Comments of the

American Lung Association
Environmental Defense
Sierra Club
on the
U.S. Environmental Protection Agency's

Proposed Revisions to the
National Ambient Air Quality Standards for Ozone

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Executive Summary

The American Lung Association and Environmental Defense submit these comments on the need to strengthen the 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.

The revisions that the U.S. Environmental Protection Agency (EPA) proposed in the primary standards are a step in the right direction; however, the proposed range fails to provide the requisite protection for the health of the public. These recommendations fail to follow the science and would provide less protection than the unanimous recommendations of the Clean Air Scientific Advisory Committee (CASAC). Further, we oppose retaining the current standards, agreeing with the CASAC that there is "no scientific justification" for such a decision.

These comments outline our concerns with EPA's interpretation of the scientific record and the exposure and risk analyses, and make a compelling case for EPA to adopt far more protective final standards than proposed.

Specifically, we urge EPA to set an 8-hour average primary standard for ozone of 0.060 parts per million or below, using a form based on the third highest daily maximum.

At a minimum, we urge EPA to follow the advice of CASAC regarding the form and level of the standard and reject calls to maintain the current standard.

We support EPA's recommendation to express the standard in terms of three decimal places, but recommend rounding values after three decimal places rather than truncating them.

We call on EPA to reduce the number of allowable exceedances, from the average of the fourth highest reading over three years, to the third or lesser highest reading.

We urge EPA to reinstate the one-hour average primary standard for ozone.

In response to other issues raised in this proposal, we recommend the Agency adopt the following actions:

- institute a notice and comment rulemaking on revisions to the Air Quality Index for the purpose of considering more stringent break points;

- eliminate the monitoring exemption for areas where concentrations are predicted to be below 85 percent of the final standard; and

- provide reasonable estimates of mortality as a health endpoint in the final Regulatory Impact Analysis, that is, estimates that do not include zero.

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 a dozen analytical flaws that dispel EPA's argument that the "exposures of concern" analysis justifies the weak proposed standards. We show that EPA's reliance on uncertainty is belied by the substantial scientific evidence of adverse effects at low concentrations. 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, based on the third highest maximum concentration.

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. In addition, EPA must give due deference to the advice of an independent panel of scientific advisors, the 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 the Clean Air Act's requirements for controlling emissions of conventional air pollutants. Once EPA establishes a NAAQS, states and EPA identify those geographic areas that fail to meet the standards. 42 U.S.C. § 7407(d). Each state must prepare an "implementation plan" designed to 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.). In short, "[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, which EPA has now proposed to revise.

NAAQS Must Protect Public Health with an Adequate Margin of Safety

In setting or revising a NAAQS, Section 109 of the Clean Air Act requires that the EPA achieve one thing at minimum: protect public health with an adequate margin of safety. But other parts of the statute, as well as guidance from the court provide significant limitations on the discretion granted to EPA in selecting a level and form for the NAAQS. The following excerpt from an opinion of the U.S. Court of Appeals for the District of Columbia sums up EPA's mandate succinctly:

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

In implementing this mandate, EPA cannot deny protection against adverse health and welfare effects merely because those effects are confined to subgroups of the population or to persons especially sensitive to air pollution. It is inherent in NAAQS-setting that adverse effects are experienced by less than the entire population, and that we do not know in advance precisely which individuals will experience a given effect. 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").

[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.").

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 and welfare. 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 a standard that protects against potential health effects—not just those impacts that have been well established by science.

In the seminal case on the NAAQS, the D.C. Circuit found that Congress "specifically directed the Administrator to allow an adequate margin of safety to protect against effects which have not yet been uncovered by research and effects whose medical significance is a matter of disagreement." *Lead 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

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.

EPA Cannot Consider Economic Cost of Meeting NAAQS

In setting or revising NAAQS, EPA cannot consider the economic impact of the standard—only the impact on public health.

Lower courts had long held that costs could not be considered in setting NAAQS, and in 2001, the Supreme Court affirmed this position. Justice Scalia, writing for a unanimous Court, found that the plain language of the statute makes clear that economic costs cannot be considered: "Were it not for the hundreds of pages of briefing respondents have submitted on the issue, one would have thought it fairly clean 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's July 11, 2007 proposal notice quotes extensively from Justice Breyer's concurrence in *Whitman*. It is the language of the majority opinion that controls, not that of a concurrence.

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 CASAC and rationally explain any important departure from CASAC's recommendations. §§ 7409(d)(2)(B), 7607(d)(3). 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.

Proposed Rule is Unlawful and Arbitrary

In the context of EPA's current proposal for the primary standard,[2] EPA has taken a detour from the mission that Congress identified for the agency through the Clean Air Act, producing a proposal that is unlawfully and arbitrarily weak. In this proposal, EPA would adopt standards allowing large continuing adverse health affects affecting many thousands of Americans each year—including premature death and serious morbidity impacts such as hospitalization and asthma attacks.

[2] References to EPA's "proposal" herein are to the agency's proposal to revise the primary standard to a level within the range of 0.070 and 0.075 ppm. EPA is also accepting comment on alternative levels down to 0.060 ppm, and – as is evident below – we support a level of 0.060 ppm.

As further detailed below, numerous peer-reviewed studies document adverse health effects at 8-hour ozone levels well below 0.070 ppm, the lowest end of the range proposed by EPA for the primary standard. 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. EPA's proposed standards would allow these documented adverse effects to persist, and therefore would not be 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.

EPA tries to justify its proposal (as well as its rejection of CASAC's recommendation that the primary standard be set in the 0.060 - 0.070 ppm range) by asserting that health effects at lower ozone levels are less certain. However, the agency fails to provide facts or reasoned support for this claim. The agency concedes that at least one controlled human exposure study has shown statistically significant lung function decrements in healthy individuals at 0.060 ppm ozone levels. The record does not support a claim that associations shown between adverse health effects and ozone by epidemiological studies are "less certain" at ozone levels at or below 0.060 ppm. As further discussed below, there are a number of such studies, and the results do not show or find some sort of increasing scale of uncertainty at lower ozone levels. EPA provides no rational explanation for claiming that uncertainty is necessarily higher at lower ozone levels. Nor does EPA provide any rational basis for concluding that whatever uncertainty that may exist as to health effects at lower ozone levels (e.g., at 0.060 or 0.070 ppm) is so great as to render such health effects improbable. And even if uncertainties below 0.070 ppm are significant, EPA fails to address – as the Act requires – the need to provide an adequate margin of safety by 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 justification for discounting adverse health effects associated with ozone levels below 0.070 ppm and for rejecting CASAC's recommendation is arbitrary and unlawful. *State Farm*, 463 U.S. at 43; *American Lung Assn.*, 134 F.3d at 392.

Equally unsupported is EPA's claim that its choice of a standard in the 0.070 to 0.075 ppm range is purely a "policy judgment." EPA concedes that substantial reductions in public health risks would occur "throughout" this range, and the risk assessment and other studies show significant additional health benefits at the low (0.070 ppm) end of the range. Because there are expected adverse health effects at the low end of the range, EPA may not, consistent with the authorities cited above, set a standard higher than that low end. EPA asserts that it has discretion to exercise policy judgment to set the standard at the higher end of the range "because there is no bright line clearly directing the choice of level." 72 Fed. Reg. 37879. 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 simply does not give EPA the option of setting the standard at a level that allows adverse health effects to persist. Equally unhelpful is EPA's assertion that its "policy" choice of a level within the range will be made "considering the strengths and limitations of the evidence, and the appropriate inferences to be drawn from the evidence and the exposure and risk assessments." Id. This assertion provides no lawful or reasoned justification for adopting a standard above the low end of the range – i.e., a standard that will allow adverse health effects. EPA is using the risk

assessment as a way of illegally balancing costs and benefits. The purpose of the risk assessment was to compare options, not to determine what level of impacts are "acceptable."

Further, by failing to incorporate impacts on infants, young children, active children, outdoor workers and other sensitive populations into the standard setting analysis for ozone, even in light of relevant available data, EPA has walked away from a critical element of its statutory obligation. It is, by design, declining to protect part of the population – an outcome that is impermissible under the Clean Air Act. EPA must specifically address the health implications of any ozone limit that it selects for infant, children, outdoor workers, responders and other sensitive populations. Absent such analysis, EPA's standard setting process is fundamentally flawed and falls short of meeting its legal obligations.

Other rationales offered by EPA for rejecting a more protective standard are deficient as well. EPA asserts that the "most certain" evidence of adverse health effects from exposure to ozone comes from the clinical studies, and that the "large bulk" of this evidence derives from studies of exposures at levels of 0.080 ppm and above. As noted above, however, the Act does not allow EPA to limit itself to considering or protecting against only the "most certain" of adverse effects. Moreover, the fact that most clinical studies involved exposures of 0.080 ppm is hardly probative that adverse effects do not occur below 0.080 ppm, particularly given that there are clinical studies showing adverse effects below 0.080 ppm. EPA further asserts that the evidence of health effects in healthy individuals at the 0.060 ppm exposure level is "too limited," but does not provide a rational explanation as to why. Such conclusory assertions are insufficient to justify rejection of peer-reviewed studies showing actual health effects, particularly when the likelihood of health effects at that level is corroborated by other evidence, as further detailed below.

EPA also seeks to bolster its proposal by asserting that "a 0.070 ppm standard would be expected to provide protection from the exposures of concern that the Administrator has primarily focused on for over 98 percent of all and asthmatic school age children." This is double talk. Because EPA arbitrarily selected the "exposures of concern" that it "primarily focused on," the degree of protection provided against such exposures hardly provides a rational basis for finding that a 0.070 ppm standard is requisite to protect public health, or that exposures below 0.070 ppm do not adversely affect public health.

EPA Raises Unwarranted Issues in the Preamble, Compromising the Integrity of the Rulemaking Process

EPA (at 37881) cites various assertions by unidentified commenters that CASAC and Staff have misinterpreted the relevant data, that various studies "should not be used" in the risk assessment, that EPA "should not rely" on exposure studies showing adverse effects at 0.060 and 0.040 ppm exposure levels, and so on. These assertions provide no basis for retaining the existing standard, or for rejecting the overwhelming body of peer reviewed studies showing that the existing standard is inadequate. The assertions are not attributed to any scientifically credible individuals or organization, nor does EPA cite any data or reasoned analysis supporting them. Moreover, these assertions have been refuted through the thoroughly vetted and rigorous process of Criteria Document development and CASAC review.

EPA further cites (at 37881) concerns allegedly expressed by "several" Governors, state legislators and local officials about a more stringent standard, ostensibly related to "implementing policy that improve air quality while at the same time achieving economic and quality of life objectives." To the extent these commenters are asking EPA to consider potential economic or other non-health impacts of a revised NAAQS, such consideration is prohibited by the Act as construed by the Supreme Court in *Whitman*. Equally irrelevant are alleged concerns about moving forward on a revised standard when states are just beginning to implement current air quality standards, or concerns that a number of areas will have difficulty in achieving timely compliance with the current 8-hour standard. Potential difficulties in implementation whether with the current or revised NAAQS do not in any way justify adopting NAAQS that are less protective than requisite to protect public health with an adequate margin of safety, as the Act expressly mandates. Congress was well aware that some areas might have difficulty attaining the NAAQS (as evidenced by Part D of Title I), but it provided no exceptions to the mandate that the primary standards themselves be based solely on what is necessary to protect health.

Also irrelevant to setting the primary standard is the potential impact of renewable fuels use or mandates for renewable fuels on compliance with a stronger ozone standard. See 72 Fed. Reg. at 37881-82. EPA has no discretion under the Act to take such factors into account in setting the primary standard, which is to be based exclusively on the level of protection requisite to protect public health with an adequate margin of safety. The Energy Policy Act of 2005 does not qualify or amend the Clean Air Act's mandates in this regard, and well-settled principles of statutory construction preclude the Energy Policy Act from amending §109 of the Clean Air Act by implication. For similar reasons, EPA cannot rely on a desire to advance goals related to increased production and use of renewable energy as a basis for adopting a NAAQS that is less protective than necessary to protect public health from ozone with an adequate margin of safety. EPA asserts that renewable energy can have national security benefits, but Congress made no provision for incorporating national security considerations into the NAAQS setting process. Congress specifically provided for addressing national security considerations in other provisions of the Act (e.g. §110(f)), but not in the provisions governing the setting of NAAQS. EPA's suggestion that national

security interests or the desire to promote renewable energy might somehow justify a weaker ozone NAAQS is therefore unlawful and arbitrary.

EPA is also completely off base in suggesting (at 37882/1) that concerns asserted by Governors and other commenters "regarding various types of uncertainties" somehow justify inviting comment on whether it would appropriate to retain the existing ozone standard and delay considering modification of the 8-hour standard until the next NAAQS review. CASAC found that there was no scientific justification for retaining the existing standard, and EPA identifies nothing in the concerns asserted by Governors and other unidentified commenters that undermines CASAC's expert judgment on this matter. Suggestions that the current standard should be retained are completely out-of-step with mainstream scientific thinking and the conclusions of the Criteria Document and the Staff Paper, both of which were extensively vetted by CASAC. The use of specious arguments by the agency to imply that retaining the existing standard could be an acceptable option is arbitrary, irrational, and inconsistent with sound science.

The comments herein should be read in the context of the legal framework described above, and the objections to EPA's analyses and conclusions construed in light of the agency's legal obligations as they are here presented.

EPA Incorporated OMB's11th Hour Language Changes to Weaken the Proposed Rule

EPA was under a court-supervised deadline to issue its proposal regarding the ozone NAAQS by June 20, 2007. The public docket shows that on that day, the Office of Management and Budget (OMB) transmitted a series of inserts to EPA that altered, and materially weakened, the proposal in the following significant respects[3]:

OMB encouraged EPA to avoid the majority opinion of the Supreme Court.

The first page of the fax from OMB contains excerpts from Justice Breyer's concurring opinion in *Whitman v. American Trucking Assns, Inc.* OMB presents the language to EPA as the basis for the Agency to avoid the majority opinion of the United States Supreme Court. The explanatory language at the top of the fax states: "EPA could follow the direction of *a* Supreme Court Justice without fear of contempt, especially if (as OIRA pointed out) the EPA risk assessment finds little health improvement nationwide." Justice Breyer's language was in fact incorporated into the final proposal. 72 FR 37820. As noted above, Justice Breyer's concurrence is not, in fact, the controlling opinion in the case.

[3] OMB Interagency Fax, available at EPA-HQ-OAR-2005-0171-0215, p. 1, Appendix C (emphasis added).

OMB encouraged EPA to cite "uncertainties" to justify a weaker proposal.

The second page of this same fax from OMB contains language laying out the rationale for EPA to retain the current ozone health standard without changes based on a host of "uncertainties" provided by OMB. This OMB transmitted language, which was incorporated in substantial part in EPA's preamble, reads as follows: "The Administrator recognizes that there is a concern that adopting a more stringent 8-hour standard now, without a better understanding of the health effects associated with O_3 exposure at these lower levels, will have an uncertain public health payoff. These questions include uncertainty in (1) the exposure estimates, (2) the estimation of concentration-response associations in epi studies, (3) the potential role of co-pollutants in interpreting the reported associations in these epi studies, and 4) [sic] the effect of background concentrations. In fact, the Agency continues to undertake a substantial research program in an effort to clarify some of these uncertainties. As a result, the Administrator acknowledges the possibility that it would be appropriate to consider modifications of the 8-hour standard with a more complete body of information in hand rather than to initiative a change in the standard at this time." This language was incorporated in significant respects into the final proposal. 72 FR 37880. The OMB transmitted litany of uncertainties associated with health effects below the current standard is in direct contrast with CASAC's unwavering unanimous statements, recounted below, that there are a suite of adverse health effects below the current standard that compel EPA action and that there is no longer significant scientific uncertainty that the standard must be lowered.

OMB provided inappropriate arguments EPA adopted to justify inaction.

The final document in the fax from OMB to EPA invokes three separate strands of argument in seeking to buttress the case for inaction. First, the OMB language argues, paradoxically, that the sluggish implementation pace of the current ozone health-standard should delay a new health standard. Second, OMB maintains that the likely delays in achieving a more protective health standard preclude the Administrator from considering the health benefits of lower ozone and, therefore, lowering the health standard will not realize public health gains. Third, it is claimed that the nation's alternative fuels program may supersede the Administrator's duty to establish standards requisite to protect public health with an adequate margin of safety. On this latter point, the language expressly cross-references back to Justice Breyer's concurrence, thereby completing the circle with the first insertion above. The actual final language incorporated at OMB's behest provides: "The Administrator is mindful that the country has important goals related to the increase production and use of renewable energy, and that these new energy sources can have important public health, environmental and other benefits, such as national security benefits. In some contexts and situations, however, the use of renewable fuels may impact compliance with a lowered ozone NAAQS standard. For example, the Agency recently promulgated final regulations pursuant to section 211(o) of the Clean Air Act, which was enacted as part of the Energy Policy Act of 2005. This provision requires the use of 7.5 billion gallons of renewable fuel by 2012, a level which will be greatly exceeded in practice. In the Regulatory Impact Analysis which accompanied the renewable fuel regulations, the Agency recognized the impact of this program on emissions related to ozone, toxics and greenhouse gases and otherwise reviewed

the impacts on energy security. The Administrator requests comment on such factors and any relationship to this rulemaking, including the extent of EPA's discretion under the Clean Air Act to take such factors into account (see section I.A)." This final portion of the OMB fax was incorporated in large part at p. 37881 of the final proposal.

While the nation's interest in renewable fuels is well-understood, OMB's language inverts the public health protection mandate of the law. OMB's approach would supersede the statute's directive to establish NAAQS that protect public health with an adequate margin of safety for ozone, particulate pollution, lead or any other pollutant by invoking a favored industrial activity or process. In such an illogical world, emissions would inexorably rise as the nation's health standards are adjusted upward to accommodate more pollution.

The rushed OMB fax, which was belatedly inserted into EPA's formal proposal, provides an array of technical, policy and legal arguments designed to justify EPA inaction. OMB also pressed for inclusion of the language in the Administrator's own voice. In one revealing passage, the OMB transmitted fax asks whether it is "Possible to include as Administrator's voice or somewhere other than the five pages of input from 'commenters'?"

Not only do the OMB comments urge positions that are arbitrary and illegal, but they represent the views of an administrative office with no statutory role in the NAAQS adoption process, and no special expertise in the health and science issues presented in developing and adopting a NAAQS. Accordingly, to the extent that EPA's proposal relies on or incorporates OMB's positions on issues of health, science, or other matters requiring technical expertise, the proposal is entitled to no deference.

Scientific Consensus Supports Stricter Standards

In recent years, a broad scientific consensus has emerged that EPA's current air quality standards for ozone are not sufficient to protect public health, and that the level and form of the standards must be greatly strengthened.

This consensus is evidenced by the strong unanimous comments of the Clean Air Scientific Advisory Committee (CASAC), which is 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.

In the face of this strong consensus, it is untenable to cite "uncertainty" as a rationale for failing to propose tighter standards. Indeed, EPA mentions uncertainty no fewer than 190 times in the preamble, despite the 1,700 new studies published since EPA's last review.

EPA's claims that uncertainty justifies less protective standards than recommended by CASAC are both unfounded and one-sided. EPA's uncertainty claims lack rational support, and arbitrarily ignore uncertainties that favor more protective standards. For instance, controlled human exposure studies typically use healthy young adults as test subjects. This creates uncertainty about what the results would be on infants, or children, or children with severe respiratory disease. When Congress wrote the Clean Air Act, scientists testified that we would never have absolute knowledge: that we would learn more and improve our ability to assess dangers, but that we would always need to protect the public even when we lack full knowledge. Congress included a simple phrase in the Clean Air Act, in the requirements for setting standards, to direct the EPA to include an "adequate margin of safety" to provide a cushion of protection. The Clean Air Act requires that the EPA address such uncertainty in favor of more public health protection, not less.

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. Unfortunately, EPA's proposed standards are weaker than those recommended by CASAC, EPA staff scientists, the CHPAC, the World Health Organization, and numerous public health and medical organizations. We urge EPA to adopt final standards that follow the strong recommendations of the scientific and medical community.

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

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.[4] 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.

[4] 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.

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:[5]

- 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.

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.[6]

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

CASAC: The current standard fails to protect public health.

The CASAC panel repeatedly stated:

> "There is no scientific justification for retaining the current primary 8-hr NAAQS of 0.08 parts per million (ppm), and the primary 8-hr NAAQS needs to be substantially reduced to protect human health, particularly in sensitive populations."

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[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's (CASAC) Peer Review of the Agency's 2nd Draft Ozone Staff Paper, EPA-CASAC-07-001, October 24, 2006.

[6] 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.

"Additionally, we note that the understanding of the associated science has progressed to the point that *there is no longer significant scientific uncertainty regarding the CASAC's conclusion that the current 8-hr primary NAAQS must be lowered.* A large body of data clearly demonstrates adverse human health effects at the current level of the 8-hr primary ozone standard. Retaining this standard would continue to put large numbers of individuals at risk for respiratory effects and/or significant impact on quality of life including asthma exacerbations, emergency room visits, hospital admissions and mortality."

"*...on the basis of the large amount of recent data evaluating adverse health effects at levels at an below the current NAAQS for ozone, it is the unanimous opinion of the CASAC that the current primary ozone NAAQS is not adequate to protect human health.*"

The point about uncertainty is particularly cogent, because many parties have raised the uncertainty issue in arguing against tighter standards. This unanimous statement from CASAC is unequivocal.

CASAC: The rounding loophole leaves millions of Americans unprotected and must be eliminated.

Under the current standard of 0.08 ppm, violations are not recorded until concentrations reach 0.085 ppm. This rounding convention allows unhealthful concentrations of ozone to continue unabated. Importantly, the CASAC specifically recommends 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."

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

Finally, 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 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."

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 if 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.

Given the strength and unanimity of CASAC's recommendations in this case, EPA has a particularly heavy burden to justify departing therefrom, a burden not met by the current proposal.

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 EPA Administrator Stephen L. Johnson about the EPA 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."

[7] http://yosemite.epa.gov/ochp/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.

CHPAC: We support the form of the new standard to be specified to the thousandths of ppm.

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 zone 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."

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. In late 2006, EPA announced final changes to the NAAQS review process that eliminate the Staff Paper. This current review of the ozone standard was subject to the preexisting NAAQS review process -- thus a final (and perhaps, last ever) Staff Paper was prepared that included staff conclusions and recommendations.

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 O_3 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 O_3-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 O_3 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 O_3 concentrations below the level of the current standard, or below even much lower O_3 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 O_3 standard that would provide increased health protection for sensitive 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.

[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.

Staff has indicated that standard levels of 0.074, 0.070, and 0.064 ppm are representative of levels within the upper, middle, and lower parts of this range, respectively.[11] In other words, a standard of 0.079 ppm, as OMB directed to include in the analysis of options in the Regulatory Impact Analysis,[12] is a mere 1 ppb below the current standard and could not be possibly considered as "somewhat below" 0.080, or within the staff recommended range. The air quality, exposure, and risk analyses specifically defined 0.074 ppm as within the upper end of the recommended range.

The Staff Paper further recommended that:

> "consideration be given to specifying the level of the primary standard to the nearest thousandth ppm, reflecting the degree of precision with which ambient O_3 concentrations can be measured and design values can be calculated."

Finally, the Staff Paper made these additional recommendations regarding the form of the standard:

> "We conclude that it is appropriate to consider a form in the range of the annual third- to fifth-highest daily maximum 8-hr average concentration, which includes the current form of the annual fourth-highest daily maximum 8-hr average concentration, averaged over three years. It is appropriate to consider a form within this range in conjunction with a standard level within the recommended range, so as to provide an appropriate degree of increased public health protection."

We highlight these specific conclusions here, but note that they are 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 Epidemiology[13] and the American Thoracic Society have gone on record in support of more stringent ozone standards.

[11] Wegman, Lydia, Director Health and Environmental Impacts Division, Office of Air Quality Planning & Standards, U.S. EPA. Briefing to HEI Annual Conference, Chicago, IL, "Current Thinking about Ozone Health Effects and Standard Setting: Update on EPA's Review of O_3 NAAQS," April 17, 2007.
[12] OMB List of Items for RIA and EPA Responses, EPA-HQ-OAR-2007-0225-0002.
[13] Letter from Daniel Wartenberg, PhD to Administrator Johnson, RE: Docket ID No. EPA-HQ-2005-0172, October 5, 2007.

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 show 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 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.[14]

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.[15]
>
> 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.[16] Hospitalizations and premature mortality have also been linked to increases in ozone.[17]

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[14] American Academy of Pediatrics Committee on Environmental Health. Ambient Air Pollution: Health Hazards to Children. *Pediatrics* 2004; 114: 1699 -1707.
[15] Ibid.
[16] Ibid.
[17] Bates DV. Ambient ozone and mortality. *Epidemiology* 2005; 16: 427-429.

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.[18] Another study found that chronic, long-term exposure to ambient ozone was associated with decreased levels of small airways function in college students.[19]

...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.[20]

"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[21]). Second, children spend more time outdoors than adults do, increasing exposure to ambient air pollutants (2)[22]. 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)[23]. 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

[18] 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.
[19] 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.
[20] 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
[21] Kim JJ. Ambient air pollution: health hazards to children. *Pediatrics* 2004;114:1699–1707.
[22] Spier CE, Little DE, Trim SC, Johnson TR, Linn WS, Hackney JD. Activity patterns in elementary and high school students exposed to oxidant pollution. *J Expo Anal Environ Epidemiol* 1992;2:277–293.
[23] Burri PH. Postnatal development and growth. In: Crystal RG, Editor. The lung: scientific foundations. Philadelphia: Lippencott-Raven; 1997. pp. 1013–1026.

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)[24]. 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)[25]. Current ozone levels in Canada's largest cities are associated with increased hospitalization for respiratory problems in neonates under 1 month of age (10)[26]. 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)[27], and with increased use of rescue medication in children with asthma under 12 years of age using maintenance medication (12)[28]. 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)[29]. 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,[30] 15[31]).

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[24] Avol EL, Gauderman WJ, Tan SM, London SJ, Peters JM. Respiratory effects of relocating to areas of differing air pollution levels. *Am J Respir Crit Care Med* 2001;164:2067–2072. Gauderman WJ, McConnell R,. Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, *et al.* Association between air pollution and lung function growth in Southern California children. *Am J Respir Crit Care Med* 2000;162:1383–1390. Schwartz JD,Katz SA, Fegley RW, Tockman MS.Analysis of spirometric data from a national sample of healthy 6- to 24-year-olds (NHANES II). *Am Rev Respir Dis* 1988;138:1405–1414.

[25] Burnett RT, Smith-Doiron M, Stieb D, Raizenne ME, Brook JR, Dales RE, Leech JA, Cakmak S, Krewski D. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. *Am J Epidemiol* 2001;153: 444–452. Lewis TC, Robins TG, Dvonch JT, Keeler GJ, Yip FY, Mentz GB, Lin X, Parker EA, Israel BA, Gonzalez L, *et al.* Air-pollution associated changes in lung function among asthmatic children in Detroit. *Environ Health Perspect* 2005; 113:1068–1075. Peel JL, Tolbert PE, Klein M, Metzger KB, Flanders WD, Knox T, Mulholland JA, Ryan PB, Frumkin H. Ambient air pollution and respiratory emergency department visits. *Epidemiology* 2005;16: 164– 174.

[26] 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.

[27] 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.

[28] 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.

[29] 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.

[30] Kunzli N, Lurmann F, Segal M, Ngo L, Balmes J, 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

[31] 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.

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)[32]. 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,[33] 18[34]). Recent studies demonstrate that some of the individuals tested experience these adverse effects at concentrations of 0.06 ppm and below (19[35])."

American Thoracic Society, American Medical Association, American College of Chest Physicians, American College of Preventive Medicine. American College of Occupational and Environmental Medicine, American Association of Cardiovascular and Pulmonary Rehabilitation and National Association for the Medical Direction of Respiratory Care

In addition to the **American Thoracic Society,** which is described above, six other national medical societies have expressed strong support for much more protective ozone standards than those proposed by EPA. They are:

- The **American Medical Association** is nation's largest professional medical society.
- The **American College of Chest Physicians** is a not-for-profit medical society representing 16,500 members in over 100 countries. Members include specialist physicians, allied health professionals, and PhDs focusing on diseases of the chest.
- The **American College of Preventive Medicine** (ACPM) is the national professional society for physicians committed to disease prevention and health promotion. ACPM has 2,000 members engaged in preventive medicine practice, teaching and research.
- The **American College of Occupational and Environmental Medicine** (ACOEM) represents more than 5,000 physicians and other health care professionals specializing in the field of occupational and environmental medicine. ACOEM is the nation's

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[32] Bell ML, Dominici F, Samet JM. A meta-analysis of time-series of ozone and mortality with comparison to the national morbidity, mortality, and air pollution study. *Epidemiology* 2005;16:436–445.

[33] 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.

[34] Horstman DH, Folinsbee 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, and0.12 ppm. *Am Rev Respir Dis* 1990;142:1158–1163

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

largest medical society dedicated to promoting the health of workers through preventive medicine, clinical care, research, and education.

- The **National Association for Medical Direction of Respiratory Care** is a national organization of pulmonologists and other physicians who provide clinical and management leadership in respiratory and critical care in nearly 2,000 hospitals nationwide.

- The **American Association of Cardiovascular and Pulmonary Rehabilitation** is the premier professional organization dedicated to the development of its members who are involved in the profession of cardiovascular and pulmonary rehabilitation.

In a letter to Administrator Johnson, dated October 9, 2007, these medical societies recommended EPA adopt a much stronger NAAQS for ozone, as noted below:

> "The undersigned medical professional societies recommend the EPA adopt the following NAAQS for ozone:
>
> - The level of the primary standard should be no higher than 0.060 ppm;
> - The degree of precision for the standard should be expressed at the thousandth ppm;
> - The form of the standard should be constructed as a three-year average of the annual third highest daily maximum 8-hour average ozone concentration.
>
> "The undersigned organizations strongly agree with the Administrator's findings that the current NAAQS for ozone is not protecting public health. We believe that the Administrator has correctly stated that, beyond any degree of scientific uncertainty, convincing and compelling evidence has demonstrated that exposure to ozone at levels below the current standard is responsible for measurable and significant adverse health effects, both in terms of morbidity and mortality. We strongly support the Administrator in his efforts to issue a more stringent standard and absolutely reject any efforts to maintain the current standard.
>
> "While we support this effort to lower the current standard, we are disappointed that the proposed rule does not go far enough to protect the public's health from the known adverse health effects of ozone pollution. The range proposed by the Administrator will not sufficiently protect the American public and will continue to expose vulnerable populations–including children, patients with respiratory diseases and the elderly–to detrimental levels of ozone.
>
> "We are especially concerned that the Administrator fails to appreciate the number and strength of studies that demonstrate adverse health effects at levels below the range being considered in the EPA's proposal."

They provided extensive evidence from these studies in their letter and argued pointedly against EPA's conclusions on the issue of scientific uncertainty, calling the evidence "robust and compelling" as well as "unassailable":

> "We note with concern that throughout the standard-setting process, senior EPA officials have taken a very conservative approach to reviewing the scientific literature on the health effects of ozone. We find the science on the health effects of ozone to be robust and compelling. There are literally hundreds of articles that provide data supporting what we have put forth in these comments.
>
> "The range of effects described is broad, including respiratory disease, cardiac disease, low-birth weights and birth defects. While continued research is needed to understand the precise pathway of disease mechanisms for all these known health effects, the reality of these health effects in unassailable. The known respiratory, cardiac and perinatal effects of ozone pollution are each in their own right major public health issues. In combination, they provide immediate actionable information and require a meaningful policy response from the EPA." [36]

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.

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[36] Letter from the American Thoracic Society, American Medical Association, American College of Chest Physicians, American College of Preventive Medicine. American College of Occupational and Environmental Medicine, American Association of Cardiovascular and Pulmonary Rehabilitation and National Association for the Medical Direction of Respiratory Care to Stephen L. Johnson, Administrator, Environmental Protectin Agency. October 9, 2007. Submitted as comments to EPA-HQ-OAR-2005-0172.

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.[37]

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."[38]

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.[39] 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.

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[37] 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

[38] 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#

[39] 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 American, 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-environ-groups-on-ozone-naaqs-04-16-07.pdf

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.[40]

In addition, dozens of additional local, state, and national organizations presented testimony in support of tighter standards at EPA public hearings held in Philadelphia, Los Angeles, Chicago, Houston, and Atlanta, on August 30 and September 5, 2007.

International Reviews

World Health Organization

In October 2006, the World Health Organization (WHO) revised their international air quality guidelines for ozone.[41] The prior guideline for 8-hour average ozone concentrations of 120 $\mu g/m^3$ (0.061 ppm) was reduced to 100 $\mu g/m^3$ (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 report specifically indicates that an 8-hour average concentration of 82 ppb, does not provide adequate protection of public health. The report notes that 1) this is the lower level of 6.6-hour chamber exposures of healthy exercising young adults where physiological and inflammatory lung effects have been observed; 2) this is the ambient level at various summer camp studies showing effects on health of children; and 3) this level is associated with an estimated 3-5% increase in daily mortality, based on the findings of daily time-series studies.[42]

The WHO recommendations were developed by a work group of dozens of leading international air quality and health scientists. According to WHO, the previously

[40] 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.

[41] 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

[42] World Health Organization. WHO air quality guidelines global update 2005. Report of a Working Group meeting, Bonn, Germany, 18-20 October 2006.

recommended guideline value, "which was fixed at 120 $\mu g/m^3$ 8-hour mean [61 ppm], has been reduced to 100 $\mu g/m^3$ [51 ppm] based on recent conclusive associations between daily mortality and ozone levels occurring at ozone concentrations below 120 $\mu g/m^3$." [43]

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 countries have promulgated 8-hour average standards that are more stringent than the current or even the proposed ozone standard.

Country	1 hour	8 hour	Exceedances Allowed per year
WHO		51	
European Union -2010		61	25
Australia	100	80	1
Bangladesh	120	80	
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	
Singapore	120	80	
Sri Lanka	102		
Switzerland		61	1
Thailand	102	71	
Viet Nam	92	61	
United Kingdom		51	10
United States		84	3

Table 1: Comparison of Ozone Standards Worldwide (ppb)[44]

[43] http://www.who.int/mediacentre/factsheets/fs313/en/index.html

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 at a level that reduces the health burden experienced by the nation's population as the result of exposure to ozone air pollution.

> "We note that the EPA's panel of expert science advisors, the Clean Air Scientific Advisory Committee (CASAC), has reviewed the scientific evidence in the EPA Criteria Document and Staff Paper and has unanimously recommended that "the primary 8-hr NAAQS needs to be substantially reduced to protect human health, particularly in sensitive subpopulations" (CASAC letter to Administrator Johnson, dated October 24, 2006). We also agree with their unanimous conclusion that "there is no scientific justification for retaining the current primary 8-hour NAAQS." Expert opinion, including recommendations by EPA staff scientists in the final Staff Paper, holds that retaining the current standard would put large numbers of people at risk for respiratory effects, asthma exacerbations, emergency room visits, hospital admissions, and mortality.
>
> The Clean Air Scientific Advisory Committee has further recommended that EPA close the "rounding loophole" which allows areas with concentrations up to 0.085 ppm to escape regulation under the current standard of 0.08 ppm, a position that we fully endorse.
>
> The Clean Air Scientific Advisory Committee has further unanimously recommended an eight-hour primary ozone standard in the range of 0.060 ppm to 0.070 ppm. The Committee specifically expressed its recommendation to the third decimal place to avoid the rounding loophole. This recommendation was unanimously reconfirmed in a March 5, 2007 meeting of the Committee.

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[44] 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/ttncatac1/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

....we strongly and solemnly request that you follow the recommendations of the Clean Air Scientific Advisory Committee and reduce the eight-hour primary ozone standard to a range between 0.060 and 0.070 ppm."[45]

The conclusions of these many scientific reviewers, governments, organizations and individuals converge on the need to significantly strengthen the existing ozone NAAQS in keeping with the recommendations of CASAC. In the next sections, we will review the basis for these conclusions and provide evidence for our recommendation for a 0.060 ppm 8-hour ozone primary standard.

No Scientific Basis Exists for Retaining the Current Standards

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.

In the previous section we provided ample evidence of scientific consensus that the primary NAAQS for ozone must be set at 0.060 ppm 8-hour average, or tighter. Despite all the evidence, the EPA has opened the door to retaining the existing standard. EPA has bent over backwards to accept comment on a standard that has repeatedly been found inadequate—even by the Administrator's own assessment. CASAC's repeated statement that there is "no justification" for the existing standard should have been enough to eliminate it from the proposal. But there it remains.

We will refrain from repeating all of the preceding scientific opinion in this discussion, which argued powerfully against keeping the existing standard. Instead, we will address arguments for keeping the current standard made by others. But we will begin with the conclusions of the Administrator himself, who agrees that the existing standard has to go.

[45] Letter to U.S. EPA Administrator Stephen Johnson re Broad Scientific Consensus to Lower the Ozone Air Quality Standard and Close the Rounding Loophole, from Jonathan I. Levy, Sc.D., Associate Professor of Environmental Health and Risk Assessment, Harvard School of Public Health; Kent Pinkerton, Ph.D., Director of the Center for Health and the Environment, University of California at Davis; and William Rom, M.D., M.P.H., Sol and Judith Bergstein Professor of Medicine and Environmental Medicine and Director of the Division of Pulmonary and Critical Care Medicine, New York University School of Medicine, and over 100 other air quality scientists and physicians, April 4, 2007. Available at: http://www.cleanairstandards.org/wp-content/uploads/2007/04/final-ozone-scientists-sign-on-letter-4-5-07.doc

EPA Administrator Stephen Johnson: "The Current Standard Does Not Protect"

EPA Administrator Stephen L. Johnson explained his decision on the proposed revisions to the ozone standard to the Senate Subcommittee on Clean Air and Nuclear Safety:

> "Based on the large body of evidence concerning the public health impacts of ozone pollution, including new evidence concerning effects at ozone concentrations below the level of the current standard, I proposed that the current standard does not protect public health with an adequate margin of safety and should be revised to provide additional public health protection, particularly for those with asthma or other lung diseases, adults who are active outdoors, and the youngest and oldest members of our population."[46]

This strong statement leaves little room for backtracking or promulgating a weaker standard than proposed, despite political pressure to do so.

Other arguments for retaining the existing standard are flawed

We have heard many arguments from opponents for not revising the standard. Here are some of the most common, most of which have been recycled from prior NAAQS reviews. Following each is a brief rebuttal. However, we repeat again, 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"

This argument alleges that since the State Implementation Plans for the 1997 ozone standard are just now complete, EPA should wait until the measures are implemented before changing the standard. The argument claims that EPA is "moving the goal post" before the work on the 1997 NAAQS is underway.

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. Even with this review, EPA is five years behind the schedule directed by the Act. 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.

In any event, reductions in pollution to meet the 1997 standards will help meet the new standard. Under the Clean Air Act, communities will have plenty of time to plan, adopt and put in place measures to meet these new standards. In fact, states that are planning now can

[46] Testimony of Stephen L. Johnson, Administrator, U.S. Environmental Protection Agency, Before the U.S. Senate Environment And Public Works Committee, Subcommittee On Clean Air And Nuclear Safety, July 11, 2007.

use this valuable information about the goal that they really will need to meet so they can begin to take steps now.

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 would mean revisions to the standard.

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 35 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 35 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 chart, Figure A below, tracking the growth of the population and the gross domestic product since the passage of the Clean Air Act Amendments of 1970 shows that stronger standards do not harm the economy:

> "The graph below shows that between 1970 and 2006, gross domestic product increased 203 percent, vehicle miles traveled increased 177 percent, energy consumption increased 49 percent, and U.S. population grew by 46 percent. During the same time period, total emissions of the six principal air pollutants dropped by 54 percent."[47]

[47] U.S. EPA. Air Quality and Emissions: Progress Continues in 2006. http://www.epa.gov/air/airtrends/econ-emissions.html.

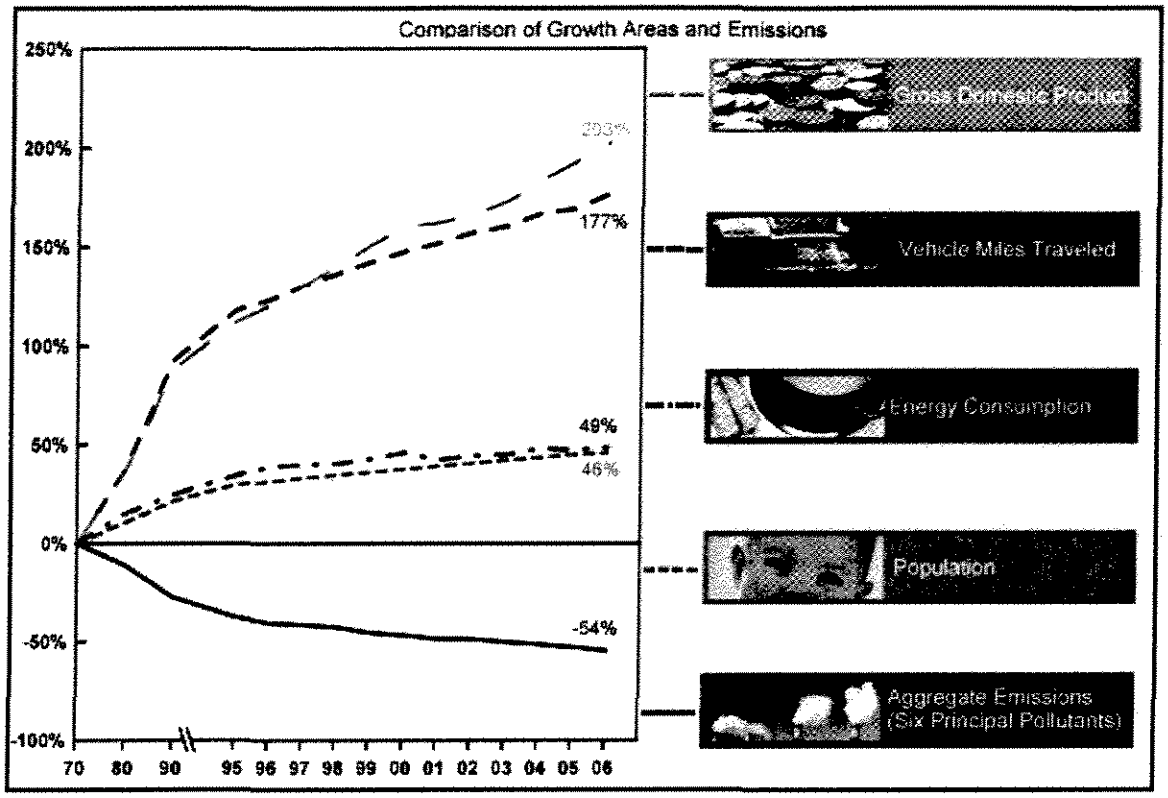

Figure A: EPA Chart showing the relative change in four measures of population and economic growth and in emission reductions from 1970 to 2006. Source: http://www.epa.gov/airtrends/sixpoll.html

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. In fact, each year the White House analyzes the costs and benefits of such regulatory requirements. Each year, EPA's air pollution regulations total benefits that outweigh the costs by as much as 40 to 1.[48] What isn't usually seen 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 37 years of experience to show that cleaning up air pollution doesn't hurt economic growth.

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:

[48] White House Office of Management and Budget, Office of Information and Regulatory Affairs. Draft 2007 Report on the Costs and Benefits of Federal Regulations. March 2007. Posted at http://www.whitehouse.gov/omb/inforeg/regpol-reports_congress.html.

> "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.

In any event, the notion that stronger ozone standards are not achievable is belied by the record. This isn't the first time we've had to stretch clean up air pollution—we've successfully done it before. 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. America leads the world in pollution control innovation.

In 2004, EPA identified some 126 communities across the nation with air pollution concentrations above the ozone health standard adopted in 1997. Today, based on preliminary air quality data, EPA estimates that all but 35 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 more than 20 percent.[49] See Figure B below. These pollution reductions have prevented hospital admissions and school absences for respiratory illnesses, and have saved lives.

[49] U.S. EPA, The Ozone Report, *Measuring Progress through 2003,* (Nov. 17, 2005).

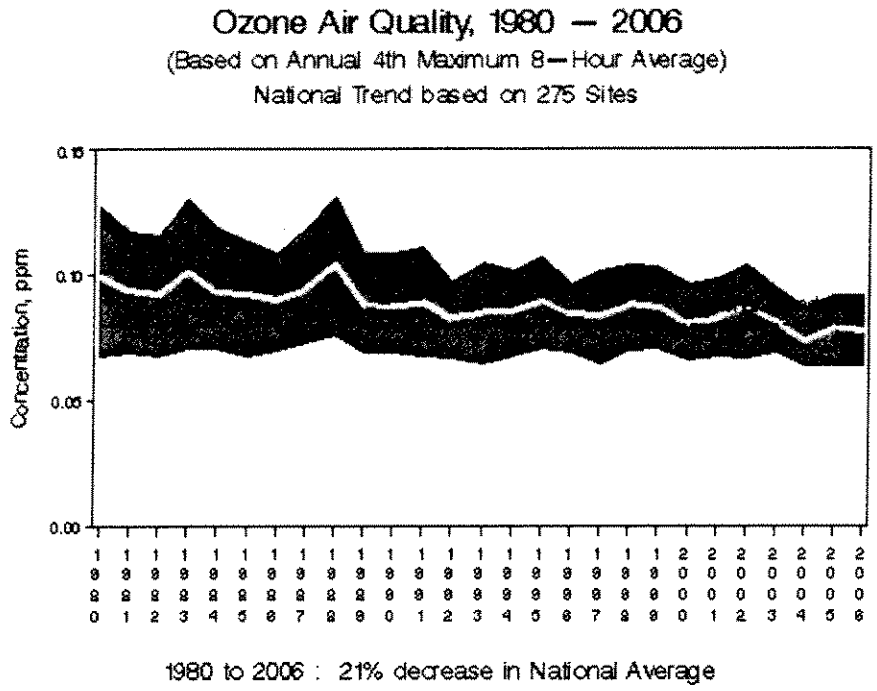

Figure B: Ozone Air Quality, 1980 - 2006. Source: http://www.epa.gov/airtrends/ozone.html

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 clean up diesel locomotives and marine engines. In addition, we will need to put tighter controls on coal-fired power plants and industrial boilers.

Scientific Evidence Shows Adverse Effects Below the Proposed Standards

EPA has proposed to set an 8-hour average primary ozone standard within the range of 0.075 ppm to 0.070 ppm. The upper end of this range clearly falls outside the range recommended by the independent CASAC which is charged with advising the EPA Administrator on these matters. Additionally, EPA has encouraged those that seek to retain the clearly inadequate current standard, by asking for public comment on this option, or a standard of 0.079 ppm. These options are clearly beyond the pale of scientific opinion. These comments will review the scientific evidence for setting a final air quality standard at the lower end of the range recommended by CASAC and by EPA staff scientists, that is, a 0.060 ppm 8-hour standard.

Before we turn to a detailed discussion of the current review, some historical perspective is in order.

The 1997 Revisions to the Ozone Standard

EPA last revised the ozone standard a decade ago in 1997, setting an 8-hour average standard of 0.08 ppm, which is still in effect today. However, this standard is effectively 0.085 ppm due to the rounding convention, which permits states to round monitored concentrations up to 0.084 ppm down to 0.08 ppm. In other words, exceedances of the standard are not measured until concentrations reach 0.085 ppm for more than several days each year in a three-year period.

In the mid-1990s, the American Lung Association argued that a standard of 0.08 ppm could not be considered protective of public health for several reasons, including:

- the standard was effectively 0.085 ppm, well above the level shown to cause adverse effects in clinical chamber studies;
- chamber studies with 0.08 ppm ozone had demonstrated adverse respiratory responses in healthy adults, thus did not allow for a margin of safety;
- sensitive populations such as infants, children, and those with severe respiratory disease were likely to experience effects at lower concentrations;
- epidemiological studies had correlated hospital admissions and emergency department visits for asthma with ozone concentrations below 0.08 ppm.

In comments filed with EPA, the American Lung Association stated[50]:

> "A six to eight-hour average standard is necessary to address the substantial body of clinical study evidence indicating that multi-hour ozone exposures produce clinically significant decreases in lung function, respiratory symptoms and biochemical evidence of inflammatory damage at ozone levels as low as 0.08 ppm. These responses occur in a significant percentage of the population. A recent analysis by McDonnell et al.[51] of three earlier EPA clinical studies indicates that a lung function drop of at least 10 percent occurred in from 7 to 25% of the 59 moderately exercising subjects exposed to 0.08 ppm ozone as the exposure duration increased from 4.6 to 6.6 hours."

[50] American Lung Association, Comments of the American Lung Association to the U.S. Environmental Protection Agency Regarding "Review of National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information -- OAQPS Staff Paper External Review Draft (August 1995). October 16, 1995.

[51] McDonnell WF, Stewart PW, Andreoni S, Smith MV. Proportion of Moderately Exercising Individuals Responding to Low-Level, Multi-hour Ozone Exposure. *Am J Respir Crit Care Med* 1995; 152: 589-586.

> "Complementing this evidence are the results of an increasing number of well designed epidemiological studies that have correlated hospital admissions and emergency department visits for asthma and other respiratory problems with ozone levels well below the current primary ozone standard. As Thurston[52] concluded in a recent paper reviewing these studies, "...the aggregate population time series studies provide strong evidence that ambient exposures to ozone are associated with significant increases in the number of exacerbations of preexisting respiratory disease in the general public, even at levels below the current U.S. standard" (emphasis added). The Staff Paper acknowledges that these studies suggest the existence of a linear, nonthreshold relationship between ozone exposure and these unequivocally adverse health endpoints. CASAC confirmed this perspective at its September 19, 1995 meeting."
>
> "There is now a substantial and coherent body of scientific evidence from clinical and epidemiological studies regarding the occurrence of adverse acute health effects of ozone levels at or below 80 parts per billion (ppb), supported by evidence from toxicological studies of chronic ozone exposure to somewhat higher levels indicating lung tissue damage, reduced lung elasticity, and accelerated loss of lung function."

The comments go on to state:

> "In accordance with the public health protection imperative under Section 109 of the Clean Air Act for setting primary national air quality standards at a level that 'allowing [for] an adequate margin of safety [are] requisite to protect the public health,' ALA recommends that EPA adopt a revised primary national ozone air quality standard at 70 ppb, the lower end of the range proposed by EPA in the Staff Paper. ALA concurs with EPA's finding in the Staff Paper that a standard set at this level would better provide public health protection with an adequate margin of safety for acute adverse effects, and would provide increased protection from long-term exposures that may be associated with serious chronic effects..."

Thus, even in 1995, there was compelling evidence to support a standard at the 0.070 ppm level. These arguments remain valid today, but are strengthened by ample new evidence accumulated in the last decade, which points to the need for a standard of 0.060 ppm to provide a margin of safety, as required by the Clean Air Act.

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[52] Thurston GD. Associations of Acute Ambient Ozone Exposures with Hospital Admissions for Respiratory Causes. Presented at the 88th Annual Meeting of the Air and Waste Management Association; June 1995.

Chamber Studies Show Need for More Protective Standards

The 0.085 ppm concentration is actually above the level shown to cause adverse respiratory effects and symptoms in healthy adults in numerous controlled human exposure studies. When EPA established the 1997 standard, it was based on numerous clinical chamber studies demonstrating effects at concentrations of 0.08 ppm ozone, under 6.6 hour exposure regimes, as well as on numerous epidemiological and field studies of community exposures.

A number 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[53] -- were evident following 6.6- to 8-hour exposures to 0.08 ppm ozone. For example, studies conducted in the EPA laboratory in Chapel Hill, North Carolina reported statistically significant lung function decrements, that is, forced expiratory volume in one second (FEV_1), and respiratory symptom responses in young healthy adults exposed to 0.08 ppm ozone for 6.6 hours while exercising.[54,55]

Interpretation of Pre-1996 Studies

Field studies demonstrating adverse effects in association with daily exposures to ozone were also important to the setting of the 1997 standards, and provide evidence of effects at concentrations below the current standards. These studies demonstrate the inadequacy of the current standard, which is not enforced until concentrations reach 0.085 ppm. Furthermore, they demonstrate that retention of the current standard, or reducing it to 0.079 ppm, would fail to protect public health with a margin of safety.

Key health studies that were considered in the setting of the 1997 standard are summarized in the following Table 2, which is extracted from the 1996 Final Ozone Staff Paper (where it was labeled Table V-2).[56] The pre-1996 6.6-hour chamber studies indicated effects at exposures to 0.08 ppm under exercise conditions. For the most part, the experimental subjects in these studies were healthy young adults such as college students.

These chamber studies from the late-1980s to mid-1990s demonstrated a string of adverse health effects including:

- reduced lung function

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[53] 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.

[54] 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.

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

[56] U.S. EPA, Office of Air Quality Planning and Standards, Review of National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information. EPA-452/R-96=007, June 1996.

- respiratory symptoms
- airway responsiveness
- inflammation
- increased susceptibility to respiratory infection

These respiratory effects were all evident in healthy adults exposed to 6.6 hour exposures of ozone of 0.08 ppm and higher, while exercising. The fact that a variety of adverse effects were evident in this study population indicates that a standard set at or even just below the level tested 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 a margin of safety.

Table 2: Key Health Studies Considered in Setting the 1997 Standards, From the U.S. EPA Criteria Document for Ozone and Related Photochemical Oxidants, July 1996.

TABLE V-2. KEY HEALTH STUDIES PUBLISHED SINCE THE LAST REVIEW OF THE PRIMARY NATIONAL AMBIENT AIR QUALITY STANDARD FOR OZONE

O_3 Concentration, ppm	Health Effect	Reference
Ambient air containing 0.01-0.154 daily 1-hr max over days to weeks	Decrements in lung function (FEV_1) in children, adolescents, and adults exposed to O_3 outdoors	Avol et al. (1990, 1991) Braun-Fahrlander et al. (1994) Castillejos et al. (1992) Hoek et al. (1993a,b) Kilburn et al. (1992) Krzyzanowski et al. (1992) Lebowitz et al. (1991) Raizenne et al. (1987, 1989) Raizenne and Spengler (1989) Schmitzberger et al. (1993) Schwartz et al. (1994a,b,c) Spektor et al. (1991) Spektor and Lippmann (1991) Stern et al. (1989, 1994) Thurston et al. (1995)
	Exacerbation of respiratory symptoms (e.g., cough, chest pain) in individuals with preexisting disease (e.g., asthma) with low ambient exposure, decreased temperature, and other environmental factors resulting in increased summertime hospital admissions and emergency department visits for respiratory causes.	Burnett et al. (1994) Cody et al. (1992) Thurston et al. (1992, 1994, 1995) Weisel et al. (1995) White et al. (1994)
≥0.12 (1-3 hr) ≥0.08 (6.6 hr) (chamber exposures)	Decrements in lung function (reduced ability to take a deep breath), increased respiratory symptoms (cough, shortness of breath, pain upon deep inspiration), increased airway responsiveness and increased airway inflammation in exercising adults	Devlin et al. (1991, 1990) Folinsbee et al. (1991, 1994, 1995) Frampton et al. (1993) Gross et al. (1991) Hazucha et al. (1992, 1987) Horstman et al. (1990, 1995) Koren et al. (1991) McKittrick et al. (1995) McDonnell et al. (1991) Linn et al. (1994)
≥0.12 (1-3 hr) ≥0.08 (6.6 hr) (chamber exposures)	Effects are similar in individuals with preexisting disease except for a greater increase in airway responsiveness for asthmatic and allergic subjects	Koenig et al. (1988) Kreit et al. (1989)

O_3 Concentration, ppm	Health Effect	Reference
≥0.12 (1-3 hr) ≥0.08 (6.6 hr) (chamber exposures)	Older subjects (>50 yr old) have smaller and less reproducible changes in lung function.	Drechsler-Parks et al. (1990) Horvath et al. (1991) Seal et al. (1993, 1994)
	Attenuation of response with repeated exposure	Hackney et al. (1989) van Bree et al. (1994)
≥0.12 with prolonged, repeated exposure (chamber exposures)	Changes in lung structure, function, elasticity, and biochemistry in laboratory animals that are indicative of airway irritation and inflammation with possible development of chronic lung disease	Catalano et al. (1995a,b) Chang et al., (1991, 1992, 1995) Costa et al., (1995) Harkema et al. (1989, 1993, 1994) Harkema and Mauderly (1994) Hirashima et al. (1989) Hotchkiss et al. (1989a,b) Hyde et al. (1992) Last et al. (1983a,b, 1994) National Toxicology Program/Health Effects Institute (1995) Parks and Roby (1994) Pinkerton et al. (1992, 1993, 1995) Pino et al. (1992a,b,&c) Plopper et al. (1991, 1994a,b) Radhakrishnamurthy (1994) Schuliheis et al. (1991) Szarek (1994) Tan et al. (1992) Tepper et al. (1994, 1995) Tyler et al. (1991) Van Bree et al. (1992)
	Increased susceptibility to bacterial respiratory infections in laboratory animals	Gilmour et al. (1993a,b) Jakab and Bassett (1990) Jakab et al. (1995) Selgrade et al. (1990)

Table 2, continued: Key Health Studies Considered in Setting the 1997 Standards, From the U.S. EPA Criteria Document for Ozone and Related Photochemical Oxidants, July 1996.

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 current 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 known concentrations.

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.[57]

- 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.

The pre-1996 studies provide strong evidence of the adverse effects of ozone below the effective level of the current standard, 0.085 ppm. Clearly, the current standards fail to provide a margin of safety even considering only the pre-1996 chamber studies since adverse effects are unmistakably demonstrated below the effective level of the standards.

1996 to Present Chamber Studies

We disagree with EPA that revisions to the standards must be justified by new evidence. If the periodic review of the standards required by the Clean Air Act supports the conclusion that earlier studies requires a more stringent standard, or that the current standards are not protective of public health based on earlier studies alone, than EPA must revise the standards. We believe this is the case with respect to the pre-1996 chamber studies. However, as the

[57] http://www.arb.ca.gov/research/aaqs/ozone-rs/aqac/pres/aqac-o3.pdf

remainder of our comments will indicate, we also believe there is plentiful convincing new evidence from chamber studies, epidemiological studies, and toxicology studies that compels revision of the standards.

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 [58]discusses a 1996 study by McDonnell that provides additional evidence of differential responses to ozone. When combining data from a number of chamber studies of 6.6 hour exposures, the analysis shows that average FEV_1 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.[59] 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.

This principle is also relevant to the evaluation of more recent chambers studies of effects of 0.06 ppm ozone, and below.

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.[60]

Since 1996, two controlled human exposure studies have been conducted that evaluated the effect on lung function -- forced expiratory volume in one second (FEV_1) -- of various exposure regimes to concentrations of ozone of 0.08 ppm, 0.06 ppm and 0.04 ppm, for 6.6. hours.[61,62] These studies by Adams were funded by the American Petroleum Institute and

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[58] U.S. Environmental Protection Agency. Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information. OAQPS Staff Paper. July 2007. Page 3-6.
[59] McDonnell WF. Individual variability in human lung function responses to ozone exposure. *Environmental Toxicology and Pharmacology* 1996; 2: 171-175.
[60] 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.
[61] 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.

were intended to address the effect of various exposure regimes on lung function responses to ozone.

The Adams (2002) study reports that "some sensitive subjects experience notable effects at 0.06 ppm." According to the Staff Paper,[63] this is based on the observation that 20 percent of the subjects exposed to 0.06 ppm ozone had a greater than 10 percent decrement in FEV_1 even though the group mean response was not statistically different from the filtered air response. In a study with a small number of subjects—the response of individual subjects is more important than the group mean response. This is particularly true for ozone exposure, where research has long recognized the variability in individual responses.

The Criteria Document[64] indicates that in the Adams (2006) study, even group mean FEV_1 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 FEV_1 of 0.06 ppm ozone compared to filtered air.[65]This relationship is illustrated in Figure C below.

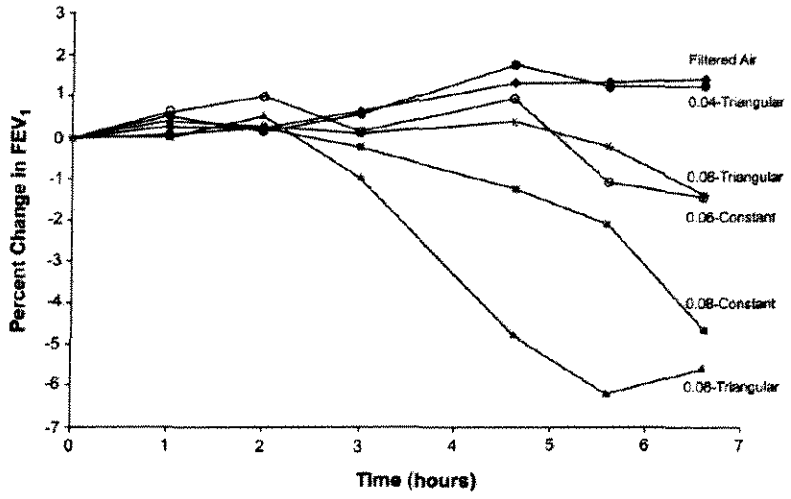

Figure 1. Hour by hour changes in FEV_1 (% change relative to preexposure) adapted from Adams (2006). Data are group mean (error bars were not provided in the published paper) responses of 30 healthy adults exposed to O_3 for 6.6 hours during quasi continuous exercise. The O_3 concentrations were either 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 C: 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.

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[62] 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.
[63] U.S. EPA Staff Paper. 2007 Page 3-9.
[64] U.S. Environmental Protection Agency. *Air Quality Criteria for Ozone and Other Photochemical Oxidants*. February 2006. Page 8-42.
[65] U.S. EPA Staff Paper. 2007. Page 3-8.

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. [66]

EPA has undertaken a careful reanalysis of the underlying data in the Adams (2002, 2006) studies to assess the change in FEV_1 following exposure to 0.06 ppm ozone and filtered air.[67] The purpose of the analysis was to note differences in statistical methods between studies, and to analyze FEV_1 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 FEV_1 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 FEV_1 responses compared to filtered air, as illustrated in following Figure D.

[66] U.S. EPA. Staff Paper, 2007. Page 3-9.

[67] 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.

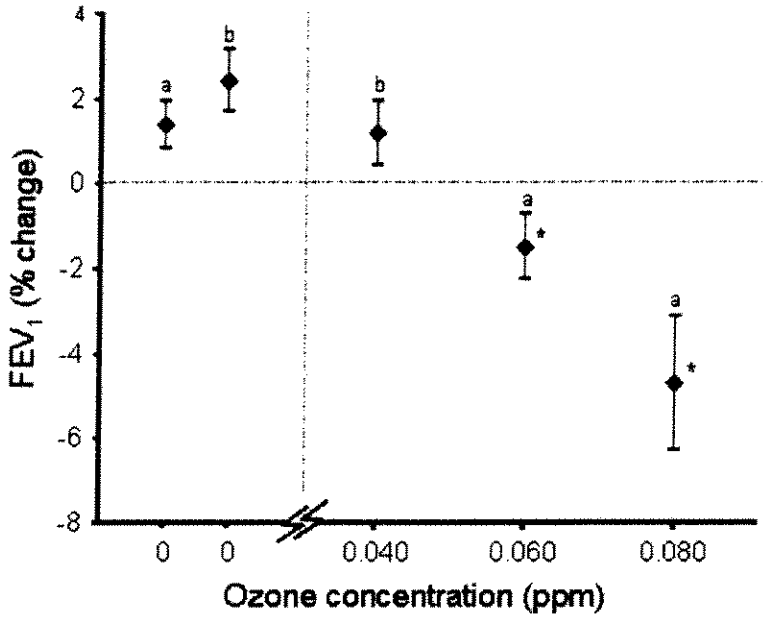

Figure 2. Effects of ozone on FEV_1 in healthy young adults exposed for 6.6 h during quasi continuous exercise to a constant (square-wave) O_3 concentration. Data are from a) Adams (2006) and b) Adams (2002). *Significantly different from responses to air exposure ($p \leq 0.001$, two-tail paired *t*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

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.

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 (less than 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 FEV_1 decrements greater than 10 percent at ozone exposures of 0.04 ppm. Seven percent experience such decrements at 0.06 ppm, and 23 percent at 0.08 ppm, as shown in Figure E taken from the EPA Staff Paper at 3-7.

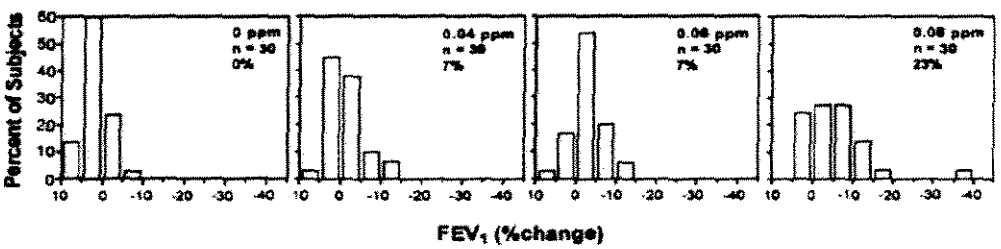

Figure 3-1C. Frequency distributions of FEV_1 changes following 6.6-h exposures to a constant concentration of O_3 or filtered air. The FEV_1 changes following O_3 exposures have been corrected for filtered air responses, i.e., they are O_3-induced FEV_1 changes. Note that the percentage in each panel indicates the distributions of % decrement.
Source: Adams (2002, 2006), pre- and post- FEV_1 data for each subject provided by author.

Figure E: 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. Currently, the U.S. population is over 303 million Americans.[68] Seven percent is 21.2 million people.

We concur with the conclusion of the EPA staff reanalysis that larger decrements in FEV_1 would be expected in more susceptible populations.

Clearly, EPA's proposed standard of 0.070 to 0.075 ppm cannot be considered protective of public health in light of experimental evidence demonstrating adverse respiratory effects in healthy individuals exposed to 0.060 ppm, and the legal requirement to protect sensitive populations with an adequate margin of safety.

Epidemiological Studies Document Effects at Low Concentrations

In the proposed rule, EPA solicits comment on the degree to which associations observed in epidemiological studies reflect causal relationships. (72 FR 37878). In the discussion below, we will discuss the evidence that provides a convincing case for causality. Further, we are not alone in our judgments.

First, we agree with the weight of evidence approach taken by the Criteria Document. 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

[68] U.S. Census Bureau. Data accessed October 9, 2007 from http://www.census.gov/.

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[69] 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 F 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.

[69] U.S. Environmental Protection Agency. *Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information OAQPS Staff Paper – Second Draft.* July 2006. Page 3-53.

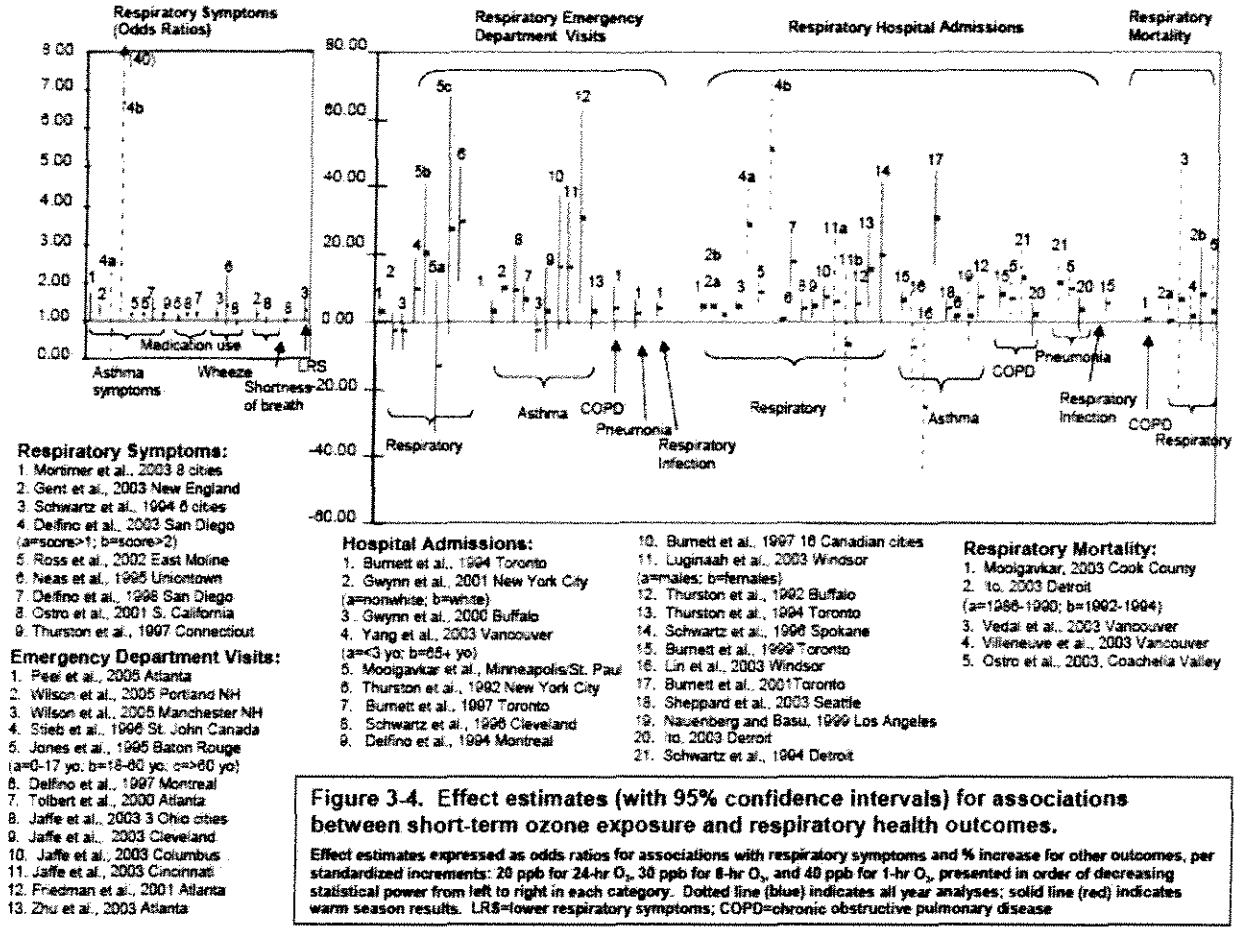

Figure 3-4. Effect estimates (with 95% confidence intervals) for associations between short-term ozone exposure and respiratory health outcomes.

Effect estimates expressed as odds ratios for associations with respiratory symptoms and % increase for other outcomes, per standardized increments: 20 ppb for 24-hr O_3, 30 ppb for 8-hr O_3, and 40 ppb for 1-hr O_3, presented in order of decreasing statistical power from left to right in each category. Dotted line (blue) indicates all year analyses; solid line (red) indicates warm season results. LRS=lower respiratory symptoms; COPD=chronic obstructive pulmonary disease

Figure F: 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 published since the last review 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.[70]

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 is can be very useful to inform the standard-setting process. Depending on the study design, a variety of statistics may be reported, for example 1-hour maximum, 8-hour average, 24-hour average, or various percentile concentrations. Investigators may make their own ozone measurements, or use publicly available databases of air quality measurements.

Accurately characterizing 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.[71] These alternate metrics attempted 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.[72]

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[73] and the 98th percentile values are not necessarily equivalent to nonattainment "design values."

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

[71] 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.

[72] 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.

[73] U.S. EPA. Staff Paper, 2007. Page 6-9.

Despite these concerns, useful information can still be gleaned from EPA's analysis. Table 3 below, drawn from Appendix 3B of the Staff Paper, arrays twenty North American studies which reported positive, statistically significant results for various health endpoints, for which EPA derived 98th percentile 8-hour daily maximum concentrations of about 85 ppb or lower.[74] 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, small confidence intervals indicate 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 demonstrates 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.

[74] Results may not be statistically significant for all endpoints examined.

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

Study Endpoints	98th percentile 8-hr daily max (ppb)
Mortality:	
Ito et al.,1996	76
Vedal et al., 2003	53.3

Table 3: Ozone Epidemiological Studies Showing Effects at Low Concentrations: EPA Derived 98th Percentile Statistics Near or Below the Current Standard[75]
American Lung Association, 2007, Derived from Staff Paper Appendix 3B. Ozone Epidemiological Study Results: Summary of effect estimates and air quality data reported in studies, distribution statistics for 8-hr daily maximum ozone concentrations for the study period and location, and information about monitoring data used in the study.

With respect to ozone and short-term mortality, which we discuss in a separate section, the conclusion in the Criteria Document 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 anyone with heart failure and pulmonary congestion, and would worsen the function of anyone with advanced lung disease."[76]

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.

[75] 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.
[76] Bates DV. Ambient Ozone and Mortality. *Epidemiology* 2005; 16: 427-429.

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.[77] The 8-hour average concentration of ozone in this study was 36 ± 12 ppb (mean ± 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 percent for a 0-day lag, 0.69 percent for a 1-day lag, and 0.52 percent 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 O_3 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 O_3 exposure at relatively low daytime ambient concentrations for healthy adults. Exercising healthy adults in New York City (USA) who were exposed to < 80 ppb O_3 were reported to have a 0.55-L/min decrease in their PEFR per 1 ppb O_3 (Spektor et al. 1988); healthy women exposed to 8-hr O_3 at 54 ppb in Connecticut and Virginia (USA) were reported to have a 0.083-L/min/ppb decrease in their PEFR per 1 ppb O_3 (Naeher et al. 1999); farm workers in Fraser Valley (Canada) who were exposed to a 1-hr daily maximum O_3 of 40 ppb were reported to have 3.3-mL and 4.7-mL decreases in their $FEV_{1.0}$ and FVC, respectively, per 1 ppb O_3 (Brauer et al. 1996). A similar dose-response relationship between O_3 and PEFR reduction was also reported in some European studies. Male cyclists in the Netherlands who were exposed to < 60 ppb O_3 were reported to have 0.57-L/min decreases in PEFR per 1 ppb O_3 (Brunekreef et al. 1994); healthy workers and athletes in Germany who were exposed to < 80 ppb O_3 were also reported to have decrements in their FEV_1 (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.[78] The Staff Paper[79] appears to dismiss the significance of this study by claiming that the exposure

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[77] 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.

[78] 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.

[79] U.S. EPA. Staff Paper, 2007. Page 6-12.

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. Eight-hour ozone concentrations exceeded 50 ppb only once during this study period, and concentrations of other pollutants were low. These low ozone concentrations were significantly associated with a decline in lung function over a race or training period. There was also an increase in respiratory symptoms, especially shortness of breath, 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.[80]

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.[81] A standard of 0.060 ppm is needed to protect hikers and others who exercise outdoors.

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 O_3 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 O_3 concentration in the study by Kinney and colleagues was 78 ppb, the threshold for responses to O_3 in ambient air for

[80] 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.

[81] 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 Perspec* 1998; 106: 93-99.

adults and children engaged in normal activities appear to be well below 80 ppb."[82]

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.[83]

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 4 and Figure G below, the mean 8-hour maximum ozone concentration was 54.5 ppb, with a standard deviation $\pm$ 13.0.[84]

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

Pollutant	Mean ± SD	Median	Range	25th–75th percentile	IQR
24-hr average O_3 (ppb)	35.2 ± 8.4	35.7	13.5–56.6	28.8–40.6	11.8
8-hr maximum O_3 (ppb)	54.5 ± 13.0	55.3	23.5–87.6	45.1–64.1	19.0
1-hr peak O_3 (ppb)	60.8 ± 13.4	60.5	26.0–95.0	52.0–70.0	18.0
$PM_{2.5}$ ($\mu g/m^3$)	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 4: from Triche et al. 2006.

[82] Spektor DM, Lippmann M, Thurston GD, Lioy 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.

[83] 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.

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

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:

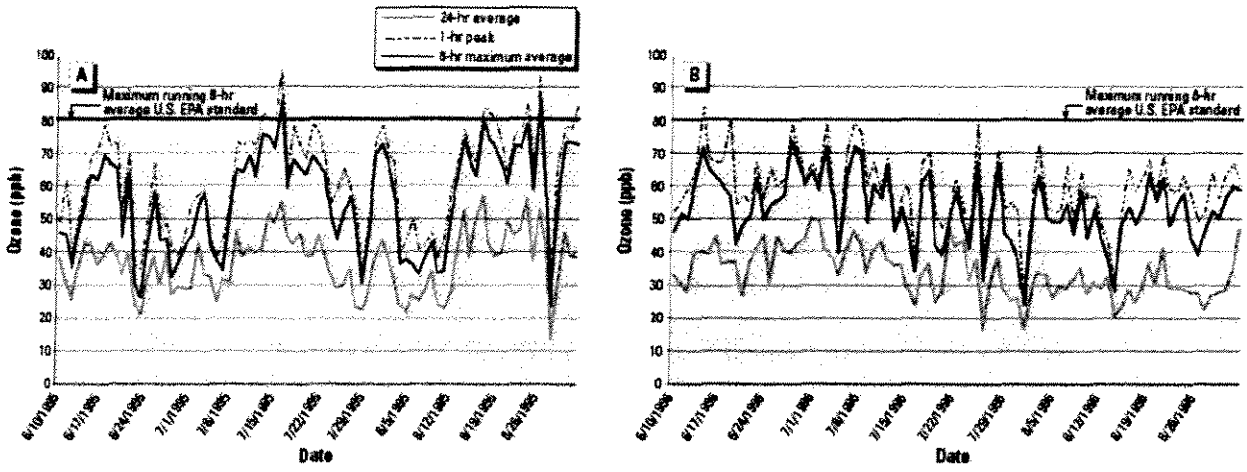

Figure G: from Triche et al. 2006.

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.[85] 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 5, 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.

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 (8, 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	46.1	72.3

Table 5: from Dales et al., 2006.

[85] 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.

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 NO_2 and O_3.

A study of the impact of ozone on daily respiratory admissions on children less than three years old and another sensitive population, the elderly, in Vancouver, British Columbia revealed associations between ozone and respiratory hospital admissions, which persisted after adjustment for copollutants and socioeconomic status. The 24-hour average ozone concentrations in this study were very low, at 13.41 ppb.[86]

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.

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

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 (21percent) and children (18 percent), as compared to the elderly and athletes (both 5 percent). This means that children and asthmatics have a higher risk of being ozone sensitive and experiencing more acute lung function decrements than these other population groups.[87] 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.[88] 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

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[86] 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.
[87] 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.
[88] 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.

cities studied were Baltimore, Chicago, Cleveland, Detroit, Bronx/East Harlem, St. Louis, and Washington DC. Eight-hour average ozone concentrations from 10 a.m. to 6 p.m. were 48 ppb. Median concentrations across cities ranged from 34 to 58 ppb (see Figure G below).[89] 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. The mean 8-hour ozone concentrations from 10 a.m. to 6 p.m. across these cities were 48 ppb, as shown in Figure H. 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."[90]

[89] 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.
[90] 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.

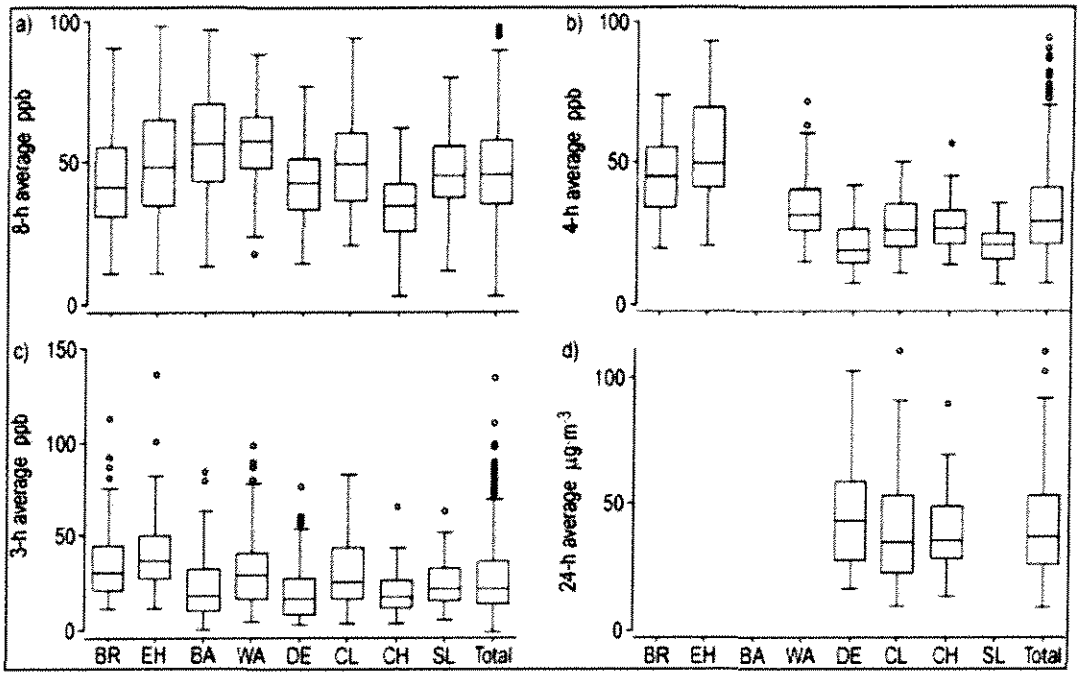

Figure H: from Mortimer et al., 2000.

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 6, mean 8-hour ozone concentrations in this study were 51.3 ppb, with a standard deviation of 15.5.[91]

[91] 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 ($PM_{2.5}$), and Temperature in Southern New England, April 1 to September 30, 2001

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

Table 6: 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 nonasthmatic 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.[92]

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.[93]

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.[94]

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[92] 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.

[93] 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.

[94] 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.[95]

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 7, 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 PM_{10} are independently associated with asthma exacerbation, and that the data "suggest continuing health risks at pollution levels that commonly occur in many US cities." This study provides strong evidence of the need to set the 8-hour average standard at 0.060 ppm.[96]

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

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

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

Table 7: from Tolbert et al., 2000.

[95] 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.

[96] Tolbert PE, Mulholland JA, MacIntosh DL, Xu F, Daniels D, Devine OJ, Carlin BP, Klein M, Dorley J, Butler AJ, Nordemberg 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 8, the mean 8-hour ozone concentrations in this study were 55.6 ppb, and the 90th percentile concentration was 87.6 ppb.[97]

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} ($\mu g/m^3$)*‡	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 $PM_{2.5}$ ($\mu g/m^3$)†	2	19.2 ± 8.9	8.9	32.3
24-h coarse PM ($\mu g/m^3$)†	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 ($\mu g/m^3$)†	9	0.028 ± 0.025	0.006	0.061
24-h $PM_{2.5}$ sulfate ($\mu g/m^3$)†	10	5.5 ± 3.7	1.9	10.7
24-h $PM_{2.5}$ acidity ($\mu \cdot equ/m^3$)†‖	15	0.018 ± 0.023	−0.001	0.045
24-h $PM_{2.5}$ organic carbon ($\mu g/m^3$)†	6	4.5 ± 2.2	2.2	7.1
24-h $PM_{2.5}$ elemental carbon ($\mu g/m^3$)†	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 PM_{10} values, 2% (46 of 1892) of ozone values, 14% (398 of 2775) of NO_2 values, 6% (161 of 2758) of CO values, and 9% (237 of 2775) of SO_2 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 $\mu \cdot equ/m^3$, a measure of pH level, accounting for the negative values. If converted into units of $nmol/m^3$, the mean is 18 and standard deviation is 23.
PPB, parts per billion; PPM, parts per million

Table 8: from Peel et al., 2005.

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).[98]

[97] 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.
[98] 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.

An important 2007 study *in press* 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.[99]

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.[100] This is a substantial segment of the overall population that is not adequately protected by the current air quality standards.

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 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 9 below, concentrations in most cities in the 40-55 ppb range.[101] This study provides powerful evidence for a standard of 0.060 ppm or below.

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[99] Babin SM, Burkom HS, Holtry RS, Tabernero 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. *Environmental Health* 2007; 6: 9 doi:10.1186/1476-069X-6-9.

[100] American Lung Association. *Trends in Asthma a Morbidity and Mortality.* August 2007.

[101] Medina-Ramón M, Zanobetti A, Schwartz J. The Effect of Ozone and PM_{10} on Hospital Admissions for Pneumonia and Chronic Obstructive Pulmonary Disease: A National Multicity Study. *American Journal of Epidemiology 2006*; 163: 579-588.

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

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

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

Table 9: From Medina-Ramón, et al., 2006.

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.[102]

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.[103]

A French study reported that ozone exacerbates symptoms in COPD patients. Thirty-nine senior adults with severe COPD were followed by their physicians in Paris, France, during a 14-month period. Daily levels of PM_{10}, ozone, sulfur dioxide and nitrogen dioxide were monitored. No evidence of symptom exacerbation and PM_{10}, SO_2, or NO_2 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 O_3. The recruitment of inflammatory cells into the lung presents a risk of tissue damage through the release of toxic mediators by activated inflammatory cells. Perhaps this phenomenon would be more serious among patients suffering from COPD, in whom a pre-existent inflammation of the small or large airways would be constant."[104]

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.

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

[102] 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.

[103] 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.

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

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 I, 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.[105]

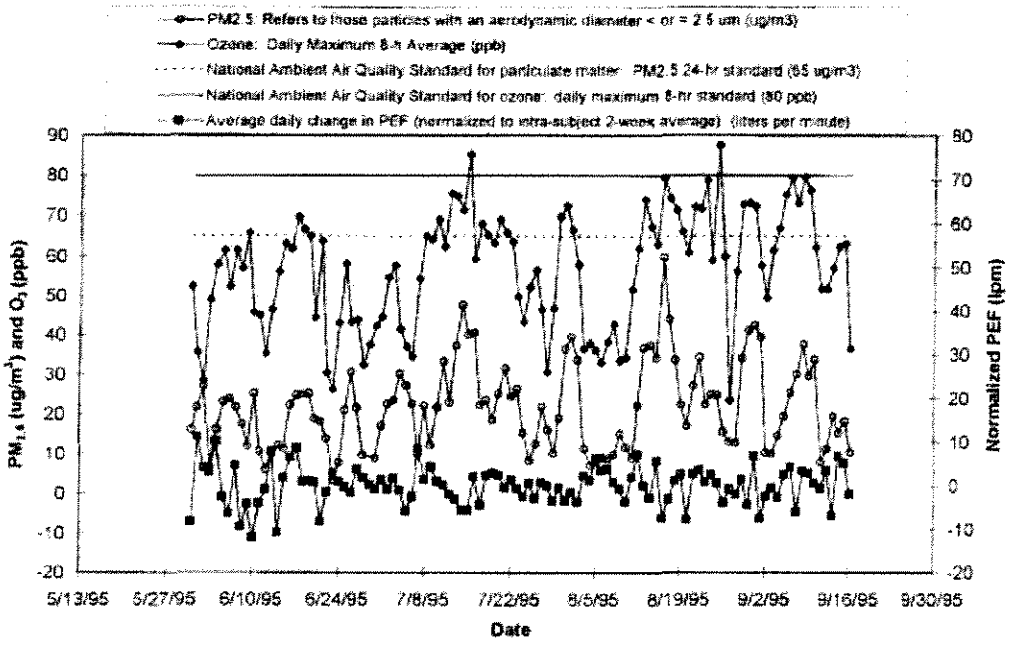

Figure 1. 24-h average $PM_{2.5}$ and daily maximal 8-h average O_3 at the Vinton stationary ambient monitoring site, and daily variations in morning PEF: summer 1995.

Figure I: From Naeher et al., 1999.

Additional Evidence from International Studies

We disagree with EPA's conclusion that only U.S. and Canadian studies are 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 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

[105] Naeher LP, Holford TR, Beckett WS, Belanger K, Triche EW, Bracken MB, Leaderer BP. Healthy Women's PEF Variations with Ambient Summer Concentrations of PM_{10}, $PM_{2.5}$, SO_4^{2-}, H^+, and O_3. *Am J Respir Crit Care Med* 1999; 160: 117-125.

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.[106]

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.[107]

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.[108]

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.[109] Children in Mexico City with the GSTM1 null genotype demonstrate significant ozone-related decrements in lung function.[110] 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 grater in individuals with certain genetic factors that make them more susceptible to oxidative stress.[111]

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[106] 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.

[107] Petroeschevsky 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.

[108] 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.

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

[110] 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.

[111] 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.[112] 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 co-pollutants.[113]

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.[114]

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.

[112] U.S. EPA, Air Quality Criteria, 2006. Page E-18.
[113] U.S. EPA, Air Quality Criteria, 2006. Page 8-78
[114] U.S. EPA, Air Quality Criteria, 2006. Page 8-38.

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. [115] 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.[116]

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:

> "[O]ur nationwide study provides strong and consistent evidence that daily changes in ambient O_3 exposure are linked to premature mortality, even at very low pollution levels, including an idealized scenario of complete adherence to current O_3 regulations."

Importantly even when days exceeding 0.060 were excluded from the analysis, the mortality effect was little changed. As indicated in Figure J 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 PM_{10}.[117] .

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[115] 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.

[116] 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.

[117] 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.

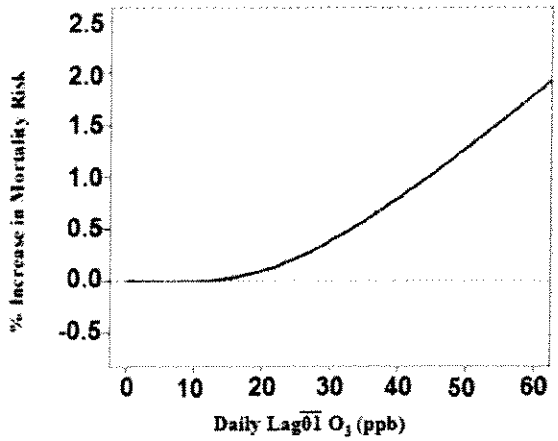

Figure J: Exposure Response Curve for ozone and mortality using the spline approach: percentage increase in daily nonaccidental mortality at various ozone concentrations.
Originally published in Bell, et al. 2006, taken from Bell, ML "Recent Evidence on the Relationship between Ozone and Mortality,"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

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.[118] 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.[119,120,121] The results of these meta-analyses are summarized in Figure K below, which illustrates the remarkable consistency in the findings.

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[118] 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 (O_3). 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.

[119] 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.

[120] Levy JI, Chermerynski SM, Samat JA. Ozone Exposure and Mortality: An Empiric Bayes Metaregression Analysis. *Epidemiology* 2005: 16: 458-468.

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

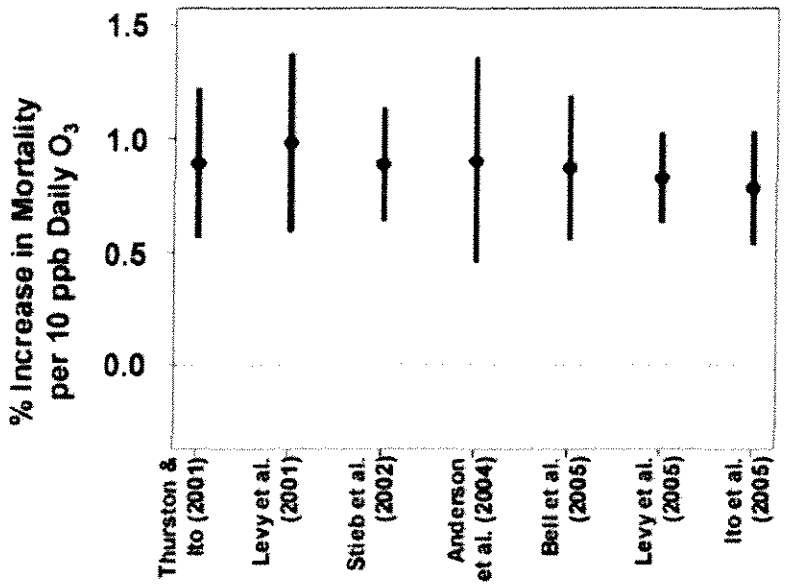

Figure K: Results of the Meta-Analyses studies. From Bell, ML. "Recent Evidence on the Relationship between Ozone and Mortality," Presentation to the Estimating Mortality Risk Reduction Benefits from Decreasing Tropospheric Ozone Exposure Panel, National Research Council on March 29, 2007

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 O_3 levels have a non-negligible impact in terms of public health."[122]

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.[123] 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

[122] 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 Perspec* 2006; 114: 1344-1347.

[123] 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.

to 2000. The study concluded that particulate matter is unlikely to confound the short-term association between ozone and mortality.[124]

These new studies estimate that cleaning up ozone air pollution could save thousands of lives each year.[125] The analysis by Bell et al. (2004) projects that nearly 4,000 lives would be saved per year by reducing ozone pollution from the current standard of 0.085 ppm to 0.075 ppm in the 95 U.S. cities studied. The larger the reduction in ozone pollution, the study concludes, the greater the number of lives that would be saved.[126] 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.[127]

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 pollutants including ozone and increased risk of infant death.[128] A study *in press* suggests that African-Americans may be at higher risk of early death from ozone pollution than the general population.[129] 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.[130]

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.[131]

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[124] 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 Perspec* 2007; doi:10.1289/ehp.10108, Online 2 August 2007.

[125] 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.

[126] 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.

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

[128] 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.

[129] 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.*

[130] 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.

[131] 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.

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 L 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.[132] 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."

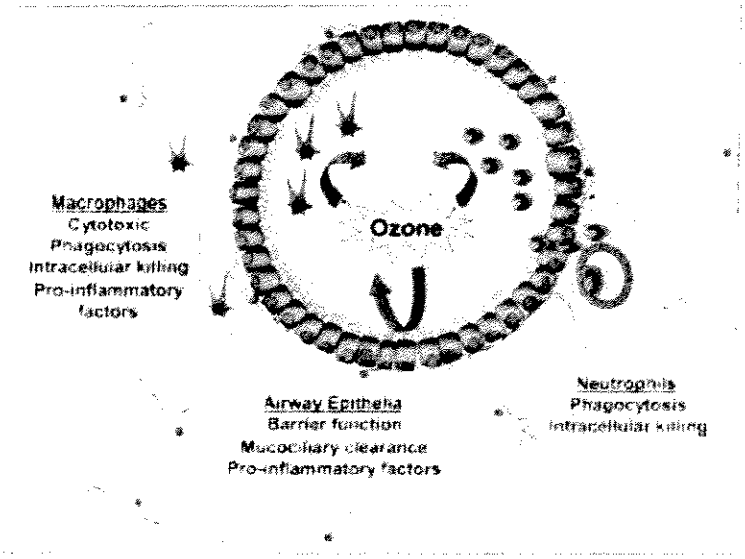

Figure L: Illustration of possible mechanisms for ozone interaction with cells in the lungs. From Hollingsworth et al 2007.

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.[133]

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

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

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.[134]

The Clean Air Scientific Advisory Committee also cites new evidence of mortality at concentrations below the current standard as part of their basis for recommending strengthened air quality standards. The committee unanimously recommended that EPA lower the ozone air quality standards due to evidence of death and disease at levels below current standards.[135] They specifically agreed that it is appropriate for EPA to include estimates of mortality risk associated with ozone exposure in its risk assessment.[136]

> "The understanding of the associated science has progressed to the point that there is *no longer significant scientific uncertainty regarding the CASAC's conclusion that the current 8-hr primary NAAQS must be lowered.* A large body of data clearly demonstrates adverse human health effects at the current level of the 8-hr primary ozone standard. Retaining this standard would continue to put large numbers of individuals at risk for respiratory effects and/or significant impact on quality of life including asthma exacerbations, emergency room visits, hospital admissions and mortality."[137]

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, researchers found a decline in lung function and an increase in respiratory symptoms in this group of amateur cyclists.

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[134] 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

[135] 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.

[136] 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. *See also*, U.S. EPA Proposed Rule, National Ambient Air Quality Standards for Ozone, June 21, 2002, p. 149.

[137] 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.

- 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 O_3 health effect, it is likely near the lower limit of ambient O_3 concentrations in the United States.[138]

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.[139]

However, using animal studies to support standard-setting requires 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).[140] Taken in this framework, the relatively high doses used in animal studies do not preclude them from consideration for standard-setting purposes.

[138] U.S. EPA. Air Quality Criteria, 2006. Page 7-159.
[139] Australian Government, National Health and Medical Research Council. Ambient Air Quality Standards Setting: An Approach to Health-Based Hazard Assessment, September 2006.
[140] 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.[141].

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 O_3....Again, caution should be exercised in extrapolating these observations to humans, due to species-specific differences...".[142]

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

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 where higher than attained in current ambient exposures, the implications are quite important."[143]

Eighty percent of lung development in humans occurs after birth continuing through adolescence.[144] 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

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[141] U.S. EPA. Air Quality Criteria, 2006. Page 5-34.
[142] U.S. EPA. Air Quality Criteria, 2006. Page 8-32.
[143] 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.
[144] American Academy of Pediatrics Committee on Environmental Health, Ambient Air Pollution: health hazards to children. *Pediatrics* 2004; 114: 1699-1707

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, hyperplasic bronchiolar epithelium, and altered smooth muscle bundle orientation in terminal and respiratory bronchioles.[145]

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.[146]

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."[147]

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[145] 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.

[146] 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.

[147] 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.

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 toxicology study in rats found that immature and aged rats displayed lung oxidative stress after ozone exposure, as compared to adult specimens. [148]

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, But Not Yet Proven, 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

[148] 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.

adults. This study reported that annual average ozone exposures were associated with poorly controlled asthma among elderly adults.[149]

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 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."[150] This effect of ozone was confirmed in a follow-up study.[151]

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."[152]

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.[153]

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 PM_{10}, nitrogen dioxide, and ozone monitors to the students residences. Lung function measurements were gathered between February and May, when the students had not had recent exposure to increased levels of ozone. The study found that lifetime exposure to ozone in adolescents 18-20 years old is associated with reduced

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[149] 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.

[150] 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.

[151] 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.

[152] 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.

[153] 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.

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.[154]

The California Children's Health Study annually measured the lung function of 1,700 fourth-graders enrolled in 1996, monitored the communities' air pollution for four years until 2000, and analyzed the relationships between their lung function growth and the levels of six pollutants. Exposure to ozone was correlated with reduced growth in peak flow rate. Larger deficits in lung function growth rate were observed in children who reported spending more time outdoors. Slower lung growth over a period of several years is evidence of a chronic effect of air pollution on children's respiratory health. Children whose lungs have grown more slowly may have lower maximum lung function as adults, making them more susceptible to respiratory diseases and chronic problems as they age.[155]

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 FEV_1 growth over the three year follow-up period were significantly associated with exposure to ozone and other pollutants in girls and boys. Over the course of the study period, 8-hour average ozone concentrations ranged from 60 ppb to 90 ppb. In multipollutant models, an interquartile range increase in mean ozone concentration of 11.3 ppb was associated with an annual deficit in FEV_1 of 12 ml in girls and 4 ml in boys. Early lung function deficits may increase the risk of developing chronic obstructive pulmonary disease later in life, as well increasing the risk of cardiovascular morbidity and overall mortality.[156]

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.[157]

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[154] 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.

[155] 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.

[156] Rojas-Martinex 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 Car Med* 2007; 176: 377-384.

[157] 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

Researchers found that the air pollution exposure produces significant chest X-ray abnormalities in the exposed children, depressed lung function, and an imbalance of blood proteins important to immune response. Twenty-two percent of the exposed children had grossly abnormal nasal mucosa, which can impair nasal defense mechanisms against inhaled gases and particles. The lung damage observed is similar to the chronic inflammatory damage observed in an earlier study of dogs in Mexico City. Researchers report that the x-ray and lung function changes they found in the exposed children could be due to pollution-associated chronic bronchiolitis, which could put the children at greater risk of developing chronic obstructive airway disease later in life. They conclude that lifelong exposure to urban air pollution causes respiratory damage in children and may predispose them to development of chronic lung disease and other problems due to suppression of the immune system.[158]

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."[159]

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

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.

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and Pulmonary Function of Children Chronically Exposed to Air Pollution. *Environ Health Perspect* 2006; 114: 1432-1437.

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

[159] Calderón-Garciduenas L, Valencia-Salazar G, Rogríguez-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.

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.[160]

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 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."[161] 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.[162]

A recent German study has reported that ozone and NOx 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.[163]

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 O_3 can directly and/or indirectly contribute to cardiovascular-related

[160] 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.
[161] Pinkerton KE, Balmes JR, Fanucchi MV, Rom WN. Ozone, a malady for all ages. *Am J Respir Crit Care Med* 2007; 176: 107-108.
[162] 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.
[163] Franze T, Weller MG, Niessner R, Pöschl. Protein Nitration by Polluted Air. *Enviro Sci Technol* 2005; 39: 1673-1678.

morbidity, but that much needs to be done to more fully substantiate links between ambient O_3 exposures and adverse cardiovascular outcomes (CD, p. 8-77)." [164].

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 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.[165]

A population-based study recently published in the journal *Circulation* after the publication of the draft Criteria Document reported that short-term exposures to ozone predict alterations in cardiac autonomic function as measured by heart rate variability among older adults.[166]

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.[167] 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.[168]

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

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[164] U.S. EPA. Staff Paper, 2007. Page 3-27.

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

[166] 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 Perspec* 2005; 113: 304-309.

[167] 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.

[168] 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 Perspec 2006*; 114: 120-123.

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 10. This study provides critical evidence for a far stricter standard than proposed by EPA.[169]

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} ($\mu g/m^3$)	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 10: 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.[170]

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.[171]

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[169] 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 Perspec* 2003; 111: 1312-1317.

[170] 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.

[171] 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.

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.[172]

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 $\pm$ 7 ppb, compared to 25.8 $\pm$ 5 ppb in controls. According to the authors, ozone may predispose children to type 1 diabetes by causing free-radical damage to ß-cells or enhancing the presentation of diabetes 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.[173]

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[174] 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 O_3, was observed in a longitudinal study of healthy you adults and was further supported by results from the controlled O_3 chamber study. In addition to published data showing effects of ambient O_3 exposure on DNA damage, common diseases and morality in humans,

[172] 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.
[173] Hathout EH, Beeson WL, Ischander M, Rao R, Mace JW. Air pollution and type 1 diabetes in children. *Pediatric Diabetes* 2006; 7: 81-87.
[174] U.S. EPA. Air Quality Criteria, 2006. Page 8-79.

> 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."[175]

Another study of 65 African American children and their mothers from Oakland, California, used geographic information systems to explore possible associations between chromosomal damage 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.[176]

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.[177]

A study in São Paulo, Brazil, found that ozone exposure was correlated with tumors of the larynx and lung.[178]

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. Salam et al 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

[175] 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.
[176] 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.
[177] 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.
[178] 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.

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.

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.[179]

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.[180]

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.[181]

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[179] 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 Perspec* 2005; 113: 1638-1644.

[180] 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.

[181] 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.

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.

Ultraviolet Radiation

The Administrator requested comment on the possible beneficial role that ground-level ozone might have on prevention of ultraviolet radiation exposure. The Criteria Document acknowledged the many risks associated with increased UV-B radiation, including "erythema, nomelanoma and melanoma skin cancers, ocular damage, and immune system suppression." The Criteria Document concluded that although some research has attempted to assess the risks of increased UV-B exposure from the reduction of stratospheric ozone, "the numerous simplifying assumptions made in the assessments limit the usefulness of the risk estimates."[182] To adequately assess the risks of humans would require significantly improved assessment of exposure in individuals who participate in outdoor activities where they might likely risk greater UV-B exposure. The Criteria Document also discussed the health-benefits of increased UV-B radiation on the production of vitamin D in humans, noting that "as with other impacts of UV-B on human health," this lacked sufficient evidence to draw conclusions.[183] We concur with the findings in the Criteria Document as cited in the Staff Paper that:

> "the effects of changes in surface-level O3 concentrations on UV-induced health effects cannot be critically assessed given the significant uncertainties summarized above." [184]

We also agree with the recommendations contained in the letter from the American Thoracic Society et al to the Administrator, cited earlier, which advised:

> "Given the known adverse health effects of ozone, however, it would appear more prudent to focus on current approaches to reducing ultraviolet radiation by preventing UV exposures, rather than permitting the harmful effects of ozone. We are aware of no compelling evidence that should persuade the Administrator to consider this issue when setting the NAAQS ozone standard." [185]

The Form of the Standard

The health protectiveness of the standard is a function of the form as well as the level of the standard. EPA's policies that allow multiple exceedances and rounding seriously erode the public health protections provided by the standard.

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[182] U.S. EPA. Air Quality Criteria, 2006. Page E-32 and page 10-38.
[183] U.S. EPA. Air Quality Criteria, 2006. Page E-31 and E-32.
[184] U.S. EPA. Staff Paper, 2007. Page 3-36.
[185] Letter from the American Thoracic Society, et al. October 9, 2007.

Number of Exceedances Must be Decreased

Currently, EPA calculates the average of the fourth highest daily maximum over a three year period to judge attainment status. EPA has asked for comment on whether an alternate number of exceedances should be considered in revising the standard. Specifically, the Agency has asked for comment on whether this form of the standard should be retained, or whether the third of fifth highest concentrations should be used to determine nonattainment. Given that range, we would urge EPA to select the most protective option—that of the third highest concentration each year. We oppose using the fifth maximum concentration, which would relax the standard and permit additional days each year of unlimited ozone exposures, with attendant health consequences, and greatly limit the number of people that receive protection under the ozone standard. Fundamentally, however, we disagree with the current method of ignoring the highest exceedances.

In the past, EPA has allowed a number of exceedances to occur before a violation of the standard is triggered, officially ignoring a certain number of days and hours with the highest concentrations. Since exposures at high concentration levels trigger adverse effects, it stands to reason that the highest concentrations can do the most harm. Concentrations at this level must not be ignored in a standard that is based on the need to protect the health of the public.

Alternate forms of the standard greatly affect the protectiveness of the standards. In fact, the percent of the population receiving protection under a particular standard is as much a function of the form as it is of the level of the standard. Thus if EPA lowers the level of the standard but relaxes the form, it effectively blunts the impact of the new standard. For example, a standard of 0.074 ppm averaging the 5th highest values, would provide protection to 5 percent fewer counties than a standard of 0.074 ppm averaging the 4th highest.[186]

Because short-term increases in ozone exposure can trigger a variety of adverse respiratory events, each excused exceedance has potentially life-threatening health consequences. Under the current standards, nine high-concentrations days (with unlimited levels) are ignored in each 3-year period. Although it would provide more protection than the current form, even a form built on the third highest value would allow six days of unlimited ozone levels to occur creating serious risks to health.

We note that California adopted a "not-to-be exceeded" form for its 8-hour ozone standard adopted in 2005, at 0.070 ppm. That form is significantly more stringent than the current form of the federal standard.

[186] U.S. EPA Staff Paper, Predicted percent of counties with monitors (and percent of population in counties with monitors) not likely to meet alternative ozone standards. p. 6A-1.

The Rounding Loophole Must be Eliminated

The standard adopted in 1997 allowed 0.084 ppm concentrations to be rounded down to the standard of 0.08 ppm before triggering a violation, effectively relaxing the level of the standard. State implementation plans were not required until the 4th highest maximum concentration averaged over 3 years reached 0.085 ppm or above. This allowed many places, like Denver, Colorado, to continually report ozone levels above the official standard of 0.08 ppm, but below the effective standard of 0.085 ppm. For example, Denver reported its highest ozone design values as 0.084 ppm in 2000-2002, marking it in compliance with the ozone NAAQS. Only in until the next year, when the 2001-2003 design value increased to 0.087 ppm, did Denver move into violation of the standard.[187]

A paper published in the *Journal of the Air and Waste Management Association* examined the rounding convention used in the 1997 ozone standard. One rationale for the rounding convention has been that the design value for the ozone standard is biased upward, and that rounding compensates for this overshoot bias. The analysis found that while there can be substantial overshoot basis in the design value of the older one-hour standard, this is much less true for the new 8-hour standard. The new ozone standard may have little overshoot bias and may be within 3 percent of the true value most of the time. Thus rounding may tend to misclassify nonattainment areas as attainment, and serves, in effect to weaken the standard by 5 ppb.[188]

According to the CASAC panel, ozone monitoring instrumentation has improved in the last ten years and is now considerably more precise, thus allowing ozone concentrations to be measured to the third significant digit.[189] Adding a significant digit to the standard, as unanimously endorsed by the CASAC Ozone panel, will eliminate the rounding problem.

We note that the 2005 8-hour ozone standard adopted by the California Air Resources Board was expressed in terms of three significant digits: 0.070 ppm.

We support EPA's proposal to eliminate the rounding loophole and set a standard in terms of three significant digits.

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[187] Colorado Air Quality Control Commission. Early Action Compact Ozone Action Plan: Revisions to the State Implementation Plan. Amended, December 15, 2005. Page 11.

[188] Fairley D. Overshoot Bias and the National Ozone Standard. *J. Air & Waste Manage Assoc* 1999; 49: 370-385.

[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.

EPA should reinstate the one-hour standard

The proposal fails to give adequate consideration to additional averaging times for the ozone standard. Health evidence of effects following one- to two-hour exposures from prior reviews clearly supports the need for a 1-hour standard. In addition, analysis showing the differences in exposure profiles in different parts of the country supports having both an 8-hour standard and a 1-hour standard.

The American Lung Association has consistently supported retention of a one-hour average standard, in conjunction with the implementation of the new eight-hour average standard for ozone.

Comments from the California Air Resources Board indicate that a stringent 1-hour ozone standard is needed to protect the cities in the coastal zone of the state where the majority of the population lives.[190]

When EPA last revised the ozone standards in 1997, the Agency was aware that Houston and parts of Los Angeles as well as four other areas analyzed have different air quality profiles that suggest the need for a stringent 1-hour standard in conjunction with the 8-hour average standard.[191] The results of the draft exposure analysis bear this out. Included in the draft Staff Paper, that analysis indicated that few children in Houston, Sacramento and Los Angeles would be exposed to 8-hour ozone exceedences above various cut points[192], as compared to children in cities in the East and Mid-West. An ozone standard cannot protect public health if it fails to respond to the reality of ozone exposures in the most notoriously polluted cities in the United States. National Ambient Air Quality Standards must be designed to protect the health of the public, even in places with atypical peak to mean ratios. The 8-hour ozone standard must be a supplement to, not a substitute for, the 1-hour standard.

Table 11 is drawn from a paper examining a large number of studies of the effects of hourly ozone concentrations on the lung function, symptom exacerbations or hospital admissions of subjects exercising outdoors. A large number of these studies found effects at ozone levels below 240 $\mu g/m^3$ (120 ppb). Effects were observed down to 80 and 60 ppb.[193]

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[190] Letter from Catherine Witherspoon, Executive Officer, California Air Resources Board, to Mr. Fred Butterfield, CASAC DFO, EPA, August 17, 2006 and attached "Comments from the California Air Resources Board on the U.S. EPA Second Draft Staff Paper for the Ozone NAAQS (including 8/8/06 revisions).

[191] Final Rule, National Ambient Air Quality Standards for Ozone, Federal Register Vol. 62, No. 138, July 18, 1997.

[192] U.S. EPA. Draft Staff Paper, 2006. Table 4-9.

[193] Brunekreef B, Dockery DW, Krzyzanowski M. Epidemiologic Studies on Short-Term Effects of Low Levels of Major Ambient Air Pollution Components. *Environ Health Perspect* 1995; 103 (Supple 2): 3-13.

Table 1. Summary of studies relating hourly ozone concentrations of less than 240 μg/m³ with specific effects on human health

	Category of health effects			
Concentration	Hospital admissions	Symptom exacerbations	Lung function changes	Reference
< 100	+			Pönkä (6)
< 240	?			Thurston et al. (7)
< 240	+			Thurston et al.(8)
< 240			+	Avol et al. (9)
< 240		–	+	Spektor et al. (11)
< 240		+		Berry et al. (12)
< 240		–	+	Hoek (16); Hoek et al. (17)
< 240		–	+	Hoek et al. (18)
< 200			+	Krzyzanowski et al. (15)
< 200			+	Korrick et al. (20)
< 160			+	Spektor et al. (19)
< 160			+	Kinney et al. (14)
< 160			–	Thurston et al. (13)
< 160			+	Braun-Fahrländer et al. (22)
< 120		–	+	Spektor et al. (10)
< 120		+	+	Brunekreef et al. (21)

Table 11: From Brunekreef et al., 1995.

There are numerous older studies of one- to two-hour exposures that formed the basis for the prior one-hour standard of 0.12 ppm set in 1979, as illustrated in Table 12.

Table 12: Key Health Studies Supporting the Current 1-Hour National Ambient Air Quality Primary Standard for Ozone, From the U.S. EPA Criteria Document for Ozone and Related Photochemical Oxidants, July 1996.

TABLE V-1. KEY HEALTH STUDIES SUPPORTING THE CURRENT 1-HOUR NATIONAL AMBIENT AIR QUALITY PRIMARY STANDARD FOR OZONE

O_3 Concentration, ppm	Health Effect	Reference
Ambient air containing 0.01-0.14 daily 1-hr max over days to weeks	Decrements in lung function in children, adolescents and adults exercising outdoors	Berry et al. (1991) Bock et al. (1985) Higgins et al. (1990) Kinney et al. (1989) Lioy and Dyba (1989) Lioy et al. (1985) Lippmann et al. (1983) Raizenne et al. (1987, 1989) Spektor et al. (1988a,b; 1991)
≥0.12 (1-3 hr) or ≥0.08 (6.6 hr) (chamber exposures)	Decrements in lung function (reduced ability to take a deep breath), increased respiratory symptoms (cough, shortness of breath, pain upon deep inspiration), increased airway responsiveness and increased airway inflammation in heavily exercising adults	Adams et al. (1981) Avol et al. (1983, 1984) Devlin et al. (1991) Folinsbee and Horvath (1986) Folinsbee et al. (1978, 1984, 1988) Gibbons and Adams (1984) Gliner et al. (1983) Horstman et al. (1990) Koren et al. (1989a,b, 1991) Kulle et al. (1985) Lauritzen and Adams (1985) Linn et al. (1980, 1983a,b, 1986, 1988) McDonnell et al. (1983, 1991) Seltzer et al. (1986)

Table 12: Key Health Studies Supporting the Current 1-Hour National Ambient Air Quality Primary Standard for Ozone, Continued: From the U.S. EPA Criteria Document for Ozone and Related Photochemical Oxidants, July 1996.

O_3 Concentration, ppm	Health Effect	Reference
≥ 0.12 (1-3 hr) (chamber exposures)	Decrements in lung function in heavily exercising children and adolescents	Avol et al. (1985a,b,c, 1987) Koenig et al. (1987, 1988) McDonnell et al. (1985)
≥ 0.12 (1-3 hr) (chamber exposures)	Effects are similar in individuals with preexisting disease except for a greater increase in airway responsiveness for asthmatic and allergic subjects	Koenig et al. (1985, 1987, 1988) Kreit et al. (1989) McDonnell et al. (1987)
≥ 0.12 (1-3 hr) (chamber exposures)	Older subjects (>50 yr old) have smaller and less reproducible changes in lung function	Bedi and Horvath (1987) Bedi et al. (1988, 1989) Drechsler-Parks et al. (1987, 1989, 1990) Reisenauer et al. (1988)
≥ 0.18 (1-3 hr) (chamber exposures)	Reduced exercise performance in heavily exercising adults	Adams and Schelegle (1983) Folinsbee et al. (1984) Gong et al. (1986) Linder et al. (1988) Schelegle and Adams (1986)
≥ 0.12 (1-3 hr) (chamber exposures)	Attenuation of lung function response with repeated exposure	Avol et al. (1988) Farrell et al. (1979) Hackney et al. (1976, 1989) Horvath et al. (1981) Kulle et al. (1982) Linn et al. (1982, 1988)

Table 12: Key Health Studies Supporting the Current 1-Hour National Ambient Air Quality Primary Standard for Ozone, Continued: From the U.S. EPA Criteria Document for Ozone and Related Photochemical Oxidants, July 1996.

O_3 Concentration, ppm	Health Effect	Reference
≥0.12 with chronic, repeated exposure (chamber exposures)	Changes in lung structure, function, and biochemistry in laboratory animals that are indicative of airway irritation and inflammation with possible development of chronic lung disease	Amdur et al. (1978) Barry et al. (1983, 1985, 1988) Boorman et al. (1980) Castleman et al. (1977, 1980) Chow et al. (1981) Costa et al. (1983) Crapo et al. (1984) Eustis et al. (1981) Filipowicz and McCauley (1986a,b) Fujinaka et al. (1985) Grose et al. (1989) Last et al. (1979,1984) Moore and Schwartz (1981) Mustafa et al. (1985) Plopper et al. (1979) Rao et al. (1985a,b) Schwartz et al. (1976) Sherwin and Richters (1985) Tyler et al. (1988) Wegner (1982) Wright et al. (1988)
≥ 0.08 (3 hr) or ≥ 0.10 with chronic repeated exposure (chamber exposures)	Increased susceptibility to bacterial respiratory infections in laboratory animals	Coffin et al. (1972) Ehrlich et al. (1977) Miller et al. (1978) Aranyi et al. (1983)

Nothing in the newer literature exploring effects of longer term exposures precludes the need to limit one-hour exposures to protect against short-term health effects. Indeed, in the 1995 review of the California ozone standard, the Air Resources Board decided to retain their one-hour standard of 0.09 ppm, while adding a new 8-hour average standard.

Effects of ozone air pollution have been studied under natural conditions in children attending summer camp. Kinney et al. performed a post-1996 reanalysis of six of the earlier summer camp studies, including two New Jersey studies, two Ontario studies, and two studies in southern California. All of the studies found that increased ozone was associated with decreased FEV_1. Mean hourly ozone concentrations in four of the six study areas were less than 71 ppb, as shown in Table 13. In two of the studies, mean ozone concentrations were less than 60 ppb.[194] According to the authors:

> "the results of this reanalysis provide strong evidence that children exposed to O_3 under natural conditions experience decreases in FEV_1 of the kind demonstrated in laboratory studies, and raise concern that other acute respiratory effects observed in those studies (e.g. pulmonary inflammation) may also occur in young people exposed to ambient O_3."

Table 1. Key descriptive statistics for six studies of the lung function response of children to air pollution

Study	Total no. of subjects	Total no. of observations	Mean observations/ subject	Mean O_3 (ppb)[a]	Maximum O_3 (ppb)	Mean FEV_1 (l)	Mean PEFR (l/sec)
Fairview Lake, 1984	91	1237	13.6	53	113	2.14	4.36
Fairview Lake, 1988	46	577	12.5	69	137	2.39	NA
Lake Couchiching, 1983	29	244	8.4	59	95	2.41	5.48
CARES, 1986	112	1228	11.0	71	143	2.34	5.51
San Bernardino, 1987	43	255	5.9	123	246	2.06	5.07
Pine Springs, 1988	295	1826	6.2	94	161	2.19	4.52

Abbreviations: FEV_1, forced expiratory volume in 1 sec; PEFR, peak expiratory flow rate; NA, PEFR data not available for this study.
[a]1-hr average, at time of afternoon lung function measurement.

Table 13: From Kinney et al., 1996.

Another more recent camp study focused on children ages 7 to 13 attending a summer "asthma camp." The pollutant most consistently associated with adverse consequences was ozone. An increase in the 1-hour daily maximum concentration of ozone from 84 to 160 ppb was associated with an increase in the number of unscheduled medications administered and an increase in the chest symptoms per day.[195]

More recent 6- to 8-hour studies that employ triangular exposures, that is, concentrations that begin at a low level, rise to a peak, and return to a low level over the course of the exposure

[194] Kinney PL, Thurston GD, Raizenne M. The effects of ambient ozone on lung function in children: a reanalysis of six summer camp studies. *Environ Health Perspect* 1996; 104: 170-174.
[195] Thurston GD, Lippmann M, Scott MB, Fine JM. Summertime haze air pollution and children with asthma. *Am J Respir Crit Care Med* 1997; 155: 654-660.

suggest that peak exposures such as are experienced in some locations such as Houston and Los Angeles are important in terms of ozone effects on respiratory function and respiratory symptoms.[196] One interpretation of the triangular exposures in the Adams studies is that peak 1-hour exposures to ozone are important and induce adverse health effects, even at low concentrations below 0.08 ppm.

These recent studies support the need to reinstate the 1-hour ozone standard.

In 1997, EPA argued that a one-hour standard was unnecessary because most areas that would fail to meet the one-hour standard would also be out of attainment with the new 8-hour standard. This argument fails on two counts. First, it assumes that successful reduction of ozone precursors to meet the 8-hour standard will protect against peak hourly ozone exposures. This is not necessarily true, as discussed below. Second, it assumes that no areas will have levels of ozone that violate the 1-hour standard if they meet the 1997 8-hour standard. This assumption is disproved by EPA's own analysis.

The Clean Air Act requirement to protect the public from harm from ozone clearly includes areas where air quality patterns are anomalous with such patterns elsewhere. EPA recognized this requirement just last year, when it adopted a 24-hour $PM_{2.5}$ standard, as well as an annual standard.

EPA has provided real-world evidence that meeting the 8-hour standard does not prevent peak 1-hour exposures. An EPA analysis of 1-hour and 8-hour ozone design values indicates that a number of metropolitan areas could potentially meet the current 8-hour ozone standard, yet have high 1-hour concentrations in excess of the prior standard.[197] These areas include such diverse cities as San Francisco, California, Portland, Maine, and Greensboro, North Carolina. Residents of these cities deserve equal protection.

Further, states will likely need to employ different air pollution control strategies might be necessary to reduce peak 1-hour concentrations, than just those focused on reducing daily concentrations. Despite dropping 8-hour concentrations over the past ten years, cities like Los Angeles and Houston still have not tamed the 1-hour peaks that would continue to violate the 1979 standard. These cities will need additional and different control strategies to eliminate these challenging peak concentrations.

We urge EPA to reinstate the one-hour ozone standard.

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[196] 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.

[197] U.S. EPA memorandum from Lance McCluney, EPA-OAQPS to Ozone NAAQS Review Docket (OAR-2005-0172). Subject: Ozone 1-Hour to 8-Hour Ratios for eh 2002-2004 Design Value Period, January 18, 2007.

"Exposures of Concern" Metric is an Inappropriate Basis for Decision-Making

EPA premises a large part of its argument in favor of a standard between 0.075 and 0.070 ppm on a metric not contemplated by the Clean Air Act called "exposures of concern." The preamble relies heavily upon the results of the exposure analysis in school-aged children to justify the proposed range:

> "The Administrator also has considered the results of the exposure assessments in reaching his judgment that a standard level below 0.070 ppm would not be appropriate. ... a 0.070 ppm standard would be expected to provide protection from the exposures of concern that the Administrator has primarily focused on for over 98 percent of all and asthmatic children even in a year with relatively high O_3 levels, increasing to over 99.9 percent of children in a year with relatively low O_3 levels (2004)." 72 FR 37880.

Fundamentally, we oppose the "exposures of concern" metric as EPA employs it here. EPA's use of the "exposures of concern" argument assumes that the basis of the standard is how much ozone Americans inhale, not what effects are induced at specific concentrations. This metric builds on the false assumptions that people who are not outdoors need less protection and, most tellingly, that EPA can raise the acceptable level of ozone if they assume fewer people are outdoors. Extending that argument would allow EPA to set a standard at 80 ppm, or 800, if they could just calculate that everyone stayed inside. EPA should set the standard based on the levels shown by the health studies to cause adverse effects.

In addition, there are serious technical flaws in EPA's analysis. In brief, the exposures of concern metric is of limited utility in assessing the effect of the proposed rule, because it considers a limited population, uses a circular argument to define exposures of concern, and considers only limited health endpoints, ignoring effects demonstrated by epidemiological studies.

We discuss exactly what is and isn't implied by the exposure analysis, and why it is erroneous to use it to justify a standard no more stringent than 0.070 ppm.

Overview of the Exposure Assessment

EPA developed the exposure assessment primarily for use in the risk assessment (more detailed discussion follows). More specifically, the exposure assessment was intended to be used as an input to the portion of the risk assessment that estimated lung function declines in school-age children in 12 urban areas associated with various ozone standards. The risk

function for FEV_1 decrements was obtained from controlled human exposure studies that measured lung function in relation to known concentrations of ozone under conditions of moderate exercise. Therefore, to estimate risks of lung function decline, EPA needed exposure estimates to characterize exposures to certain concentrations of ozone under conditions of moderate exercise.

To generate these exposure estimates, EPA used the Consolidated Human Activity Database (CHAD) to obtain time-activity data for use in the Air Pollutants Exposure (APEX) model in conjunction with air quality monitoring information for the 12 cities examined in the risk assessment. The CHAD database used activity diaries to collect information on the amount of active time spent outdoors in sample populations.

Weaknesses in the Characterization of "Exposures of Concern"

EPA cannot use a very limited subset of the already narrow exposure assessment to draw policy conclusions about the level of the proposed standards. Even if it were appropriate, however, there are at least twelve fundamental limitations in the "exposures of concern" metric that preclude its use in making rational judgments about standards requisite to protect public health with an adequate margin of safety.

Limitation 1: EPA bases its rationale on a circular argument. The EPA Administrator defines "exposures of concern" as a benchmark level of 0.070 ppm. It is a totally circular argument to state that "exposures of concern" would be almost eliminated with a standard of 0.070, when EPA is defining "exposures of concern" as concentrations above 0.070 ppm. As a member of the CASAC ozone panel stated: "thousands of sensitive children will continue to experience ozone exposures of concern and resulting lung function decrements (and other health effects) at or below 0.07 ppm."[198] If EPA pursues this analysis over our objections, commenters assert that "exposures of concern" must be defined as 0.060 ppm, based on the considerable health evidence of adverse effects occurring at this level. It is disingenuous for EPA to argue that exposure response relationships are "uncertain" down to the 0.060 ppm level for this "exposures of concern" analysis. EPA's risk assessment is based on the exposure-response function in the Adams studies, in which experimental exposure regimes extended down to 0.04 ppm.

Limitation 2: School absences, increased use of asthma medication, emergency room visits, and hospital admissions are not accurately reflected. Contrary to EPA's assertion, the exposure estimates cannot be generalized beyond the effects studied in the controlled human exposure studies -- that is, primarily lung function decrements and respiratory symptoms. Other health endpoints, such as school absences, increased use of asthma medications, long-term deficits in lung function and associated risk of illness, emergency room visits, hospital admissions, and premature deaths, have been characterized principally

[198] Comments of Henry Gong, Jr., M.D. in 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.

in epidemiological studies, where increased risks are a function of the ambient concentrations. The exposure analysis, which relies on activity profiles to minimize assumed population exposures, is not relevant to the estimation of those health endpoints which have been reported in epidemiological studies. This limitation contradicts EPA's desired use of the exposure assessment to "provide some perspective on the public health impacts of health effects that we cannot currently evaluate in quantitative risk assessments." 72 FR 37853.

Limitation 3: Risks to children are underestimated. The chamber studies did not test children, so there is uncertainty entailed in extrapolating the response rate of healthy adults to responses in healthy children, or in children with lung disease. It is more than likely that this assumption leads to an underestimation of risks and "exposures of concern."

Limitation 4: EPA's model underestimates repeated exposures. As EPA acknowledges, CHAD underestimates the frequency of occurrences of "repeated routine behavior."[199] This results in underestimates of exposures to children who spend large portions of their summers playing outside or in summer camps.

Limitation 5: The exposure assessment discounts the most vulnerable. The exposure assessment focuses on the average child rather than the highly exposed. This analytical approach is then used to argue against adequate protection for those active children or adults that spend a lot of time outdoors. Because EPA averages the activity patterns of active and sedentary people, standards may not protect the most exposed individuals.

Limitation 6: Exposure estimates don't account for ozone avoidance behaviors. The exposure analysis does not consider the effect of ozone avoidance behavior on activity profiles. People living in the 12 cities examined experience frequent ozone alerts warning them of unhealthy air quality and the need to avoid exercising outdoors. Schools, day cares and day camps routinely confine children indoors on code red days. The analysis fails to consider the extent to which ozone avoidance behavior has diminished the estimates of outdoor exercise in children.

Limitation 7: Active children are not well characterized. The exposure and risk assessments do not adequately capture risks to active children. EPA had initially profiled exposures of "active" children as a separate subpopulation, but subsequently dropped this category, and considers only exposures to average children.

Limitation 8: Babies, toddlers and preschoolers are left out of EPA's count of number of children. Toddlers and preschoolers, an important segment of the population that spends lots of time playing outdoors, are not factored into the analysis. Only school-aged children are included in EPA's estimates.

Limitation 9: Outdoor workers and other adults aren't considered. The "exposures of concern" assessment does not include outdoor workers or outdoor recreation enthusiasts who receive higher inhaled doses of ozone due to their increased ventilation rates.

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[199] U.S. EP. Staff Paper, 2007. Pp. 4-36 - 4-37.

Limitation 10: Most of the country is excluded from the analysis. The geographic scope of EPA's analysis is limited to just 12 metropolitan areas.

Limitation 11: Exposures to downwind populations are excluded. The exposure assessment does not account for exposures and health impacts that result from ozone transported from the 12 MSAs analyzed, which can actually result in people downwind being exposed to higher concentrations.[200]

Limitation 12: Choice of baseline year distorts the estimates. Ozone concentrations vary from year to year with different weather conditions. Estimates of "exposures of concern" are subject to great variability depending on whether the baseline year for comparison is 2002, a relatively dirty year, or 2004, a relatively clean year. The risk and exposure analysis must focus on 2002 as a baseline year. Use of a year with favorable meteorology as the baseline year distorts exposure estimates.

EPA Cannot Use the "Exposures of Concern" Metric to Justify a Weak Standard

Taken with our fundamental objection, these twelve flawed assumptions render the "exposure of concern" analysis unsuitable to justify the proposed standards. EPA claims that by choosing a standard in the range of 0.070 to 0.075 ppm, it will protect all but some 2 to 4 percent of school-age children with asthma from high ozone levels in the 12 cities studied. 72 FR 37879. EPA argues that these percentages are acceptable. As shown Table 14 below, if EPA adopts a standard in the proposed range, roughly 39,000 to 78,000 asthmatic children will continue to be exposed to demonstrably unhealthy levels of ozone pollution in these 12 cities. As discussed above, the Act does not give EPA the option choosing a NAAQS that allows such adverse effects. Of course, children with asthma are only one of the many groups of Americans at increased risk, and these are only 12 cities—clearly not an assessment of the total population left unprotected.

[200] CASAC March 2007, Comments of Barbara Zielinska (p. C-35) and Mort Lippman (p. C-20).

	Number of Children with Asthma	2% unprotected under EPA Proposal	4% unprotected under EPA proposal
Atlanta	123,505	2,470	4,940
Boston	135,080	2,702	5,403
Chicago	226,479	4,530	9,059
Cleveland	62,360	1,247	2,494
Detroit	123,691	2,474	4,948
Houston	134,713	2,694	5,389
Los Angeles	438,040	8,761	17,522
New York	474,998	9,500	19,000
Philadelphia	138,859	2,777	5,554
Sacremento	49,948	999	1,998
St. Louis	61,333	1,227	2,453
Washington DC*	181,576	3,632	7,263
Totals	2,150,582	43,012	86,023

*Washington DC-Baltimore Combined Metro Area

Table 14: Estimated Number of Children with Asthma Unprotected by EPA's Proposed Range of 0.070 to 0.075 ppm in 12 U.S. Cities, 2005. American Lung Association calculations based on 2005 National Health Interview Survey.

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 proposed range for the revised ozone standard of 0.070– 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. For example, whereas an estimated 220,000 children experienced decreased lung function capacity during the 2002 ozone season in Los Angeles, the adoption of a 65 ppb ozone standard would have reduced this number by 97% to 7,000 children.[201] According to the risk assessment, similar improvements in lung function would be seen in cities across the country.

[201] U.S. EPA. 2007 RIA, Tables 3-16, and 3-17.

The EPA risk assessment shows that relative to the current standard, a standard of 0.065 ppm—not our preference, but 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;
- 30 percent the days that asthmatic children in Boston experience respiratory symptoms;
- 35 percent the respiratory hospital admissions in New York City attributable to ozone exposures; and
- 75 percent fewer ozone related deaths in the 12 cities studied.[202]

These estimates demonstrate that while a standard of 0.065 ppm would significantly decrease ozone-related lung function decrements, respiratory symptoms, hospital admissions, and mortality, we need additional protection. 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 a more adequate margin of safety.

More specifically, the analysis of symptoms in asthmatic children in the Boston area estimates that every 100,000 children living in the Boston area will experience 20,000 – 30,000 symptoms of chest tightness and shortness of breath during the ozone season, depending on conditions. Adoption of the 65 ppb ozone standard would decrease reported symptoms by 58 percent to 66 percent.[203] As EPA itself notes,

> "...even after the current 8-hr standard is met in a year with generally better air quality, among children with moderate to severe asthma in the Boston area, as many as one symptom day in 8 during the O_3 season is estimated to be attributable to O_3 exposure. In a year with generally poorer air quality, as many as one symptom day in 6 is estimated to be attributable to O_3 exposure."[204]

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[202] 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 O_3 NAAQS. HEI Annual Conference, Chicago, IL April 17, 2007.

[203] U.S. EPA. Regulatory Impact Analysis of the Proposed Revisions to the National Ambient Air Quality Standards. EPA-452/R-07-008, July 2007. Tables D3, D4, (p. D-10-D-11) and Tables E8, and E11 (p. E-15 and E-18).

[204] U.S. EPA. 2007. Staff Paper, p. 6-36.

The Risk Assessment Systematically Underestimates Health Risks

The EPA limited risk assessment was developed to explore the health implications of alternate standards, but its estimation of risks is extremely conservative. 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 include failure to analyze risks from exposure below so-called "background" concentrations, 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.

EPA Should Not Discount Background Concentrations

In the Risk Assessment, estimates of health effects are calculated based on ozone exposure above policy relevant background (PRB) concentrations. EPA defines PRB ozone as "the distribution of O_3 concentrations that would be observed in the U.S. in the absence of anthropogenic (man-made) emissions of precursor emissions (e.g., VOC, NOx, and CO) in the U.S., Canada, and Mexico."[205]

The labeling and subsequent discounting of a portion of the ozone burden in the U.S. as "policy relevant background" greatly understates the risks of ozone exposure. There is no rational basis for excluding background concentrations from the risk assessment. The actual risk from ozone exposure comes from the total exposure, not just a portion thereof. Risks must be evaluated based on total exposure to ozone. Excluding exposures below background from the risk estimates distorts the risks of exposure to stated concentrations.

EPA's definition of policy relevant background includes, and consequently devalues, anthropogenic ozone that comes from outside North America. Ozone blown in from Asian nations, for example, is currently considered background ozone, even though it is heavily anthropogenic.[206] Controlling such sources would require international agreements, which would likely be difficult. However, the ozone they create contributes to the same health effects as ozone generated by domestic sources.

[205] U.S. EPA. Staff Paper, 2007. page 2-48.

[206] Garrett L, Long, JCS. "Cutting Through China's Smoke," *Los Angeles Times*, October 7, 2007.

Members of CASAC argued that calculation of PRB may be unnecessary—that the purpose of the risk assessment is to evaluate relative risks of various policy options, and that the difference between options is more important than the absolute risks.[207]

EPA has illegally understated the health effects of ozone by basing its risk assessment solely on risks in excess of policy-related background levels. Section 109 of the Act requires the primary NAAQS to be set at a level requisite to protect public health with an adequate margin of safety: it does not allow EPA to protect only against health risks presented by ozone attributable to anthropogenic sources. A person breathing the air is exposed to the total concentration of ozone in the air, including background, and the NAAQS must protect against that total exposure. There is nothing in the Act that allows EPA to ignore or discount the risk presented by the background component of such exposures.

While we disagree with EPA's approach of estimating risks only above policy relevant background, 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 NOx; long range transport of O_3 and O_3 precursors from outside of North America, and exchange of ozone between the stratosphere and troposphere.[208] 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.[209] 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.[210]

While the GEOS-CHEM model has received generally high marks from both EPA and the CASAC, it does overestimate PRB ozone in the southeastern U.S., which minimizes risk estimates in Atlanta, one of the cities modeled in the risk assessment. According to the EPA, several papers have evaluated the accuracy of the GEOS-CHEM simulation and demonstrated that PRB ozone values are inflated in the southeastern U.S. by as much as 10 ppb.[211]

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[207] 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, p. 12.
[208] U.S. EPA. 2007. Staff Paper , p. 2-54.
[209] 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.
[210] 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.
[211] U.S. EPA. 2007. Staff Paper, p. 2-54.

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,[212] 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.[213]

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.

[212] White House Office of Management and Budget, Bulletin No. 07-01 Update of Statistical Area Definitions and Guidance on Their Uses. December 18, 2006.
[213] 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 15: 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 Table 16 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.[214] Without a doubt, 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,

[214] http://www.arb.ca.gov/research/aaqs/ozone-rs/rev-staff/rev-staff.htm

one often evaluates the risk to the most vulnerable."[215] 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 under five years old, 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."[216] Additionally, prenatal exposures have been documented to correlate with lower birth weight and intrauterine growth retardation.[217] 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,[218] 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.[219] 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.[220]

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

[216] 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.

[217] 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.

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

[219] 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, Lioy 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.

[220] 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,[221] mail carriers,[222] and others.[223] 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 16 indexes some categories of outdoor workers and provides estimates of population size. Note that this tabulation does not include members of the military forces.

[221] 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.

[222] 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.

[223] 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 16: 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/eeo2000/index.html.[224]

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."[225] The CASAC found that new studies have provided evidence for an increase in mortality associated with ozone exposure levels well below the current standard.[226] 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.

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

[225] U.S. EPA. Staff Paper, 2007. p. 6-14.

[226] 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.

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

According to the calculations in the Regulatory Impact Assessment presented in Table 17 below, both the 75 and 70 ppb standards would leave 1,924,650 – 2,595,855 people unprotected from the hazards of ozone exposure, compared to a standard of 65 ppb, the most stringent option analyzed by EPA. A standard of 65 ppb presents significant public health advances, reducing health impacts in a projected 4,105,530 people, although it will still leave vulnerable populations at elevated risk; therefore legally required option is the most public health protective one: 60 ppb (0.060 ppm).

	Estimated Decrease in Number of Individuals Affected, Alternative Standards			
HEALTH ENDPOINT	**65 ppb**	**70 ppb**	**75 ppb**	**80 ppb**
Mortality	530	280	200	19
Hospital Admissions (Ages 0-1)	3,100	1,800	1,400	120
Hospital Admissions (Ages 65-99)	4,300	2,300	1,800	160
Emergency Department Visits (Asthma)	2,600	1,500	1,200	94
School Absences	1,300,000	780,000	610,000	50,000
Minor Restricted Activity Days	3,500,000	2,100,000	1,600,000	130,000
Projected total decrease in affected individuals	**4,810,530**	**2,885,880**	**2,214,675**	**180,393**

Table 17: Projected Benefits (Decrease in Affected Individuals) under Alternative Standards. Source: Environmental Defense derived from RIA, Tables 6-5, 6-6, 6-11, 6-12, 6-17, 6-18, 6-23, and 6-24.

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

Unjustifiable Regulatory Option Analyzed

First, we note in the preamble that EPA intended to include three options for analysis in the draft RIA -- 0.075, 0.070, and 0.065 ppm. (72 FR 37907). However, it is apparent from correspondence in the docket that OMB pressured EPA to include an additional option of 0.079 ppm in the RIA.[227] A one ppb reduction in the standard is not a credible policy alternative, given that the CASAC has unanimously concluded that the current standard is not protective of public health. We can see no scientifically acceptable justification for considering such a blatantly weak option in this analysis.

The Benefits Assessment Underestimates the Value of Ozone Mortality Reductions

According to the Staff Paper, the association between short-term ozone exposure and premature mortality is "robust and credible."[228] We are extremely pleased that EPA included the value of ozone mortality benefits in the RIA, which is an advance over other recent rulemakings such as the locomotive rule.

Nevertheless, in the RIA, EPA has included analysis of the possibility that ozone mortality benefits could be zero. The zero effects case was not analyzed in the Risk Assessment, and it is not plausible to include it here. We find no evidence for inclusion of the "zero" estimate in the lengthy and thorough recommendations of CASAC. Nor do the Criteria Document, Staff Paper, or Risk Assessment suggest that the effects of premature deaths attributable to ozone could credibly be considered to be zero. Inclusion of the zero risk assumption is arbitrary and must be dropped.

For the estimation of benefits, EPA first compared the results based on Bell et al. 2004 with those using a synthesis of three meta-analyses (Bell et al. 2005, Ito et al. 2005, and Levy et al. 2005) whose estimates of mortality are significantly higher than the estimates based on Bell et al. 2004. The RIA then selected the Bell et al. 2004 study as a basis for the estimation of ozone reduction benefits, while also assessing mortality benefits of zero. [229]

In contrast, 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. The monetary benefits of $PM_{2.5}$ reduction therefore reflect this range.

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

[227] OMB List of Items for RIA: EPA Responses, Docket # EPA-HQ-OAR-2007-0225-0002; and Bureau of National Affairs, *Daily Environment Reporter*. EPA Analysis Shows Range of Estimates for Benefits from Tightening Ozone Standard. August 2, 2007.
[228] U.S. EPA., Staff Paper, p. 6-14.
[229] U.S. EPA, Regulatory Impact Analysis of the Proposed Revisions to the National Ambient Air Quality Standards. EPA-452/R-07-008, July 2007, p. 6-73.

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.[230]

Benefits Assessment Ignores Related Photochemical Compounds

To reduce ground level ozone, emissions of precursor chemicals, such as nitrogen oxides and volatile organic chemicals, must be reduced. This brings important co-benefits, as well. In addition to its role as an ozone precursor, NOx is an individual criteria pollutant. Furthermore, the reaction of NOx and VOCs in the presence of sunlight produces more than just ozone. The interaction of these chemicals also results in the formation of a range of photochemical products, such as peroxyacetyl nitrate (PAN), nitric acid (HNO_3), and hydrogen peroxide (H_2O_2), and other compounds, such as formaldehyde (HCHO), other aldehydes and ketones.[231] Like ozone, these chemicals also damage the lungs. EPA includes no assessments of the contribution of these additional oxidants to an assessment of human health risks. For example, formaldehyde is a carcinogen, and the inclusion of cancer risks from formaldehyde exposure would have increased the estimated benefits of a more stringent ozone standard. Taken together, the co-benefits of ozone reduction are likely to be underestimated.

$PM_{2.5}$ Reduction is an Important Co-Benefit

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 ($PM_{2.5}$).[232] $PM_{2.5}$ 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 NOx 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 18, the combined value – in terms of lives saved and the estimated monetary benefits – is staggering. EPA estimates that full attainment of an ozone standard of 65 ppb (with the exception of some regions in California) would result in as many as 6,400 lives saved and mortality benefits of up to $39 billion.

[230] RIA, p. 6-31
[231] CD p. 2-2.
[232] U.S. EPA. 2005. ACQD PM. p. 2-2.

We also note that although premature mortality accounts for more than 95 percent of the total monetized benefits (RIA p. 6-74), ozone-related morbidity touches the lives of millions of people.

Alternative O_3 Standards	NMMAPS Data O_3 Benefits (Bell et al. 2004)		Synthesis of Bell et al. 2005, Ito et al. 2005, and Levy et al. 2005 O_3 Benefits		Literature-Derived $PM_{2.5}$ Benefits		TOTAL BENEFITS $
	Lives Saved	O_3 Benefits (Morbidity + Mortality) $	Lives Saved	O_3 Benefits (Morbidity + Mortality) $	Lives Saved	$PM_{2.5}$ Benefits (Morbidity + Mortality) $	
65 ppb	530	3.7 B	2,100 - 2,400	14 - 16 B	1,800 – 4,000	10 - 23 B	13.7 – 39 B
70 ppb	280	2 B	1,100 - 1,400	7.4 - 9.1 B	1000 - 2,300	6 - 14 B	8 – 23.1 B
75 ppb	200	1.6 B	880 - 1,100	5.9 - 7.3 B	620 - 1,400	3.6 – 8.6 B	5.2 – 15.9 B
79 ppb	19	140 M	78 - 85	510 - 560 M	480 - 1,100	2.8 – 7 B	2.94 B – 7.56 B

B – Billion Dollars, M = Million Dollars Data Source: U.S. EPA. 2007. RIA.

Table 18: EPA Estimates of Ozone and $PM_{2.5}$ Co-Benefits Associated with Declines in O_3 Source: Environmental Defense, derived from RIA.

Additional Issues

EPA solicits public comment on some additional miscellaneous issues.

Data Reporting

The FR notice raises the question of whether rounding or truncation should be used if for monitoring data reported to the fourth decimal place. With truncation, all concentrations above the standard would be ignored. With rounding, half the concentrations would be rounded up when assessing compliance with the standards. In this instance, we favor rounding to three digits over truncation because it is more health-protective.

However, we agree with EPA that any extra significant digits reported with monitoring data should be preserved in the Air Quality System (AQS) database.

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, if EPA sets the ozone standard a 0.075 ppm as proposed, monitoring would not be required in areas with ozone concentrations of 0.064 or below.

Even if EPA does not set the standard at that level, such concentrations are clearly important to monitor. First, adverse health effects have been reported concentrations 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.

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.

Air Quality Index for Ozone

EPA proposes to automatically finalize revisions to the Air Quality Index (AQI), used for public reporting of ozone concentrations, to align the index with any final revisions made to the primary NAAQS. Commenters oppose this approach. While we recognize the value of making timely changes to the AQI, the Agency must carefully evaluate the need for more stringent AQI cutpoints, depending upon the level and form of the final standard, and provide a meaningful opportunity to comment on the specifics of the index proposed. Because EPA has failed to conduct such an evaluation, and because the agency has failed to provide notice of the specific index numbers that it proposes to correspond to specific ozone levels under a new NAAQS, EPA must first propose the specifics of how the revised index will work and provide notice and opportunity to comment thereon. CAA §307(d); Administrative Procedure Act. §553. Indeed, §319 of the Act -- the provision requiring EPA to establish the air quality index -- expressly mandated notice and opportunity for a public hearing before EPA promulgates air quality index rules.

Although EPA has not provided adequate notice of the specific ozone levels that will correspond to specific index values, commenters are concerned about the adequacy of the index based on the limited description provided. In the event that EPA establishes a NAAQS for ozone that does not conform to the lower end of the CASAC-recommended range, then having the AQI of 100 correspond to the primary NAAQS would be arbitrary and irrational. As currently defined, air quality with an AQI of 101-150 is characterized as "unhealthy for sensitive groups." But if EPA sets the 8-hour primary NAAQS at 0.070 or 0.075 ppm, and the 100 index level corresponds to that level, then -- based on the record now before EPA-- a 100 index will reflect ozone levels that are in fact unhealthy to healthy individuals. As discussed above, substantial evidence before the Agency shows adverse health effects to healthy individuals at ozone levels below 0.070 ppm. For these reasons, it would be arbitrary as well as grossly and irresponsibly misleading to the public to adopt an index indicating that such ozone levels are of concern only to "sensitive" persons.

There is well-established precedent for setting the AQI of 100 as corresponding to a pollutant level below the NAAQS, in the example if the AQI for $PM_{2.5}$, established after the 1997 revisions to the NAAQS. In that instance, EPA determined that $PM_{2.5}$ concentrations of 40.5 $\mu g/m^3$ should correspond to an AQI of 100, which triggers public notification of unhealthy concentrations. The equivalent 24-hour air quality standard was 65 $\mu g/m^3$. The need to evaluate and propose such alternative approaches for ozone highlights the need for additional notice and comment on this issue.

Conclusion: Current Standard Does Not, and Proposed Standards Will Not Protect Public Health

EPA has included both the current standard of 0.08 ppm, along with minor adjustments to this standard, 0.080, 0.079, 0.075 and 0.070 ppm as possible options for the revised ozone standard. None of these alternatives will provide the requisite protection of public health, as required by the Clean Air Act. The health evidence and the EPA Risk Assessment clearly demonstrate that the proposed standards will not fully protect public health. Adverse health effects have been documented to occur in healthy individuals at levels at and below 0.08 ppm, and some individuals exhibit adverse effects at ozone concentrations at 0.06 ppm. Vulnerable populations, including children with asthma, are expected to be more sensitive than healthy individuals. Given the magnitude and severity of the risks, delay of a decision on revisions to the standard is not a viable policy option.

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 evidence shows that considerable residual risk persists down to 64 ppb, the most stringent option analyzed. We therefore strongly object to consideration of current standards, or any of the following alternative standards -- 0.080, 0.079, 0.075 and 0.070 ppm—as a basis for updating the ozone NAAQS, and call upon EPA to adopt a more stringent standard of 0.060 ppm, using the third highest maximum, to minimize adverse health effects from ozone.