Presentation to the Estimating Mortality Risk Reduction Benefits from Decreasing Tropospheric Ozone Exposure Panel, National Research Council on March 29, 2007

¹⁰¹ Levy JI. Assessing the Public Health Benefits of Reduced Ozone Concentrations. *Environ Health Perspect* 2001; 109: 1215-1226; Thurston CD, Ito K. Epidemiological Studies of Ozone Exposures and Acute Mortality. *J Exposure Analysis and Environ Epidemiology* 2001; 11: 286-294; Anderson HR, Atkinson RW, Peacock JL, Marston L, Konstantinou K. Meta-Analysis of Time-Series Studies and Panel Studies of Particulate Matter (PM) and Ozone (O₃). Report of a WHO Task Group. Copenhagen: World Health Organization, 2004; and Stieb DM, Judek S, Burnett RT. 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* 2002; 52: 470-84.

¹⁰² Bell ML, Dominici F, and Samet JM. A Meta-Analysis of Time-Series Studies of Ozone and Mortality with Comparison to the National Morbidity, Mortality, and Air Pollution Study. *Epidemiology* 2005; 16: 436-445.

¹⁰³ Levy JI, Chermerynski SM, Sarnat JA. Ozone Exposure and Mortality: An Empiric Bayes Metaregression Analysis. *Epidemiology* 2005; 16: 458-468.

¹⁰⁴ Ito K, De Leon SF, Lippmann M. Associations Between Ozone and Daily Mortality: Analysis and Meta-Analysis. *Epidemiology* 2005; 16: 446-429.

Role of temperature and particulate pollution

Numerous studies have reported positive associations between both ozone and high temperatures on short-term mortality. Filleul et al. attempted to tease out the relative contribution of heat and ozone air pollution during the August 2003 heat wave in Europe through advanced statistical analysis of nine French cities. The study found that the ozone mortality effect was present even during the heat wave. The authors conclude: “These results confirmed that in urban areas O₃ levels have a non-negligible impact in terms of public health.”¹⁰⁵

Analyses clearly indicate that the death effect of ozone is distinct from the effect of temperature and particle pollution. A recent case-crossover study of 14 U.S. cities was designed to control for the effect of temperature on daily deaths attributable to ozone. 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.¹⁰⁶ A study *in press* in *Environmental Health Perspectives* investigated whether particulate matter is a confounder of the ozone and mortality association using data for 98 U.S. urban communities from 1987 to 2000. The study concluded that particulate matter is unlikely to confound the short-term association between ozone and mortality.¹⁰⁷

These new studies estimate that cleaning up ozone air pollution could save thousands of lives each year.¹⁰⁸ The analysis by Bell et al. (2004) projects that nearly 4,000 lives would be saved per year by reducing ozone pollution from the prior standard of 0.085 ppm to 0.075 ppm in the 95 U.S. cities studied. The larger the reduction in ozone pollution, the greater the number of lives that would be saved.¹⁰⁹ Researchers looking solely at California data estimated that an ozone standard of 0.070 ppm would reduce annual deaths from ozone by an estimated 630 cases in that state alone.¹¹⁰

Specific Populations at Risk

New evidence warns that some large sub-populations may be at greater risk, including infants, African-Americans and women. Tsai et al. used a case-crossover approach to examine the relationship between various air pollutants and infant mortality in a large city in Taiwan. Positive, though not statistically significant, relationships were reported for a number of specific

¹⁰⁵ Filleul, L, Cassadou S, Médina S, Fabres P, Lefranc A, Eilstein D, Le Tertre A, Pascal L, Chardon B, Blanchard M, Declercq C, Justot J-F, Prouvost H, Ledrans M. The Relation Between Temperature, Ozone, and Mortality in Nine French Cities During the Heat Wave of 2003. *Environ Health Perspect* 2006; 114: 1344-1347.

¹⁰⁶ Schwartz J. How sensitive is the association between ozone and daily deaths to control for temperature? *Am J Resp Crit Care Med* 2005; 171: 627- 631.

¹⁰⁷ Bell ML, Kim JY, Dominici F. Potential Confounding of Particulate Matter on the Short-Term Association Between Ozone and Mortality in Multi-Site Time-Series Studies. *Environ Health Perspect* 2007; doi:10.1289/ehp.10108, Online 2 August 2007.

¹⁰⁸ Bell ML, Peng RD, Dominici F. The Exposure-Response Curve for Ozone and Risk of Mortality and the Adequacy of Current Ozone Regulations. *Environ Health Perspect* 2006; 114: 532-536.

¹⁰⁹ Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. Ozone and short-term mortality in 95 US urban communities, 1987-2000. *JAMA* 2004; 292: 2372-2378.

¹¹⁰ Ostro BD, Tran H, Levy JI. The Health Benefits of Reduced Tropospheric Ozone in California. *J. Air & Waste Manage Assoc.* 2006; 56: 1007-1021.

pollutants including ozone and increased risk of infant death.¹¹¹ A study *in press* suggests that African-Americans may be at higher risk of early death from ozone pollution than the general population.¹¹² A draft analysis prepared for a committee of the National Academy of Sciences indicates that women may be more susceptible to the ozone-mortality effect.¹¹³

Mortality Displacement Issues

With mortality studies, the question always rises over whether the deaths from ozone exposure are just advanced by a few days. Schwartz and Zanobetti, who researched this question for particulate matter mortality studies, used data from 48 U.S. cities between 1989 and 2000 to study the question for ozone. They found that deaths from ozone are not due to “mortality displacement,” and that the deaths are greater when looking three weeks out.¹¹⁴

Biological Plausibility

New 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. As shown in Figure O 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. Ozone can impair neutrophil phagocytosis and intracellular killing.¹¹⁵ Hollingsworth et al. conclude that “understanding the fundamental mechanisms that regulate the biologic response to commonly encountered inhaled environmental toxins will provide a better understanding the increased morbidity and mortality associated with high levels of ambient air pollution.”

¹¹¹ Tsai S-S, Chen C-C, Hsieh H-J, Chang C-C, Yang C-Y. Air Pollution and Postneonatal Mortality in a Tropical City: Kaohsiung, Taiwan. *Inhalation Toxicology* 2006; 18: 185-189.

¹¹² Bell ML, Dominici F. Effect Modification by Community Characteristics on the Short-Term Effects of Ozone Exposure and Mortality in 98 U.S. Communities. *In press. American Journal of Epidemiology*.

¹¹³ Schwartz J. Harvesting, Susceptibility, and the Association of Ozone with Daily Deaths. Draft Presentation to the National Academy of Sciences Committee on Ozone Mortality, July 9, 2007.

¹¹⁴ Schwartz J and Zanobetti A. Is there Short Term Mortality Displacement in the Association of Ozone with Mortality: An Analysis of 48 U.S. Cities. Draft paper presented to the National Academy of Sciences Committee on Ozone Mortality, July 9, 2007.

¹¹⁵ Hollingsworth JW, Kleeberger SR, Foster WM. Ozone and Pulmonary Innate Immunity. *Proc Am Thorac Soc* 2007; 4: 240-246.

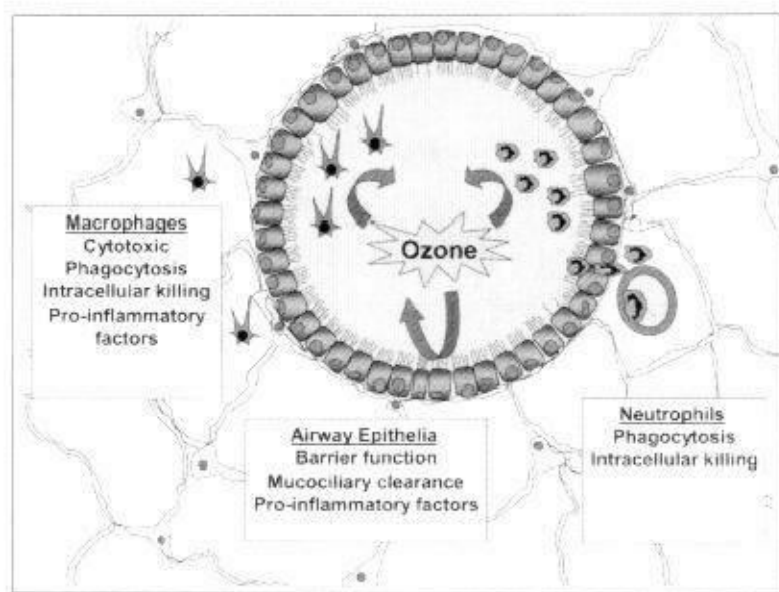


Figure O: Illustration of possible mechanisms for ozone interaction with cells in the lungs. From Hollingsworth et al 2007.

A newly published 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. The ozone primes the immune system to hyper-respond and destroys some of the protective immune cells, leaving the lungs possibly vulnerable to later bacterial infections.¹¹⁶

Finally, the recognition that ozone exposure increases the risk of premature death is driving consensus policy recommendations from scientists. The World Health Organization recently tightened its air quality guidelines for ozone, in part, because of concern about deaths from exposure to low concentrations.¹¹⁷

Effects Persist Even After Excluding Concentrations Above a Certain Level

We would like to emphasize a number of studies which excluded observations above a certain concentration and still found effects. This study design provides compelling evidence of associations evident at low concentrations, and is very pertinent to regulatory standard-setting.

- Brunekreef, 1994: Even after removing all observations with hourly ozone concentrations greater than 60 ppb, a decline in lung function and an increase in respiratory symptoms is evident in this group of amateur cyclists.

¹¹⁶ Hollingsworth JW, Maruoka S, Li Z, Potts EN, Brass DM, Garantzotis S, Fong A, Foster WM, Schwartz DA. Ambient Ozone Primes Pulmonary Innate Immunity in Mice. *J Immunology* 2007; 179: 4367-4375

¹¹⁷ 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>

- Brauer 1996: 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 2002: After excluding days when 8-hour average ozone was greater than 0.080 ppm, the associations with morning lung function decrements remained statistically significant.
- Bell, 2004: Estimates of premature mortality attributable to ozone changed little when days with 24-hour average concentrations greater than 0.06 ppm were excluded.
- Bell, 2006: There was little difference in the mortality effect estimate when days with 24-hour ozone concentrations above 0.02 ppm were excluded.

The Criteria Document concludes:

“While no fully confident conclusion can be made regarding the threshold issue from epidemiologic studies alone, the limited currently available evidence suggests that if a population threshold level exists in O₃ health effect, it is likely near the lower limit of ambient O₃ concentrations in the United States. (CD p. 7-159).

Toxicological Studies Indicate Serious Adverse Effects

Toxicological studies are an extremely valuable complement to the chamber and epidemiological studies because they provide information on biological modes of action and biological plausibility. A major advantage of animal studies is that exposures can be carefully controlled, and experiments can be designed so that the highest exposure results in measurable adverse effects. These adverse effects can be monitored through both in-life observation and measurements and through examination of tissues upon death.¹¹⁸

A limitation in using animal studies to support standard-setting stems from the need to extrapolate findings to humans. This is typically managed by the use of safety factors that take into account intra-species variability, say from rat to humans, and individual variability in human populations. EPA typically applies a safety factor of 10 to each of these factors, and reference concentrations are set at 1/100 of the No Observed Effect Level (NOEL) or 1/1000 of the Lowest Observed Effect Level (LOEL).¹¹⁹ Taken in this framework, the relatively high doses used in animal studies do not preclude them from consideration for standard-setting purposes.

¹¹⁸ Australian Government, National Health and Medical Research Council. Ambient Air Quality Standards Setting: An Approach to Health-Based Hazard Assessment, September 2006.

¹¹⁹ Barnes DG, Dourson M and USEPA Reference Dose (RfD) Work Group. Reference Dose (RfD); Description and use in health risk assessments. *Reg Tox and Pharm* 1988; 8:471-486.

One of the most important developments in recent years has been the series of studies evaluating the long-term morphological effects of ozone exposure in infant rhesus monkeys. The Criteria Document reports that these studies in primates have demonstrated that long-term exposures can lead to “remodeling” of the distal airways; abnormalities in tracheal basement membrane; eosinophil accumulation in conducting airways; and decrements in airway innervation. (CD p. 5-34).

The Criteria Document acknowledges that these are disturbing findings. But when discussing them in the integrated synthesis, the Criteria Document states: “Most of the research results alluded to [in] the ensuing discussion come from toxicology studies using various laboratory animal species that were usually exposed to higher, non-ambient concentrations of O₃....Again, caution should be exercised in extrapolating these observations to humans, due to species-specific differences...” (CD p. 8-32).

Laboratory studies of test animals almost always employ high doses because of the small number of animals tested. This compelling body of research should not be so readily dismissed because of the necessity of high doses. Such long term studies simply could not be conducted in humans and the animal studies provide valuable insights into the pathophysiology underlying human functional responses to prolonged inhalation of ozone. In many other contexts, EPA relies on effect levels in animal studies in conjunction with multiple safety factors to derive environmental standards.

In its review of the state ozone standards, the California EPA Staff Report stated:

“A series of studies conducted in infant rhesus monkeys indicates that ozone exposure alone and especially in combination with allergen results in altered lung development. This series of studies is particularly important because of concerns that the ozone standards recommended adequately protect infants and children. Lung development in the infant rhesus monkey parallels that in humans. Thus, although the concentrations employed in the studies were higher than attained in current ambient exposures, the implications are quite important.”¹²⁰

Eighty percent of lung development in humans occurs after birth continuing through adolescence.¹²¹ Lung development is studied in rhesus monkeys because their airway structure and postnatal lung development is similar to those of humans. A study in infant rhesus monkeys tested whether repeated cycles of injury and repair caused by ozone exposure lead to chronic airway disease and decreased lung function by altering normal lung maturation. One month old monkeys were exposed to 0.5 ppm ozone episodically over a five month period. Compared with control monkeys, the ozone exposed animals had major differences in airway structure and morphology: four fewer nonalveolarized airway generations, hyperplastic bronchiolar

¹²⁰ California Environmental Protection Agency, Air Resources Board. Review of the California Ambient Air Quality Standard for Ozone, Staff Report Volume 1, March 11, 2005. p. 9-132.

¹²¹ American Academy of Pediatrics Committee on Environmental Health, Ambient Air Pollution: health hazards to children. *Pediatrics* 2004; 114: 1699-1707

epithelium, and altered smooth muscle bundle orientation in terminal and respiratory bronchioles.¹²²

An important 2003 study found that ozone alters the development of the trachea in infant rhesus monkeys. This study examined the development of the "basement membrane zone" in the trachea of infant rhesus monkeys exposed to ozone, filtered air, and ozone plus allergen from house dust mites. In monkeys, this structure develops after birth, allowing studies of the effects of environmental exposures. The study identified significant differences, including irregular width, in the tracheal "basement membrane zone" in monkeys exposed to either ozone alone, or ozone plus allergens, during the developmental period. This resulted in altered regulation of proteins that may explain the atypical development of the lung observed in rhesus monkeys after exposure to ozone.¹²³

A review article summarizing the large body of research on infant rhesus monkeys explores which early life influences affect airway structure and function and how postnatal exposure to ozone and allergens may alter airway development leading to the development of asthma.

“Evaluation of the pathobiology of airway remodeling in growing lungs of neonates, using an animal model where exposure to allergen generates reactive airways disease will all the hallmarks of asthma in humans, illustrates that exposure to environmental pollutants and allergens early in life produces a large number of disruptions of fundamental growth and differentiation processes. All the compartments of the epithelial mesenchymal tropic unit are changed, including acceleration of mucous cell development, disruption of basement membrane growth and reorganization, alterations in the organization and orientation of airway smooth muscle, down regulation of innervation of the epithelial compartment, and disruption of the sites of residence for migratory inflammatory and immune cells. In addition, airway remodeling in neonatal lungs also involves restriction in the growth of tracheobronchial airways as well as fundamental alterations in branching number. Most of these disruptions do not appear to be easily correctable by subsequent extended periods in an environment free of either oxidant stressors or allergens.”¹²⁴

Studies in other test animals have also bolstered the clinical and epidemiological studies and provide plausibility for effects reported in other studies. A newly published laboratory

¹²² Fanucchi MV, Plopper CG, Evans MJ, Hyde DM, Van Winkle LS, Gershwin LJ, Schelegle ES. Cyclic Exposure to Ozone Alters Distal Airway Development in Infant Rhesus Monkeys. *Am J Physiol Lung Cell Mol Physiol* 2006; 291: 644-650.

¹²³ Evans MJ, Fanucchi MV, Baker GL, Van Winkle LS, Pantle LM, Nishio SJ, Schelegle ES, Gershwin LJ, Miller LA, Hyde DM, Sannes PL, Plopper CG. Atypical Development of the Tracheal Basement Membrane Zone of Infant Rhesus Monkeys Exposed to Ozone and Allergen. *American Journal of Physiology - Lung Cellular and Molecular Physiology* 2003; 285: 931-939.

¹²⁴ Plopper CG, Smiley-Jewell SM, Miller LA, Fanucchi MV, Evans MJ, et al. Asthma/Allergic Airways Disease: Does Postnatal Exposure to Environmental Toxicants Promote Airway Pathobiology. *Toxicologic Pathology* 2007; 35: 97-110.

toxicology study in rats found that immature and aged rats displayed lung oxidative stress after ozone exposure, as compared to adult specimens.¹²⁵

These studies suggest that ozone may be causing serious long-lasting effects in infants and young children whose airways are undergoing rapid growth and development. Toxicological studies must employ high doses because of the small number of animal subjects tested. Since humans cannot be studied experimentally, these studies were designed to use a non-human primate model to provide information about health effects and mechanisms in humans. EPA's interpretation of these studies should give them meaning in the context of setting standards to protect against acute and chronic effects in humans.

EPA Must Adopt a 0.060 ppm Standard to Protect Against Anticipated Effects

Under the Clean Air Act, EPA must take into account effects that are anticipated but not yet proven in determining an appropriate margin of safety. In the case of ozone, EPA reviewed a number of studies in the Criteria Document showing effects of long-term exposures of ozone on lung function, asthma induction, and cancer, as well as reproductive and perinatal effects. In each of these cases, EPA found that there was insufficient evidence to draw strong conclusions about cause-effect relationships. However, EPA's findings, even if valid, do not absolve EPA from regulating to protect against these effects. To the contrary, the Clean Air Act requires EPA to account for effects anticipated but not yet proven in providing for a "margin of safety" when setting air quality standards. Here, we briefly review evidence from some key studies on effects including chronic effects, cancer effects, cardiovascular effects, and reproductive effects which demand that EPA set a standard of 0.060 ppm or below to provide an adequate margin of safety.

Long-term Exposures Diminish Lung Reserves

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.

A recent 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.¹²⁶

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. The highest and

¹²⁵ Servais S, Boussouar A, Molnar A, Douki T, Pequignot JM, Favier R. Age-Related Sensitivity to Lung Oxidative Stress During Ozone Exposure. *Free Radic Res* 2005; 39: 305-316.

¹²⁶ Meng YY, Wilhelm M, Rull RP, English P, Ritz B. Traffic and outdoor air pollution levels near residences and poorly controlled asthma in adults. *Ann Allergy Asthma Immunol* 2007; 98: 455-463.

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."¹²⁷ This effect of ozone was confirmed in a follow-up study.¹²⁸

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."¹²⁹

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.¹³⁰

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 on spatial interpolation of PM₁₀, 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.¹³¹

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

¹²⁷ Frischer T, Studnicka M, Gartner C, Tauber E, Horak F, Veiter A, Spengler J, Kühr J, Urbanek R. Lung Function Growth and Ambient Ozone: A Three-Year Population Study in School Children. *Am J Respir Crit Care Med* 1999; 160: 390-396.

¹²⁸ Horak F Jr, Studnicka M, Gartner C, Spengler JD, Tauber E, Urbanek R, Veiter A, Frischer T. Particulate Matter and Lung Function Growth in Children: A 3-yr Follow-up Study in Austrian Schoolchildren. *Eur Respir J* 2002; 19: 838-845.

¹²⁹ Galizia A, Kinney PL. Long-Term Residence in Areas of High Ozone: Associations with Respiratory Health in a Nationwide Sample of Nonsmoking Young Adults. *Environ Health Perspect* 1999; 107: 675-679.

¹³⁰ Künzli N, Lurmann F, Segal M, Ngo L, Balmes J, and Tager IB. Association between Lifetime Ambient Ozone Exposure and Pulmonary Function in College Freshmen: Results of a Pilot Study. *Environ Res* 1997; 72: 8-23.

¹³¹ Tager IB, Balmes, Lurmann F, Ngo L, Alcorn S, and Künzli. Chronic Exposure to Ambient Ozone and Lung Function in Young Adults. *Epidemiology* 2005; 16: 751-759.

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.¹³²

A recent 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 FEV₁ 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 FEV₁ 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.¹³³

These studies are reinforced by the findings of animal toxicology studies discussed earlier, and from human studies discussed below.

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.¹³⁴

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

¹³² Gauderman WJ, Gilliland GF, Vora H, Avol E, Stram D, McConnell R, Thomas D, Lurmann F, Margolis HG, Rappaport EB, Berhane K, Peters JM. Association between Air Pollution and Lung Function Growth in Southern California Children: Results from a Second Cohort. *Am J Respir Crit Care Med* 2000; 162: 1383–1390.

¹³³ Rojas-Martinez R, Perez-Padilla R, Olaiz-Fernandez G, Mendoza-Alvarado L, Moreno-Macias H, Fortoul T, McDonnell W, Loomis D, Romieu I. Lung Function Growth in Children with Long-Term Exposure to Air Pollutants in Mexico City. *Am J Respir Crit Care Med* 2007; 176: 377-384.

¹³⁴ Calderón-Garcidueñas L, Mora-Tiscareño A, Fordham LA, Cheng CJ, Valencia-Salazar G, Flores-Gómez S, Solt AC, Gomez-del-Campo A, Jardón-Torres R, Henriquez-Roldán C, Hazucha MJ, Reed W. Lung Radiology and Pulmonary Function of Children Chronically Exposed to Air Pollution. *Environ Health Perspect* 2006; 114: 1432-1437.

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.¹³⁵

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."¹³⁶

These findings are extremely significant to EPA's evaluation of long-term effects.

Modern epidemiological studies are subjected to rigorous statistical analysis to control for the possible confounding effect of multiple pollutant exposures. Many studies, for instance, have demonstrated an independent association of short-term exposures of ozone to premature mortality. In a large U.S. cohort study, Jerrett et al. (2009) reported that long-term exposure to ozone was associated with a significant increased risk of death from respiratory causes.¹³⁷

¹³⁵ Calderón-Garcidueñas L, Mora-Tiscareño A, Fordham LA, Valencia-Salazar G, Chung CJ, Rodríguez-Alcaraz A, Paredes R, Variakojis D, Villarreal-Calderón A, Flores-Camacho L, Antunez-Solis A, Henríquez-Roldán, Hazucha MJ. Respiratory Damage in Children Exposed to Urban Pollution. *Pediatric Pulmonology* 2003; 36: 148-161.

¹³⁶ Calderón-Garcidueñas L, Valencia-Salazar G, Rogríquez-Alcaraz A, Gambling TM, García R, Osnaya N, Villarreal-Calderón A, Devlin RB, and Carson JL. Ultrastructural Nasal Pathology in Children Chronically and Sequentially Exposed to Air Pollutants. *Am J Resp Cell Molec Bio* 2001; 24: 132-138.

¹³⁷ Jerrett M, Burnett RT, Pope CA III, Kazuhiko I, Thurston G, Krewski, D, Shi Y, Calle E, Thun M. Long-Term Ozone Exposure and Mortality. *N Engl J Med* 2009; 360: 1085-1095.

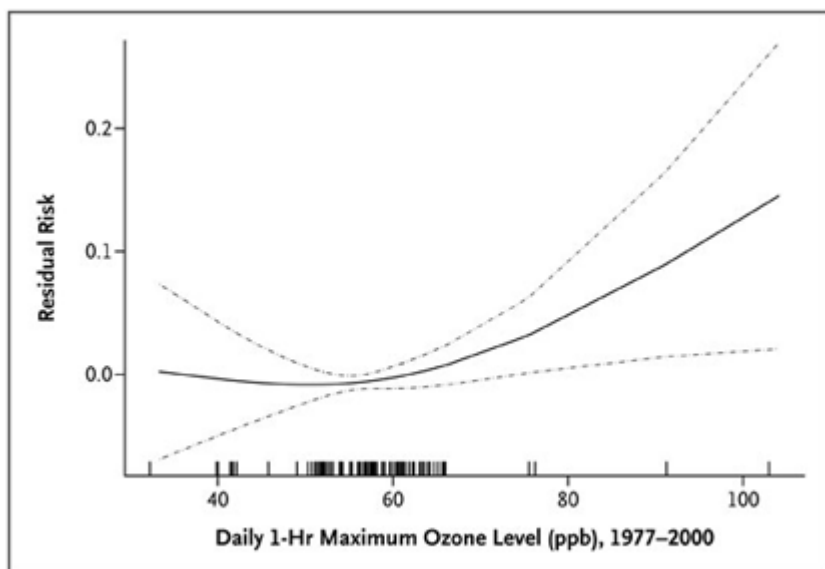


Figure 2. Exposure–Response Curve for the Relation between Exposure to Ozone and the Risk of Death from Respiratory Causes.

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.

Figure P: from Jerrett et al., 2009.

Asthma development

Two 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.¹³⁸

An 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

¹³⁸ McDonnell WF, Abbey DE, Nishino N, and Lebowitz MD. Long-Term Ambient Ozone Concentration and the Incidence of Asthma in Nonsmoking Adults: The Ashmog Study. *Environ Res* 1999; 80: 110-121.

California communities to determine the potential health damage caused by growing 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."¹³⁹ 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.¹⁴⁰

A recent German study has reported that ozone and NO_x air pollution modifies proteins from pollen and other sources in ways likely to make them more allergenic and more likely to trigger an asthma attack.¹⁴¹

While the Staff Paper concluded that there was insufficient evidence at this time to establish a long-term standard for ozone, EPA must set the 8-hour standard with a margin of safety sufficient to account for the likelihood that future studies will confirm that exposures to ozone are causing chronic adverse effects on lung capacity. The currently available information on long-term effects supports the need for an 8-hour standard at the lower end of the range recommended by CASAC and EPA staff scientists.

Cardiovascular Effects

The Criteria Document and Staff Paper address the effect of ozone on cardiovascular responses. "Based on the evidence from animal toxicology, human controlled exposure, and epidemiologic studies, the CD concludes that this generally limited body of evidence is highly suggestive that O₃ can directly and/or indirectly contribute to cardiovascular-related morbidity, but that much needs to be done to more fully substantiate links between ambient O₃ exposures and adverse cardiovascular outcomes (CD, p. 8-77)." (SP p. 3-27).

Emerging research is adding to the weight of evidence about the potential cardiovascular effects of ozone. Numerous recent studies point to adverse associations between ozone exposure and various cardiovascular health endpoints. For example Henrotin et al. recently reported that short-term exposures to ozone are associated with ischemic stroke occurrence. This 10-year case-crossover analysis from a population-based study in Dijon, France found a positive

¹³⁹ Pinkerton KE, Balmes JR, Fanucchi MV, Rom WN. Ozone, a malady for all ages. *Am J Respir Crit Care Med* 2007; 176: 107-108.

¹⁴⁰ McConnell R, Berhane K, Gilliland FD, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. Asthma in Exercising Children Exposed to Ozone. *The Lancet* 2002; 359: 386-391.

¹⁴¹ Franze T, Weller MG, Niessner R, Pöschl. Protein Nitration by Polluted Air. *Enviro Sci Technol* 2005; 39: 1673-1678.

association for a 5 ppb increase in ozone exposure and ischemic stroke occurrence in men, with a 1-day lag. This association remained significant after accounting for particulate matter.¹⁴²

A population-based study recently published in the journal *Circulation* after the publication of the draft CD reported that short-term exposures to ozone predict alterations in cardiac autonomic function as measured by heart rate variability among older adults.¹⁴³

A case-crossover study in France has reported that ozone exposure within a period of 1 to 2 days is associated with heart attacks in middle-aged adults without heart disease. The study design allowed for control of long-term seasonal trends, and adjusted for temperature, relative humidity, and influenza epidemics.¹⁴⁴ Rich et al. evaluated cardiac arrhythmias in patients with implanted cardioverter defibrillators in association with various measures of community air pollution. Breathing increased ambient ozone concentrations during the previous hour was associated with increased risk of episodes of a particular type of cardiac arrhythmia, suggesting that community air pollution may precipitate of these events. Associations with PM_{2.5}, nitrogen dioxide, and black carbon were positive, but not statistically significant. These episodes, known as atrial fibrillation, are not generally considered lethal, but are tied to an increased risk of premature death. People with this condition have a five-fold increased risk of stroke if their episodes are not controlled by medication.¹⁴⁵

A large number of epidemiologic studies from around the world have reported an association between various air pollutants and hospital admissions for cardiovascular causes.

An important study tracked hospital admissions for cardiovascular diseases at all 11 Denver County hospitals during July and August, two extreme temperature months, for a four year period. The study focused on men and women older than 65 years of age. Researchers found that ozone increases the risk of hospitalization for acute myocardial infarction, coronary atherosclerosis, and pulmonary heart disease. Researchers conclude that "exposures to higher air pollutant concentrations (except for particulate matter and NO), *even at levels that meet federal air quality standards*, appear to have an effect of increasing the number of hospital admissions for cardiovascular diseases as a whole." (Emphasis added). In this study, 24-hour average ozone concentrations were 25.0 ppb $\pm$ 6.61, and maximum concentrations were 40.2 ppb, as shown in Table 9. This study provides critical evidence for a far stricter standard than proposed by EPA.¹⁴⁶

¹⁴² Henrotin JB, Besancenot JP, Bejot Y, Giroud M. Short-term effects of ozone air pollution on ischaemic stroke occurrence: a case-crossover analysis from a 10-year population-based study in Dijon, France. *Occup Environ Med* 2007; 64: 4439-445.

¹⁴³ Park SK, O'Neill MS, Vokonas PS, Sparrow D, and Schwartz J. Effects of Air Pollution on Heart Rate Variability: The VA Normative Aging Study. *Environ Health Perspect* 2005; 113: 304-309.

¹⁴⁴ Ruidavets J-B, Cournot M, Cassadou S, Giroux M, Meybeck M, Ferrières J. Ozone Air Pollution is Associated with Acute Myocardial Infarction. *Circulation* 2005; 111: 563-569.

¹⁴⁵ Rich DQ, Mittleman MA, Link MS, Schwartz J, Luttmann-Gibson H, Catalano PJ, Speizer FE, Gold DR, and Dockery DW. Increased Risk of Paroxysmal Atrial Fibrillation Episodes Associated with Acute Increases in Ambient Air Pollution. *Environ Health Perspect* 2006; 114: 120-123.

¹⁴⁶ Koken PJ, Piver WT, Ye F, Elixhauser A, Olsen LM, Portier CJ. Temperature, air pollution, and hospitalization for cardiovascular diseases among elderly people in Denver. *Environ Health Perspect* 2003; 111: 1312-1317.

Table 2. Summary statistics for daily levels of environmental variables in Denver, July–August 1993–1997.

Variable	Source	No.	Mean	Minimum	Percentile			Maximum	SD
					25th	50th	75th		
NO ₂ (ppb)	AIRS	303	32.7	13.0	28.0	33.2	37.9	51.3	7.24
SO ₂ (ppb)	AIRS	310	5.7	0.4	3.8	5.3	7.2	18.9	2.94
O ₃ (ppb)	AIRS	310	25.0	5.4	20.0	25.2	29.7	40.2	6.61
CO (ppm)	AIRS	310	0.9	0.3	0.8	0.9	1.1	1.6	0.27
PM ₁₀ (µg/m ³)	AIRS	298	24.2	7.0	20.0	24.0	28.0	51.6	6.25
T _{max} (°F)	NCDC	310	87.4	62.0	83.0	88.5	92.5	99.0	7.37
DPT (°F)	NCDC	310	48.6	26.8	44.2	49.3	53.3	62.4	6.23

Data from U.S. EPA (2002) and NCDC (2002).

Table 9: From Koken et al., 2003.

Researchers examined data on 4 million emergency department visits during a 7 year period to 31 hospitals in Atlanta in a study of air pollution risks. Visits for cardiovascular disease were examined in relation to levels of various ambient air pollutants using a case-crossover approach. Stronger associations were observed for cerebrovascular visits among people with COPD, particularly in association with ozone levels. These findings provide further evidence of increased susceptibility to adverse cardiovascular events associated with ozone air pollution among persons with COPD. Eight-hour mean ozone concentrations in this study were 55.6 ppb, with the 90th percentile concentration 87.6 ppb.¹⁴⁷

Von Klot et al. evaluated the short-term effects of urban air pollution on cardiac hospital readmissions in survivors of heart attacks in five European cities. Positive associations between same day concentrations of a number of pollutants including ozone and increased risk of hospital cardiac readmissions were reported.¹⁴⁸

A recent finding echoing the possible development of asthma in children is evidence that ozone may be associated with the development of type 1 diabetes in children. Hathout et al. studied the role of ambient air pollutants in type 1 diabetes in children. Pre-diagnosis exposure to five air pollutants was studied in two subgroups with onset of type 1 diabetes before and after five years of age, and two matched subgroups of healthy children. The study concluded that increased ozone exposure may be a contributory factor to the increased incidence of type 1 diabetes.¹⁴⁹

A follow-up study of 400 children reported that cumulative exposure to ozone, and to a lesser extent sulfate, in ambient air may predispose children to the development of type 1 diabetes. Mean cumulative ozone exposures in children with diabetes averaged 29.4 ± 7 ppb, compared to 25.8 ± 5 ppb in controls. According to the authors, ozone may predispose children to type 1 diabetes by causing free-radical damage to β -cells or enhancing the presentation of diabetes

¹⁴⁷ Peel JL, Metzger KB, Klein M, Flanders WD, Mulholland JA, Tolbert PE. Ambient Air Pollution and Cardiovascular Emergency Department Visits in Potentially Sensitive Groups. *Am J Epidemiol* 2007; 165: 625-633.

¹⁴⁸ von Klot S, Peters A, Aalto P, Bellander T, Berglind N, D'Ippoliti D, Elosua R, Hörmann A, Kumala M, Lanki T, Löwel H, Pekkanen J, Picciotto S, Sunyer J, Forastiere F. Ambient Air Pollution is Associated with Increased Risk of Hospital Cardiac Readmissions of Myocardial Infarction Survivors in Five European Cities. *Circulation* 2005; 112: 3073-3079.

¹⁴⁹ Hathout EH, Beeson WL, Nahab F, Rabadi A, Thomas W, Mace JW. Role of Exposure to Air Pollutants in the Development of Type 1 Diabetes Before and After 5 Yr of Age. *Pediatr Diabetes* 2002; 3: 184-188.

promoting antigens. “There is experimental evidence that ozone alters T-cell-dependent immune responses and adversely affects DC4⁺ cells, an internal milieu which is quite typical of autoimmune diseases including type 1 diabetes.”¹⁵⁰

A study in which mice were exposed to ozone in a cyclic, intermittent pattern (or filtered air as a control exposure) researchers reported that inhaled ozone, in the absence of other environmental toxicants, promotes increased vascular dysfunction, oxidative stress, mitochondrial damage, and atherogenesis (the formation of plaque deposits on the lining of blood vessels).¹⁵¹

These and many other studies of the cardiovascular effects of ozone show the need for EPA to finalize a more protective standard that accounts for these anticipated but not yet proven effects.

Ozone and Cancer

While the Criteria Document concludes that the weight of evidence from animal toxicology studies and epidemiologic studies does not support ambient ozone as a pulmonary carcinogen (CD p. 8-79) a number of recent studies discussed briefly below provide suggestive evidence of an association between ozone exposures and cell damage, formation of DNA adducts, and neoplasms.

A recent longitudinal study using a micronuclei (MN) assay provides suggestive evidence of an association between ozone air pollution and cytogenic damage in oral epithelia cells. These results were corroborated in a controlled acute ozone exposure in a chamber, where a similar result was seen in two cell types, blood lymphocytes, and buccal (cheek) cells. Investigators concluded:

“The suggestive evidence of MN induction by summer-time air pollution, characterized by elevated ambient O₃, was observed in a longitudinal study of healthy you adults and was further supported by results from the controlled O₃ chamber study. In addition to published data showing effects of ambient O₃ exposure on DNA damage, common diseases and morality in humans, cytogenic data by the MN assay in human lymphocytes and exfoliated cells also indicate a possibility that high oxidant environments may pose a greater threat to public health than previously thought.”¹⁵²

Another study of 65 African American children and their mothers from Oakland, California used geographic information systems to explore possible associations between chromosomal damage

¹⁵⁰ Hathout EH, Beeson WL, Ischander M, Rao R, Mace JW. Air pollution and type 1 diabetes in children. *Pediatric Diabetes* 2006; 7: 81-87.

¹⁵¹ Chauang GC, Yang Z, Westbrook DG, Pompilius M, Ballinger CA, White CR, Krzywanski DM, Postlethwait EM, Ballinger SW. Pulmonary ozone exposure induces vascular dysfunction, mitochondrial damage, and atherogenesis. *Am J Physiol Lung Cell Mol Physiol* 2009; doi:10.1152/ajplung.00102.2009.

¹⁵² Chen C, Arjomandi M, Qin H, Balmes J, Taber I, Holland N. Cytogenic damage in buccal epithelia and peripheral lymphocytes of young healthy individuals exposed to ozone. *Mutagenesis* 2006; 21: 131-137.

and proximity to traffic and regional ozone levels. Regional ozone levels were strongly associated with micronuclei frequency in blood and buccal cells in children and adults.¹⁵³

A case-control study nested in the European Prospective Investigation into Cancer and Nutrition was designed to investigate the ability of DNA adducts to predict cancer and to explore the role of air pollutants as precursors to adducts. Adducts were found to be associated with the subsequent risk of lung cancer, and a positive association was found between DNA adducts and ozone concentration. Researchers concluded that DNA adducts may predict lung cancer risk in never-smokers, and that the association of DNA adduct levels with ozone indicates a possible role for photochemical smog in determining DNA damage.¹⁵⁴

A study in São Paulo, Brazil found that ozone exposure was correlated with tumors of the larynx and lung.¹⁵⁵

EPA must ensure that it promulgates a final standard that provides a margin of safety sufficient to protect against effects such as cancer that are suggested in the current literature but not yet proven.

Perinatal Effects

A fourth category of effects anticipated but not yet proven relates to effects of ozone on the developing fetus and newborns. Several recent studies provide evidence of effects at low levels of exposure to ozone air pollution, and reinforce the need for a standard of 0.060 ppm or below to protect public health, including the health of infants, with an adequate margin of safety.

Prenatal exposure to ozone has been linked to reduced birth weight. A study investigated the effects of air pollutants on birth weight among term infants who were born in California during 1975-1987 and who participated in the Children's Health Study. Birth certificates provided maternal reproductive history and residence at time of birth. Information on sociodemographic factors and maternal smoking during pregnancy were collected by questionnaire. Monthly average air pollutant levels were interpolated from monitors to the zip code of the mother's residence at childbirth.

The researchers observed an association between lower birth weight and intrauterine growth retardation with ozone concentrations. Second- and third-trimester ozone levels were most strongly associated with deficits in birth weight, followed by carbon monoxide exposures during the first trimester. They reported a clear pattern of increasing deficits in birth weight with increasing levels of ozone for 24-hour ozone levels above 30 ppb.

¹⁵³ Huen K, Gunn L, Duramad P, Jeng M, Scalf R, Holland N. Application of a Geographic Information System to Explore Associations Between Air Pollution and Micronucleus Frequencies in African American Children and Adults. *Environ Mol Mutagen* 2006; 47: 236-246.

¹⁵⁴ Peluso M, Munnia A, Hoek G, Krzyanowski M, Veglia F, et al. DNA Adducts and Lung Cancer Risk: A Prospective Study. *Cancer Res* 2005; 65: 8042-8048.

¹⁵⁵ Pereira GA, de Assuncao JV, Saldiva PH, Pereira LA, Mirra AP, Braga AL. Influence of air pollution on the incidence of respiratory tract neoplasm. *J Air Waste Manag Assoc* 2005; 55: 83-87.

Although the differences in birth weight were small on average, those in the highest ozone exposure group had deficits of a magnitude equivalent to those observed after exposure to cigarette smoke. “Because exposures to the levels of ambient air pollutants observed in this study are common, and fetal growth is an important determinant for childhood and adult morbidity and mortality, our findings are likely to have important public health and regulatory implications,” conclude the researchers.¹⁵⁶

An Australian study assessed preterm birth in relation to maternal exposure to ambient air pollution. Over 28,000 births occurring over a three year period in Brisbane were retrospectively assessed. Exposure to ozone during the first trimester was associated with a 26 percent increase in the risk of preterm birth. Mean 8-hour ozone concentrations were 26.7 ppb, and maximum ozone concentrations were 61.1 ppb. The authors concluded that maternal exposure to low levels of ambient air pollution is associated with preterm birth.¹⁵⁷

A UCLA study provides compelling evidence that contemporary concentrations of ozone air pollution may play a role in causing some birth defects. Pregnant Los Angeles-area women living in regions with higher levels of ozone and carbon monoxide pollution were as much as three times as likely to give birth to children who suffered from serious heart defects. Researchers analyzed information collected by the California Birth Defects Monitoring Program on more than 9,000 babies born from 1987 to 1993 in Los Angeles, Orange, San Bernardino and Riverside counties. Using measurements made regularly at 30 locations by the South Coast Air Quality Management District, researchers compared air quality near the homes of cases to air quality in the neighborhoods of children born healthy. Pregnant women who were exposed to increased levels of ozone and carbon monoxide faced an elevated risk of having a child with conotruncal heart defects, pulmonary artery/valve defects and aortic artery/valve defects. This group of heart defects occurs 1.76 times per 1,000 births, with about 935 cases in California each year. Many of these babies face open-heart surgery before age one.¹⁵⁸

As research continues on perinatal effects of ozone and other ambient air pollutants, we urge that EPA set a standard at the low end of the range recommended by CASAC to protect against this important category of effects that may be proven in the future.

¹⁵⁶ Salam MT, Millstein J, Li Y-F, Lurmann FW, Margolis HG, Gilliland FD. Birth Outcomes and Prenatal Exposure to Ozone, Carbon Monoxide, and Particulate Matter: Results from the Children’s Health Study. *Environ Health Perspect* 2005; 113: 1638-1644.

¹⁵⁷ Hansen C, Neller A, Williams G, Simpson R. Maternal exposure to low levels of ambient air pollution and preterm birth in Brisbane, Australia. *BJOG* 2006; 113: 935-941.

¹⁵⁸ Ritz B, Yu F, Fruin S, Chapa G, Shaw GM, Harris JA. Ambient Air Pollution and Risk of Birth Defects in Southern California. *Am J Epidemiology* 2002; 155: 17-25.

The EPA Risk Assessment Supports a 0.060 ppm Standard

The EPA's health risk assessment estimates the health effects associated with short-term exposures to ozone in select urban areas. The risk assessment demonstrates that the EPA's current standard of 0.075 ppm will result in significant residual public health risks, particularly in vulnerable populations.

Risk estimates were generated for several ozone-related morbidity endpoints. Lung function is considered to be a sensitive measure of response to ozone, and the risk assessment clearly demonstrates that a more stringent ozone standard will result in significant improvement in lung function in active children. The EPA risk assessment shows that relative to the prior standard of 0.085 ppm, a standard of 0.065 ppm -- the most stringent option analyzed -- would reduce by up to 80 percent the number of school-aged children estimated to experience moderate lung function decrements in the 12 cities analyzed.¹⁵⁹ To emphasize the obvious, if a standard of 0.065 ppm reduces the number of children experiencing lung function decrements by 80 percent, 20 percent of the children remain unprotected by a standard at that level. Further, these children are estimated to experience multiple incidences of lung function decline.

To reduce the considerable residual risk evident at 0.065 ppm, EPA must adopt a more stringent ozone standard of 0.060 ppm or below -- a level that incorporates an adequate margin of safety.

The Risk Assessment Systematically Underestimates Health Risks

The EPA limited risk assessment was developed to explore the health implications of alternate standards, but it is extremely conservative in its estimation of risks. While it is easy to focus on the numbers, it is important to recognize that the risk assessment is quite limited, in terms of cities included, populations covered, and health endpoints analyzed, in addition to the numerous limitations of the exposure assessment which feeds into the risk assessment. If the resulting risk estimates seem small, it is because they are leaving out most of the health impacts, and most of the country, and many affected populations. For example, the risk assessment examines respiratory symptoms in asthmatic children in just one city, Boston.

While the EPA risk assessment does document the presence of significant health risks to populations exposed to ozone concentrations well below the current standards, the risk assessment contains several flaws that actually underestimate these risks. These flaws are the exclusion of key health endpoints and the exclusion of certain vulnerable populations. Because of these serious underestimates, the risk assessment very likely understates risk to a substantial degree.

¹⁵⁹ Wegman, Lydia, Director, Health and Environmental Impacts Division, Office of Air Quality Planning & Standards, EPA. Current Thinking about Ozone Health Effects and Standard Setting: Update on EPA's Review of O₃ NAAQS. HEI Annual Conference, Chicago, IL April 17, 2007.

Geographic Scope is Limited

The geographic scope of the Risk Assessment is quite limited, covering just 12 metropolitan statistical areas (MSAs). The vast majority of metropolitan areas, 351 of the 363 MSAs in the United States,¹⁶⁰ are excluded from the analysis.

The main constraint appears to be EPA's preference to apply risk functions only in the cities in which they were generated by the original study. This narrow interpretation of the epidemiological studies greatly limits the scope of the analysis. We note that EPA does not adhere to this principle in preparing regulatory impact analyses, which frequently apply risk functions from one or several cities to the national population.

Use of 2004 air quality data, a year with relatively high ozone concentrations in many though not all regions of the country, is another factor that skews the risk estimates downward.

Risk Assessment Excludes Health Endpoints

The EPA Risk Assessment systematically underestimates risk by excluding health endpoints and important sensitive subpopulations. The following endpoints are included in the EPA's quantitative analyses:

- Lung function decrements (i.e., $\geq 15\%$ and $\geq 20\%$ reductions in FEV1) in all school age children for 12 urban areas;
- Lung function decrements (i.e., $\geq 10\%$ and $\geq 20\%$ reductions in FEV1) in asthmatic school age children for 5 urban areas (a subset of the 12 urban areas);
- Respiratory symptoms (i.e., chest tightness, shortness of breath, wheeze) in moderate to severe asthmatic children for the Boston area;
- Respiratory-related hospital admissions for 3 urban areas;
- Non-accidental and cardiorespiratory mortality for 12 urban areas.¹⁶¹

Several important health endpoints of public health importance were not quantified hence the risk assessment underestimates risks. Table 15, excerpted from the Staff Paper, identifies eight additional health endpoints associated with ozone exposure that were not quantified, noting that the list is not intended to be comprehensive.

¹⁶⁰ White House Office of Management and Budget, Bulletin No. 07-01 Update of Statistical Area Definitions and Guidance on Their Uses. December 18, 2006.

¹⁶¹ U.S. EPA. 2007. Staff Paper, p. 6-29.

Table 5-2. Health Endpoints and Associated Population Groups Not Included in the Quantitative Risk Assessment*

Health Effect	Population
Lung function decrements	Adults (outdoor workers, recreational exercisers, athletes)
Respiratory symptoms (cough, chest discomfort)	Adults (outdoor workers, recreational exercisers, athletes)
School absences for respiratory illness	Children
Asthma-related emergency department visits	Asthmatics
Doctors visits	Adults and children
Lung inflammation	Adults and children
Increased medication usage	Asthmatic children and adults
Decreased resistance to infection, impaired host defense	Adults and children

*The list of health endpoints and populations not included in the risk assessment is not a comprehensive list, but rather provides a general indication of the types of health endpoints that are associated with exposures to ozone but not included in the quantitative risk assessment.

Table 10: Health Endpoints and Associated Population Groups Not Included in the Quantitative Risk Assessment; From U.S. EPA, 2007, Staff Paper, p. 5-10.

A number of the health effects identified in Table10 have been quantified in other analyses, including EPA’s Regulatory Impact Assessment, and in the benefits assessment conducted by California for the review of the state ambient air quality standard for ozone, both of which used EPA’s BenMAP model.¹⁶² Thus the methodology for quantitatively assessing these other health endpoints is well established.

Among the other health endpoints that were excluded and exacerbate the limitations of the risk assessment include health effects resulting from chronic ozone exposures, and cardiovascular health effects. The number of individuals affected by these endpoints would significantly increase the estimated human toll of ozone. And since the quantifiable health effects form the basis for the assessment of benefits, these gaps in the risk assessment ultimately translate into underestimates in the health benefits of improved air quality.

Risk Assessment Excludes Vulnerable Populations

According to the National Research Council, “...estimates of individual risk are generally developed to address concerns for the most vulnerable people in a population -- who, almost by definition, lie at the tails of the probability distribution. To protect the entire population, one

¹⁶² <http://www.arb.ca.gov/research/aaqs/ozone-rs/rev-staff/rev-staff.htm>

often evaluates the risk to the most vulnerable.”¹⁶³ The EPA has failed to consider some of the most vulnerable populations in the assessment of ozone risks.

By vulnerable populations we mean those individuals who exhibit increased susceptibility to the effects of ozone due to biological or intrinsic factors as well as those individuals that may be vulnerable due to higher or more frequent ozone exposures. The EPA risk assessment falls short on both of these counts, excluding key subpopulations that are either more susceptible to ozone health effects, experience greater exposure, or both. In particular, some specific populations not quantitatively assessed include children less than five years of age, active children, outdoor workers, and senior citizens.

There is scientific evidence that the current ozone standard is inadequate to protect infants and children. For example, in a recent study Triche et al. conclude that “at levels of ozone exposure near or below current U.S. EPA standards, infants are at increased risk of respiratory symptoms, particularly infants whose mothers have physician-diagnosed asthma.”¹⁶⁴ Additionally, prenatal exposures have been documented to correlate with lower birth weight and intrauterine growth retardation.¹⁶⁵ This is of particular concern as birth weight is an important determinant of later risks of morbidity and mortality.

There is also evidence of a pronounced relationship between daily mortality and ozone exposure in elderly,¹⁶⁶ and that ozone exposure increases hospital admission rates in the elderly.

In addition to the outdoor workers discussed below, recreational exercising adults and children will experience increased ozone exposure due to increased breathing rates.¹⁶⁷ 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.¹⁶⁸

¹⁶³ National Research Council. 2007. Scientific Review of the Proposed Risk Assessment Bulletin from the Office of Management and Budget. National Academies Press: Washington DC

¹⁶⁴ Triche EW, Gent JF, Holford TR, Belanger K, Bracken MB, Beckett WS, Naeher L, McSharry JE, Leaderer BP. Low-level ozone exposure and respiratory symptoms in infants. *Environ Health Perspect* 2006; 114: 911-916.

¹⁶⁵ Salam MT, Millstein J, Li YF, Lurmann FW, Margolis HG, Gilliland FD. Birth outcomes and prenatal exposure to ozone, carbon monoxide, and particulate matter: results from the Children's Health Study. *Environ Health Perspect* 2005; 113: 1638-1644.

¹⁶⁶ Cakmak S, Dales RE, Vidal CB. Air pollution and mortality in Chile: susceptibility among the elderly. *Environ Health Perspect* 2007; 115: 524-527.

¹⁶⁷ Brunekreef B, Hoek G, Breugelmans O, Leentvaar M. Respiratory Effects of Low-level Photochemical Air Pollution in Amateur Cyclists. *Am J Respir Crit Care Med* 1994; 150: 962-966.

Spektor DM, Lippmann M, Thurston GD, Liou PJ, Stecko J, O'Connor G, Garshick, E, Speizer FE, Hayes C. Effects of Ambient Ozone on Respiratory Function in Healthy Adults Exercising Outdoors. *Am Rev Respir Dis* 1988; 138: 821-828.

Kinney PL, Nilsen DM, Lippmann M, Brescia M, Gordon T, McGovern T, Fawal HE, Devlin RB, Rom WN. Biomarkers of lung inflammation in recreational joggers exposed to ozone. *Am J Respir Crit Care Med* 1996; 154: 1430-1435.

¹⁶⁸ McConnell R, Berhane K, Gilliland FD, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. Asthma in Exercising Children Exposed to Ozone. *The Lancet* 2002; 359: 386 -391.

The Importance of Protecting 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,¹⁶⁹ mail carriers,¹⁷⁰ and others.¹⁷¹ The Exposure Assessment and Risk Assessment completely ignore health risks to outdoor workers, a population that is exposed to ambient ozone while under exertion. 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 11 indexes some categories of outdoor workers and provides estimates of population size. Note that this tabulation does not include members of the military forces.

¹⁶⁹ Brauer M; Blair J; Vedal S. Effect of ambient ozone exposure on lung function in farm workers. *Am J Respir Crit. Care Med* 1996; 154: 981-987.

¹⁷⁰ Chan C-C, Wu T-H. 2005. Effects of ambient ozone exposure on mail carriers' peak expiratory flow rates. *Environ Health Perspect* 2005; 113: 735-738.

¹⁷¹ Tovalin H, Valverde M, Morandi MT, Blanco S, Whitehead L, Rojas E. DNA damage in outdoor workers occupationally exposed to environmental air pollutants. *Occup Environ Med* 2006; 63: 230-236.
O'Neill MS, Ramirez-Aguilar M, Meneses-Gonzalez F, Hernández-Avila M, Geyh AS, Sienra-Monge JJ, Romieu I. Ozone exposure among Mexico City outdoor workers. *J Air Waste Manag Assoc* 2003; 53: 339-346.

Occupations	Number of workers
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

Occupations	Number of workers
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 WOKERS	8,939,670

Table 11: Census 2000 Worker Counts for Occupations likely to Involve Outdoor Work

Source: Environmental Defense derived from Census 2000, Census 2000 EEO Data Tool,

<http://www.census.gov/eo2000/index.html>.¹⁷²

Risk Function for Short-Term Mortality Understates Effects

The EPA Staff Paper characterizes the evidence supporting the association between short-term exposures to ozone and premature mortality as “robust and credible.”¹⁷³ The CASAC found that new studies have provided evidence for an increase in mortality associated with ozone exposure levels well below the current standard.¹⁷⁴ The inclusion of mortality estimates based on exposure-response functions derived from Bell et al. 2004 is a positive addition to the EPA ozone risk assessment.

However, the risk assessment likely underestimates ozone-related premature mortality because the assessment is solely based on NMMAPS (National Morbidity, Mortality, and Air Pollution

¹⁷² The Census Bureau tabulation excludes the four military categories and 35 occupation categories that fall below a 10,000 person threshold.

¹⁷³ Staff Paper p. 6-14.

¹⁷⁴ Dr. Rogene Henderson, Chair, CASAC, to Stephen Johnson, EPA Administrator, “Clean Air Scientific Advisory Committee’s (CASAC) Peer Review of the Agency’s 2nd Draft Ozone Staff Paper,” (Oct. 24, 2006). p. 4.

Study) data that likely understate the magnitude of the increased risk of premature mortality due to ozone exposure. The NMMAPS study design includes a large number of degrees of freedom in the model that diminish the observed association. The use of the three meta-analyses of ozone-mortality effects would be more representative because they rely on a synthesis of results from numerous studies. These meta-analyses were commissioned by EPA specifically for use in benefits analyses.

Comments on Updated Regulatory Impact Assessment

Executive Order 12866 requires EPA to prepare a Regulatory Impact Assessment (RIA); however, under the Clean Air Act, cost-benefit considerations are precluded from consideration in decision-making about revision of the ozone standards. The RIA is most valuable in highlighting the health impacts of ozone, as well as the relative health benefits of the alternate standards under consideration. As part of the reanalysis of the 2008 decision, EPA has published a Supplemental Analysis of the Regulatory Impact Assessment.¹⁷⁵

The Supplemental Analysis detailing the results of the updated Regulatory Impact Analysis reflects several changes to the benefits calculations:

- The assumption of no causality for ozone mortality, was removed, as recommended by the National Academy of Science (NAS).¹⁷⁶
- Two additional more ozone multi-city studies were included, per NAS recommendation.
- Concentration-response function thresholds were removed for PM_{2.5}, consistent with EPA's Integrated Science Assessment for Particulate Matter.

Additionally, the benefits calculations were extended to lower alternative ozone standards.

The adjustments that impact the assessment of premature mortality are particularly welcome. According to the Staff Paper, the association between short-term ozone exposure and premature mortality is "robust and credible."¹⁷⁷ In its initial assessment of ozone-related premature mortality, EPA acknowledged this association yet retained consideration of the assumption of no causality in the RIA. Since the publication of the Final RIA, the National Research Council has affirmed the strength of the association between ozone and premature mortality.¹⁷⁸ In response, EPA has now removed the assumption of no causality from the updated RIA, a move that we applaud. This addresses a significant criticism that we raised in our previous comments.

¹⁷⁵ EPA. 2009. Supplemental Analysis, Regulatory Impact Assessment.

¹⁷⁶ National Research Council (NRC). 2008. Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution. National Academies Press. Washington, DC.

¹⁷⁷ Staff Paper, p. 6-14.

¹⁷⁸ National Research Council (NRC). 2008. Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution. National Academies Press. Washington, DC.

The report by a committee of the National Academy of Sciences (NAS) confirmed that short-term exposure to ozone smog is likely to contribute to premature deaths.¹⁷⁹

The EPA asked the committee to resolve a controversy about whether it should include the benefits of mortality reductions in evaluating air pollution control regulations aimed at controlling smog. The NAS report resoundingly concluded that the health-benefits estimates “should give little or no weight to the assumption that there is no causal association between estimated reductions in premature mortality and reduced ozone exposure.” In other words, EPA should consider the relationship between ozone and premature deaths to be real, and should calculate the benefits of saved lives due to reductions in ozone air pollution.

This is a powerful consensus statement from a panel of the National Academy of Sciences.

In light of this report it is significant to note that EPA’s revised Regulatory Impact Analysis estimates that by 2020, a standard of 60 ppb would prevent more than five times as many premature deaths from ozone and PM_{2.5} compared to the current standard of 75 ppb.¹⁸⁰

We will comment briefly on several important aspects of the draft RIA.

The estimate of particulate matter co-benefits for mortality is based on a synthesis of studies and is presented in terms of a range of estimates.

To calculate the ozone reduction benefits using only Bell et al. 2004, rather than presenting benefits as a range reflecting the diversity of estimates present in the literature, may understate ozone benefits from the implementation of reduction strategies for alternative standard scenarios.

In addition, the rollback approach used to estimate the benefits of the attainment of a given standard is likely to underestimate benefits. The precursor emission controls that must be implemented to reduce ozone at the highest monitor would likely lower ozone concentrations at all downwind monitors.¹⁸¹

The calculations in the Supplemental RIA on ozone-only impacts, presented in Table 12 below, demonstrate that the 70 ppb standard would leave over a million people unprotected from the hazards of ozone exposure, compared to a standard of 60 ppb. A standard of 60 ppb presents significant public health advances, reducing health impacts in over a million people.

¹⁷⁹ National Research Council (2008). Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution. Washington, DC: National Academy Press.

¹⁸⁰ U.S. EPA, Summary of the updated Regulatory Impact Analysis (RIA) for the Reconsideration of the 2008 Ozone National Ambient Air Quality Standard (NAAQS), January 2010.

¹⁸¹ RIA, p. 6-31

Ozone-Specific Health Impacts	Estimated Decrease in Adverse Effects Alternative Standards		
HEALTH ENDPOINT	60 ppb	65 ppb	70 ppb
Mortality	890-4,000	450-2,100	250-1,100
Hospital Admissions (Ages 0-1)	5,600	3,000	1,700
Hospital Admissions (Ages 65-99)	6,900	3,400	1,900
Emergency Department Visits (Asthma)	3,600	1,800	990
School Absences	2,100,000	1,100,000	600,000

Table 12: Projected Benefits (Decrease in Health Impacts) under Alternative Standards.

Source: Environmental Defense Fund derived from Supplemental RIA, Tables S2.10 and S3.4.

Since the same precursors that are responsible for the formation of ozone also contribute to the formation of particulate matter of an aerodynamic diameter less than or equal to 2.5 microns (PM_{2.5})¹⁸² consideration of PM_{2.5}-associated impacts is a critical component of the benefits analysis. PM_{2.5} is associated with a broad array of adverse health effects, some of which overlap with those associated with ozone, while others appear to be unique.

The reduction in NO_x and hydrocarbon emissions and subsequent decrease in ozone formation contribute to impressive co-benefits in terms of PM_{2.5} reductions and the associated health impacts. It is entirely appropriate to include these co-benefits in the RIA for the ozone NAAQS as EPA has done. As illustrated in Table 13 the combined value – in terms of lives saved – is staggering. EPA estimates that full attainment of an ozone standard of 60 ppb (with the exception of some regions in California) would result in as many as 12,000 lives saved.

ALTERNATIVE O ₃ STANDARDS	SYNTHESIS OF BELL ET AL. 2005, ITO ET AL. 2005, AND LEVY ET AL. 2005 O ₃ + PM _{2.5} BENEFITS
60 ppb	4000 – 12,000
65 ppb	2,500 – 7,200
70 ppb	1,500 – 4,300
75 ppb	760 – 2,100

Table 13: EPA Estimates of Ozone and PM_{2.5} Co-Benefits Associated with Declines in Ozone Data Source: U.S. EPA. 2009. Updated RIA Supplemental Analysis. S1.2

¹⁸² U.S. EPA. 2005. ACQD PM. p. 2-2.

These co-benefits are also displayed below in Figure Q, which clearly illustrates the enormity of the benefits that would be obtained with the selection of 60 ppb (0.060 ppm) as the revised ozone standard. Five times as many premature deaths could be averted with a standard of 60 ppb compared to the current standard of 75 ppb.

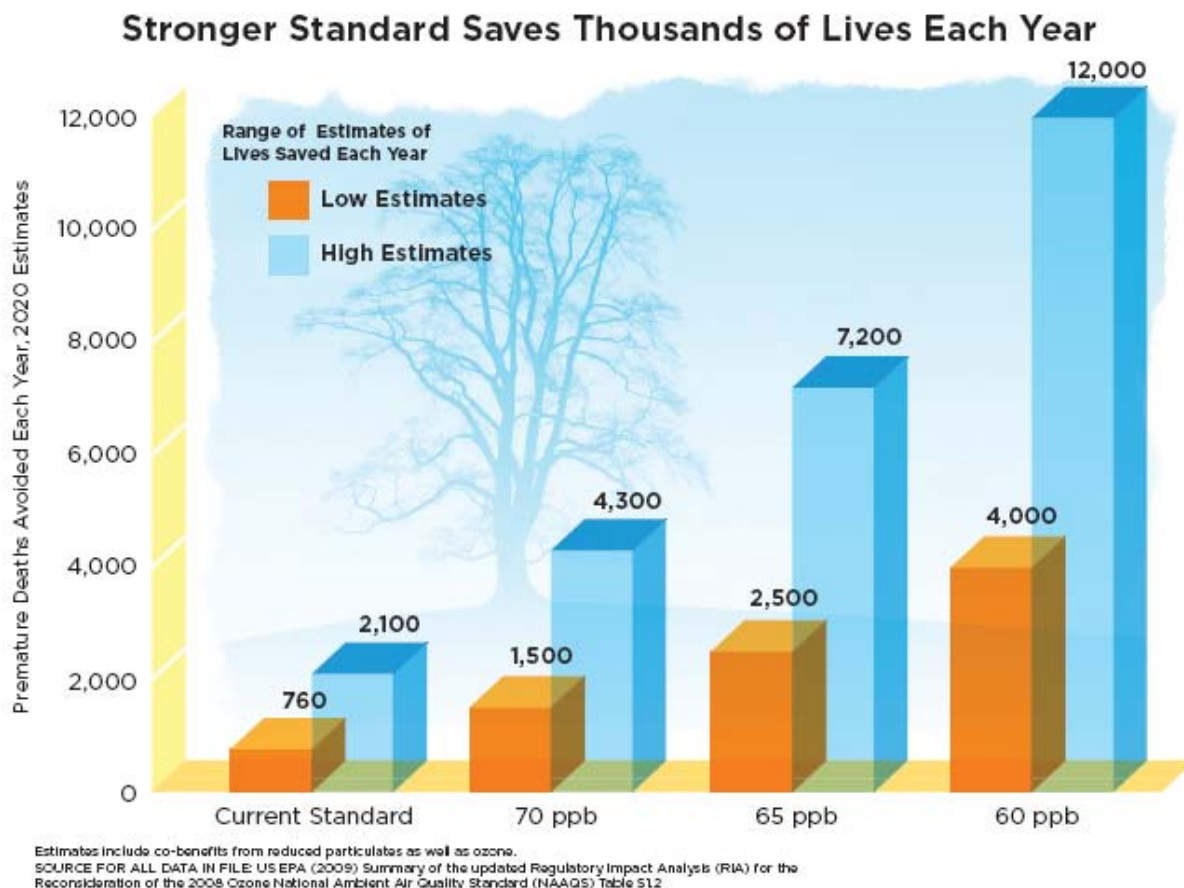


Figure Q: Source: U.S. EPA. 2009. Summary of the updated RIA for the Reconsideration of the 2008 Ozone NAAQS.

In addition to reductions in premature mortality, significant benefits will be obtained in terms of reductions in morbidity. The 2007 EPA Staff Paper identifies evidence for positive and robust associations between ambient ozone concentrations and respiratory hospital admissions, asthma emergency department visits, and respiratory symptoms and lung function effects in asthmatic children.¹⁸³

The same precursors that are responsible for the formation of ozone also contribute to the formation of particulate matter of an aerodynamic diameter less than or equal to 2.5 microns (PM_{2.5}).¹⁸⁴ PM_{2.5} is associated with a broad array of adverse health effects, some of which overlap with those associated with ozone, while others appear to be unique.

¹⁸³ U.S. EPA. 2007. Ozone Staff Paper. Page 3-36.

¹⁸⁴ U.S. EPA. 2005. ACQD PM. p. 2-2.

The reduction in NO_x and hydrocarbon emissions and subsequent decrease in ozone formation contribute to impressive co-benefits in terms of PM_{2.5} reductions and the associated health impacts. It is entirely appropriate to include these co-benefits in the RIA for the ozone NAAQS as EPA has done.

Figure R compares the benefits of combined reductions in both hospital admissions and emergency department admissions under alternate ozone standards, demonstrating the superior benefits of the 60 ppb ozone standard. Based on EPA's assessment, a standard of 60 ppb would prevent nine times as many hospital and emergency department visits compared to the current standard of 75 ppb.¹⁸⁵

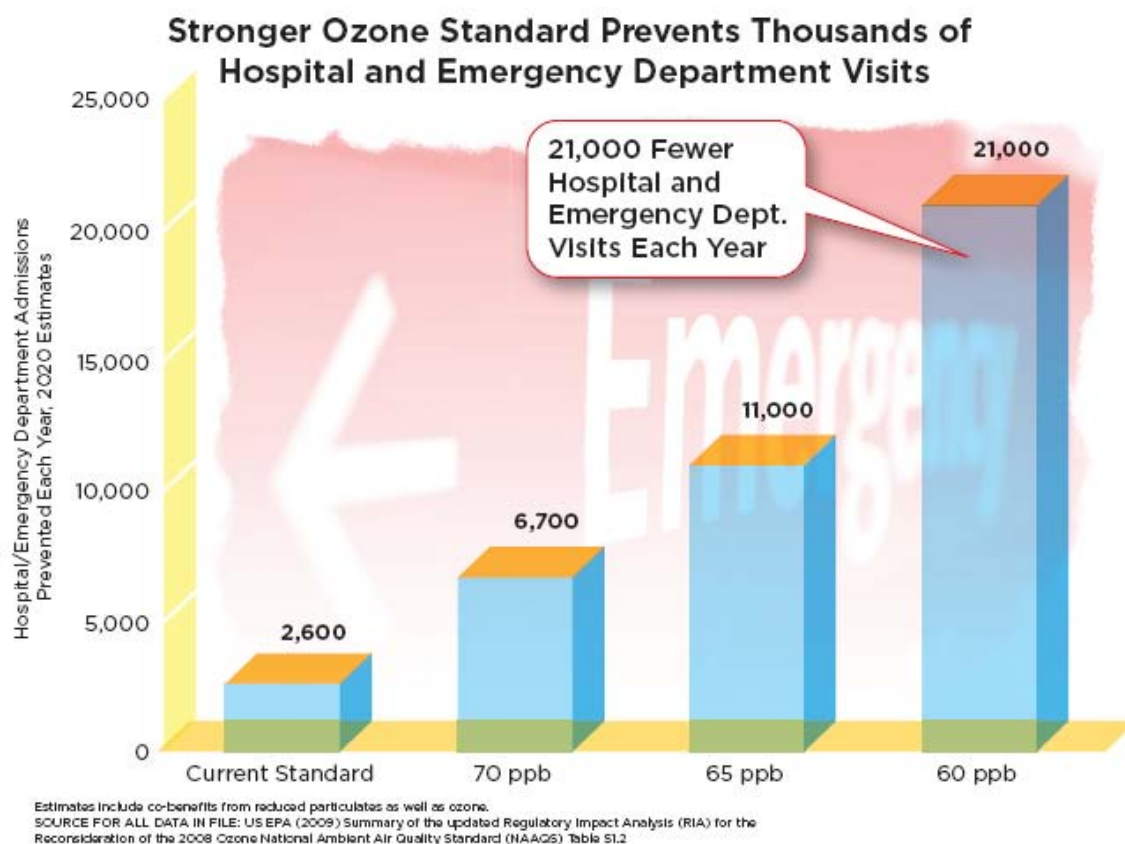


Figure R: Source: U.S. EPA. 2009. Summary of the updated RIA for the Reconsideration of the 2008 Ozone NAAQS

¹⁸⁵ U.S. EPA. 2009. Supplemental Analysis of the RIA, Table S2.11, addition of estimated benefits for hospital admissions and emergency room visits.

Similarly, Figure S compares the expected reduction of asthma exacerbations under alternate ozone standards, once again demonstrating the superior benefits of the 60 ppb ozone standard. A standard of 60 ppb would prevent nearly five times as many asthma attacks each year, compared to the current standard of 75 ppb.¹⁸⁶

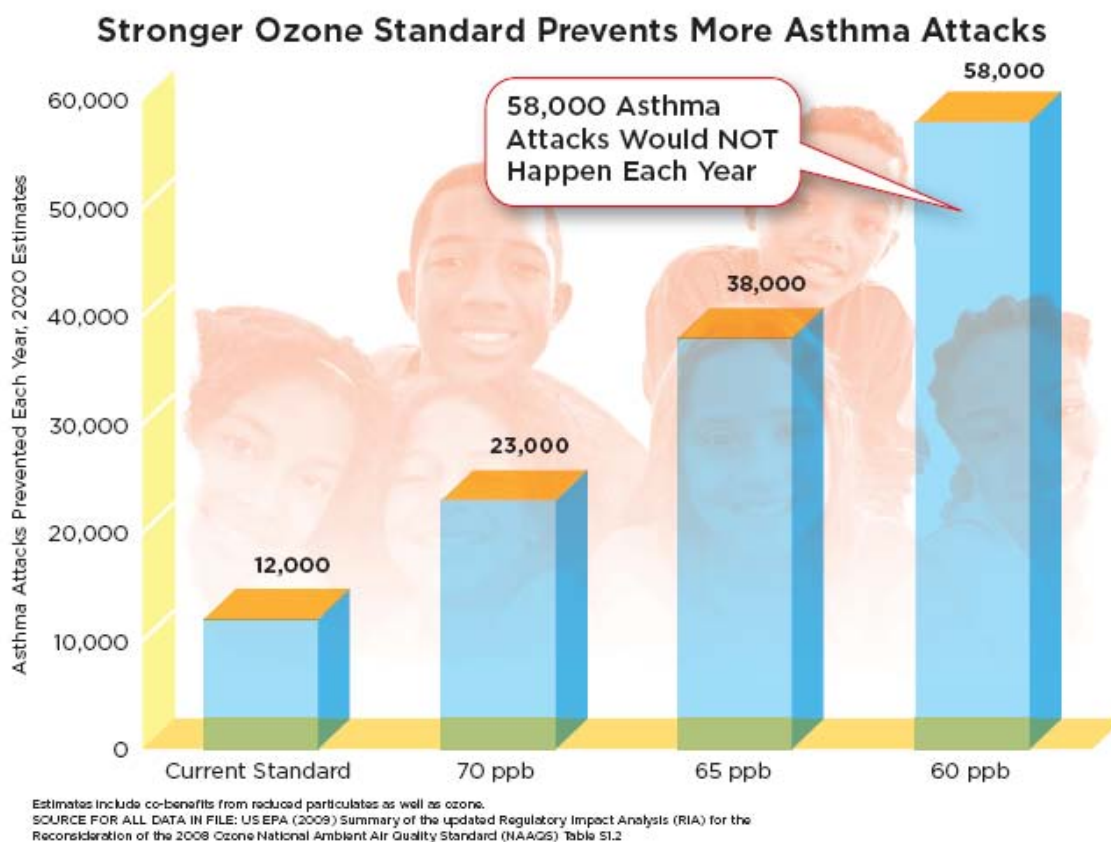


Figure S: Source: U.S. EPA. 2009. Summary of the updated RIA for the Reconsideration of the 2008 Ozone NAAQS

¹⁸⁶ EPA 2009. Supplemental Analysis of the RIA, Table S2.11, addition of estimated benefits for hospital admissions and emergency room visits.

Figure T compares the expected reduction in heart attacks (acute myocardial infarction) under alternate ozone standards. The selection of the 60 ppb ozone standard would prevent four times more heart attacks than the current standard of 75 ppb.¹⁸⁷

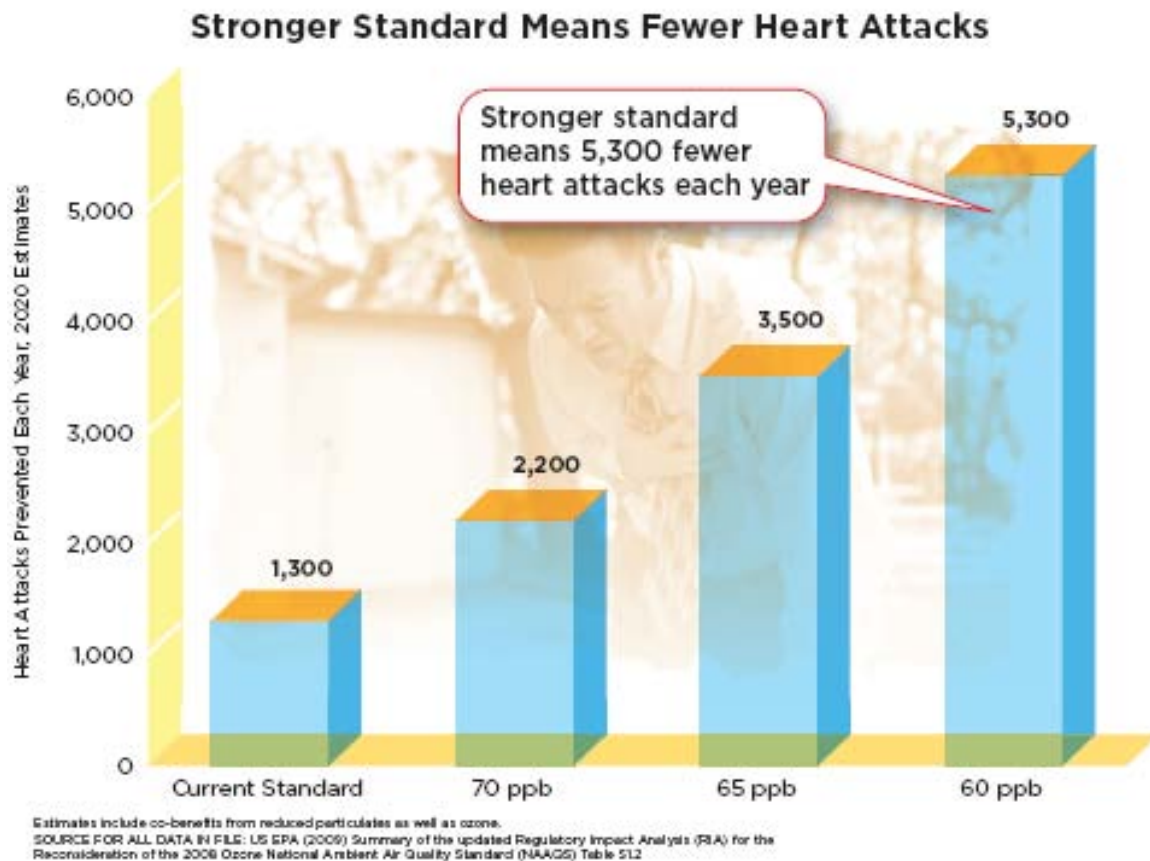


Figure T: Source: U.S. EPA 2009. Summary of the updated RIA for the Reconsideration of the 2008 Ozone NAAQS

¹⁸⁷ EPA 2009. Supplemental Analysis of the RIA, Table S2.11, addition of estimated benefits for hospital admissions and emergency room visits.

Morbidity associated with ozone results in a surprising number of school and work loss days, which is costly to our society in terms of lost productivity. Figure U compares the expected reduction in such loss of school and work days under alternate ozone standards. The selection of the 60 ppb ozone standard confers clear benefits in terms of decreased loss of school and work days.¹⁸⁸

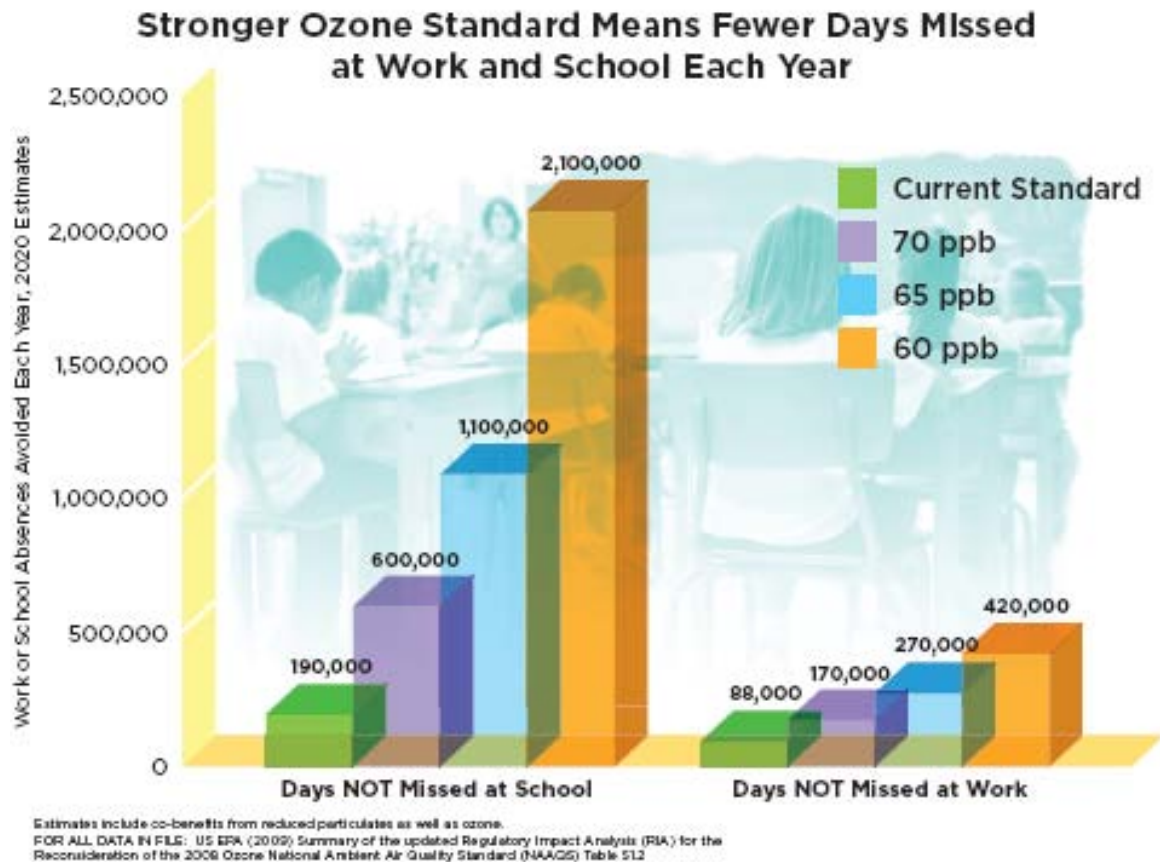


Figure U: Source: U.S. EPA. 2009. Summary of the updated RIA for the Reconsideration of the 2008 Ozone NAAQS

¹⁸⁸ EPA 2009. Supplemental Analysis of the RIA, Table S2.11, addition of estimated benefits for hospital admissions and emergency room visits.

Data Reporting: Rounding versus Truncation

In the rulemaking that concluded in 2008, EPA decided to truncate measured ozone values at three decimal places. This decision marked a shift from the traditional practice of rounding to two decimal places, in recognition that the typical incremental sensitivity of current ozone monitors is 0.001 ppm. The EPA is now proposing that “(1) 1-hour concentrations continue to be reported to only three decimal places, the same as is now specified in Appendix P, *i.e.*, that the current practice of truncation of the 1-hour data to the nearest 0.001 ppm be retained; (2) all digits resulting from the calculation of 8-hour averages be retained; and (3) the three-year average of annual fourth highest daily maximum 8-hour concentrations be rounded to three decimal places before comparison to the NAAQS.”

We supported the 2008 decision because under the previous standard of 0.08 ppm, violations were not recorded until concentrations reach 0.085 ppm. This rounding convention allowed unhealthful concentrations of ozone to continue unabated. Importantly, the CASAC also recommended elimination of this “rounding loophole.”

“The CASAC further recommends that the ozone NAAQS should reflect the capability of current monitoring technology, which allows accurate measurement of ozone concentrations with a precision of parts per *billion*, or equivalently to the third decimal place on the parts per million scale. In addition, given that setting a level of the ozone standard to only two decimal places inherently reflects upward or downward “rounding,” *e.g.*, 0.07 ppm includes actual measurements from 0.0651 ppm to 0.0749 ppm, the CASAC chooses to express its recommended level, immediately below, to the third decimal place.”¹⁸⁹

EPA’s new proposal retains 3-decimal point reporting for the 1-hour data, based on the ability of most ozone monitors to register ozone to the nearest 0.001 ppm in their measurements. As this proposal is consistent with the changes instituted with the 2008 decision, we continue to support this approach to evaluating measured data.

The proposed changes pertain to the calculation of averages:

- For the calculation of the eight hour average, EPA would retain all digits;
- For the calculation of the three year average, which is based on the annual fourth highest daily maximum 8-hour concentrations, the calculation would be rounded to three decimal places before comparison to the NAAQS.

¹⁸⁹ Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee to Stephen L. Johnson, Administrator, U.S. Environmental Protection Agency, re Clean Air Scientific Advisory Committee’s (CASAC) Peer Review of the Agency’s 2nd Draft Ozone Staff Paper, EPA-CASAC-07-001, October 24, 2006.

We support this new proposal. We share the EPA's concern that the data lost by regular truncation of the monitor readings repeatedly underestimates exposures, or as EPA states, it creates a continual downward bias. We've seen too long how the rounding convention has created situations where the community was counted as meeting the standard merely because it could take advantage of rounding's inherent downward bias. Maintaining all available data to be retained despite truncation protects against that tendency. Furthermore, this approach is consistent with data interpretation procedures for other criteria pollutants. Finally, the value that will be compared to the NAAQS will still contain three decimal places.

The Protectiveness of the Proposed Standard is Linked to the Form of the Standard

EPA has proposed to lower the level of the ozone standard, without altering the form of the standard. Consequently, our comments do not discuss in detail issues related to the form of the standard. The health-protectiveness of the ozone NAAQS is as much a function of its form as its level. Our comments are predicated on the assumption that EPA will not relax the form of the standards in conjunction with lowering the level.

Monitoring Requirements

Monitoring Requirements Must be Extended

EPA has not proposed any changes to the ozone monitoring requirements, but invites comment on several issues.

One issue of particular concern relates to the ozone monitoring network requirements for urban areas. At present, EPA uses a breakpoint of 85 percent of the standard to determine if an ozone monitor is required in an MSA. (It is unclear how EPA determines if an area would be below the 85 percent threshold without monitoring.) This requirement is sorely inadequate. For instance, for the current EPA standard of 0.075 ppm, monitoring is not required in areas with ozone concentrations of 0.064 or below. With the proposed standard of 70 ppb, 85 percent would be 60 ppb, which is also in the proposed range.

It is important to monitor even where concentrations are well below the current standard. First, adverse health effects have been reported concentrations far below 0.060 ppm in numerous studies. Second, people have a right to know what concentrations of ozone they are breathing. Third, monitoring of background areas assumed "clean" in the past have found that they were in violation of the standards and have helped to shape our knowledge of the extent of ozone exposure problems. And fourth, monitoring is vital to enable epidemiological studies of health effects at low concentrations. Failing to require monitoring in areas with projected concentrations less than 85 percent of the standard is arbitrary and irrational.

While some additional monitoring may be triggered by lowering the ozone standard as proposed, EPA must also revisit the monitoring regulations to eliminate or drastically lower the 85 percent breakpoint, to fifty percent, or below.

Secondly, existing minimum monitoring requirements are inadequate for MSAs with populations less than 350,000. The FR notice indicates that 100 such MSAs are without any ozone monitors. EPA cannot assume for regulatory purposes that these areas meet or almost meet the standard. Further, there are no requirements for ozone monitoring in areas outside MSAs or in rural areas. People living in smaller cities and rural areas are as deserving of monitoring and protection as those who live in large urban areas. Given the nature of ozone transport, these areas that lie downwind of urban areas may be experiencing even higher concentrations than the locations where the ozone is generated.

The ambient air quality monitoring network funded and operated by EPA and the states is critically important to research and regulatory programs deserves the highest priority in terms of resources.

EPA must revise its monitoring regulations and greatly increase the resources allocated to monitoring of ambient ozone concentrations.

Length of monitoring season

EPA proposes to leave the length of ozone monitoring seasons unchanged from those proposed to implement the 2008 standard of 0.075 ppm. 75 Fed. Reg. at 3036. EPA's decision is unreasonable.

EPA points to the analysis prepared to support the 2009 ozone monitoring proposal. 75 Fed. Reg. at 3035-36. In that analysis, EPA looked at the frequency of observed occurrences of 8-hour ozone levels of 0.060 ppm or higher. EPA chose 0.060 ppm as the cutoff because it represented 80 percent of the 0.075 standard, which EPA claimed was reasonable to ensure monitoring would provide an alert to potential exceedances as well as enable warnings to those unusually sensitive to ozone exposures. *See id.* at 3036. The 2009 analysis found that ozone levels above 0.060 ppm outside of the previously defined ozone seasons occurred very frequently in a total of 32 states, "with some States experiencing between 31 to 46 out-of-season days during 2004 to 2006 at a high percentage of all operating year-round monitors." *Id.*

EPA now proposes to use this same monitoring season even with a standard as low as 0.060 and to abandon the previous objective of ensuring that monitoring can provide an alert of potential exceedances or inform sensitive individuals. 75 Fed. Reg. at 3036. EPA does not explain why providing a broader monitoring envelope around the period of concern is no longer reasonable. EPA's only rationale is that "the traditional practice had been to base the length of required ozone monitoring seasons on the likelihood of measuring exceedances of the level of the NAAQS." *Id.*

The first problem with EPA's rationale is that the limited analysis relied upon for the 2009 proposal does not in fact ensure that monitoring will occur during all periods likely to experience

an exceedance. Looking only at three years of data does not provide a statistically significant prediction of when exceedances of a lower standard are likely to occur. The statistical sample is further limited by the fact that EPA only considered areas with year-round ozone monitors. Defining the monitoring season based on this limited data with no margin to account for the uncertainty associated with these limitations is simply unreasonable. This narrow sampling is of particular concern in a world with changing climate patterns that will likely expand ozone seasons in many areas. *See* 74 Fed. Reg. 18886, 18901 (April 24, 2009) (proposed endangerment finding).

The second problem with EPA's rationale is that it undermines the other stated values associated with expanded monitoring seasons. In addition to providing the alerts of potential exceedances and informing the public on potential health threats below the level of the NAAQS, requiring monitoring during periods immediately before and after the season of most concern is vital to enable epidemiological studies of health effects from ozone concentrations below *and above* the NAAQS. By ensuring that the "run up" period is monitored, studies will be better able to identify potential thresholds of concern.

We urge EPA to reinstate the 80 percent cutoff previously proposed and to define the ozone monitoring season based on the period when concentrations above 0.048 ppm have been measured. This will provide a margin of safety to ensure that all exceedances are measured and also provide benefits for informing the public and future studies.

Data Completeness Requirements

Data completeness over the 8-hour period

EPA has proposed data substitution options for monitoring sites that collect less than 6 hours of valid ozone concentrations. 75 Fed. Reg. at 3029. EPA's proposed approach would allow the substitution of either the lowest hourly average concentration observed for that hour of the day on any day during the ozone monitoring season or one-half of the method detection limit, whichever is higher. *Id.* EPA's proposed approach is an improvement over the existing instructions which use only the latter method detection limit substitution. Both of these data substitutions, however, are too heavily weighted to avoiding false positive determinations of an exceedance. The data allowed to be substituted for the missing hours has no reasonable connection to the likely ozone levels occurring at the location of the monitor during the period the monitor is down.

A better approach that is still conservative but has at least some relationship to actual ozone levels near the monitor on a given day would be to assign to the missing hours the average of the actual, valid concentration readings immediately before and after the gap (*i.e.*, interpolate to fill in missing data). Thus where a monitoring station has readings for hours 3 and 5, the ozone concentration substituted for hour 4 would be halfway between. If the gap is for more than one hour, the same average value could be assigned to all of the missing hours reflecting a smoothing

of any peaks, which should ensure the results are conservative and would avoid false positive determinations of exceedances.

It is not reasonable to assume that the intervening hours would see a precipitous drop to the lowest reading ever measured during that hour over the course of the ozone season or to one-half of the method detection limit. Given the influence of temperature on ozone formation, there is no reason to expect that ozone concentrations at 2:00 pm in mid-May will be a reasonable predictor for the missing concentrations at 2:00 pm in August.

EPA must choose a mechanism for filling missing data that has a reasonable connection to predicting the concentrations that were likely missed by the monitor. While commenters agree that false positive determinations of exceedances should be avoided, we are also concerned about the effect of false negatives.

EPA has offered important safeguards to prevent areas from achieving attainment through failures of monitoring. We support EPA's conclusion that it is inappropriate to include those days with fewer than 6 hourly measurements in determining whether an area has attained the standard. The 8-hour averages calculated using data substitution will underestimate actual concentrations and do not demonstrate that air quality in fact meets the health-based standards.

EPA must also protect against underestimating the design value concentrations that are used in the modeling to develop attainment plans. Errors at this stage can result in years of wasted effort that fails to achieve required public health protections. Underestimates also result in false trends reporting, which can undermine vital health protection efforts. We believe that once an area has been designated nonattainment, data substitution procedures should be more concerned with avoiding underestimating concentrations. Not only will this avoid the problems noted above, it will also provide the appropriate incentive to state and local air quality agencies to maintain their monitors in order to minimize such gaps.

Data completeness over the ozone season

EPA's data adequacy provisions require that in order for a design value equal to or less than the standard to be valid, at least 75 percent of the ozone season days in each of the three years must have valid 8-hour average values (*i.e.*, must have at least 6 hourly readings of actual concentrations). *See* 75 Fed. Reg. at 3031. The provisions further require the percentage of valid days be at least 90 percent on average over the three-year period (*i.e.*, if one year only meets the 75 percent minimum requirement, the following two years must capture 97.5 days of the season in order to ensure a 90 percent average). *See id.* EPA proposes to eliminate the 90 percent requirement. EPA's rationale again appears to be based on the concern that "clean" areas will be unable to prove that they have attained. *Id.*

We do not agree that merely requiring 75 percent capture in an ozone season is sufficient to provide an accurate or reasoned showing of compliant air quality in an area. Allowing areas to miss 25 percent of the ozone season radically weakens the protection level assured by the standard. A full month at the peak of the ozone season could be missed. EPA must justify how such large gaps in air quality monitoring will ensure protection of public health.

EPA should require states and local air districts to be held accountable for the maintenance of air quality monitors. Commenters believe a minimum of 85 percent capture is reasonably achievable given proper maintenance. If EPA believes that requiring a high rate of capture is not possible, EPA should adjust the stringency of the standard to compensate for such gaps. For example, if an area is missing data for one in four days, instead of looking at the 4th highest 8-hour concentration, the area should look at the 3rd highest day.

In the end, any rationale for allowing areas to demonstrate attainment based on incomplete monitoring data must be rooted in a demonstration that public health will be protected with an adequate margin of safety. See CAA § 109(b)(1). Allowing incomplete data to be used for purposes of demonstrating attainment or designing attainment strategies effectively weakens the standard without a lawful or reasoned basis. EPA's choices on data adequacy must demonstrate that such choices reasonably assure that unsafe air quality conditions will not be allowed to persist.

Administrator's discretion

EPA proposes to give the Administrator general discretion to use incomplete data to calculate design values. Commenters object to such open-ended discretion. Any such discretion must be cabined to assure protection of public health. Thus, the Administrator should have no such discretion to expand the exemptions or otherwise lower the minimum data adequacy requirements in order to lift air quality protections. While circumstances may warrant EPA acting in the face of incomplete data to impose air quality requirements, additional discretion to lift such requirements would undermine the statutory obligation to demonstrate that the standards protect public health. Such revisions to the protection provided by the standard must be made through a public process and supported by the necessary factual rationale. EPA cannot show that the standard will be adequate under section 109(b)(1) of the Act if the standard is accompanied by open-ended discretion to find compliance based on whatever quality of data EPA deems sufficient.

Multiple monitors at a site

Commenters similarly object to the proposed open-ended discretion to choose (even retrospectively) which monitoring data to use from a site that has co-located monitors. If both monitors are adequately maintained, the highest 8-hour concentrations measured at the site should be used. A decision to use lower values must be based on a reasoned and compelling explanation as to why the lower value reflects a more accurate measurement of air quality.

Implementation Issues

Schedule for designations under primary standard

We support EPA's proposed accelerated schedule for designating areas with respect to the primary standards. We believe the proposed schedule to allow the designations to become effective within 1 year is reasonable and necessary to provide the public health protections that have been delayed by the previous administration's promulgation of an inadequate ozone standard. Much of the data will already have been collected for purposes of designations under the 2008 standard, and can be updated on an expedited schedule to reflect any new standard adopted with this rulemaking.

Conclusion: Standards Must be Strengthened to Protect Public Health

Taken together, the data from the health studies and the Risk Assessment clearly demonstrate that exposures to ozone at and below the current regulatory standard pose a significant health risk. The health evidence from multiple lines of research supports an 8-hour ozone standard of 60 ppb or lower. We therefore call upon EPA to adopt a more stringent standard of 60 ppb or lower, to protect the public health, including the health of sensitive populations, with an adequate margin of safety.