

Comments on EPA’s Proposed Revisions to National Ambient Air Quality Standards for Ozone

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Submitted on behalf of:

Alliance of Automobile Manufacturers
American Bakers Association
American Chemistry Council
American Coke & Coal Chemicals Institute
American Forest & Paper Association
American Iron and Steel Institute
American Petroleum Institute
American Trucking Associations
Chevron Corporation
Corn Refiners Association
Council of Industrial Boiler Owners
Edison Electric Institute

Engine Manufacturers Association
Exxon Mobil Oil Corporation
Independent Liquid Terminals Association
Institute of Shortening and Edible Oils
National Association of Manufacturers
National Cotton Council
National Mining Association
National Oilseed Processors Association
National Petrochemical & Refiners Ass’n
National Rural Electric Cooperative Ass’n
Portland Cement Association
U.S. Chamber of Commerce

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I. INTRODUCTION AND SUMMARY

A. Introduction

On July 11, 2007, the United States Environmental Protection Agency (EPA) proposed revisions (the Proposed Rule) to the National Ambient Air Quality Standards (NAAQS) for ozone (O₃), and established an October 9, 2007 deadline for submission of comments on the Proposed Rule (72 Fed. Reg. 37818 *et seq.*). These comments on the Proposed Rule are submitted jointly by the trade associations and companies listed on the cover (the Commenters), which collectively represent a large portion of the U.S. economy. Several of these associations and companies have submitted or are submitting separate comments on the Proposed Rule. However, they have all come together to voice their strong support for retaining the current NAAQS for ozone and their opposition to adopting any of the more stringent alternatives proposed by EPA. These comments focus principally on the primary (health-based) standard, but also discuss briefly the secondary (welfare-based) standard.

B. Executive Summary

Under the Clean Air Act (CAA or Act), primary NAAQS must be set at a level requisite to protect the public health with an adequate margin of safety. In 1997, EPA revised the primary NAAQS for ozone from a one-hour average standard of 0.12 parts per million (ppm) (with one allowable exceedance per year) to the current 8-hour standard of 0.08 ppm (based on the annual 4th highest daily maximum 8-hour average concentration over a three-year period). In the Proposed Rule, EPA proposes to reduce the level of the 8-hour standard to a level within the range of 0.070 to 0.075 ppm, and to specify that level to three decimal places. EPA also solicits comment on alternative levels down to 0.060 ppm and up to and including the current standard level of 0.08 ppm.

EPA relies on five principal lines of evidence to support its proposal to reduce the level of the standard. As shown in these comments, on each of those lines of evidence, EPA has consistently overinterpreted the data in an effort to show the need for a reduction in the standard, when in fact none of those lines of evidence supports such a reduction. As a result, the

Commenters believe, EPA's conclusions cannot be considered scientifically valid. The reasons are summarized as follows:

1. EPA asserts that the controlled human clinical studies provide "strong" evidence of "medically significant" effects in healthy people exposed to ozone levels at and above 0.080 ppm, with some indication of effects at lower levels. In fact, the human clinical studies on healthy subjects have not shown medically significant effects at levels at and below the current standard level of 0.08 ppm. Both the lung function changes and respiratory symptoms reported in these studies at such levels, both on an average basis and in individual subsets of subjects, consist of transient, reversible responses that are not of adverse health consequence.
2. EPA claims that the clinical evidence indicates that asthmatics are likely to experience larger and more serious effects than healthy people. However, EPA's claims are based on assumptions about asthmatics' response profile that have not been established and, in any event, do not address the key question of whether the asserted effects would occur at levels at and below the current standard. The actual clinical studies on asthmatics exposed to ozone (which are limited to exposures of around 0.12 ppm and above and most of which were available during EPA's 1997 review) show that, at around 0.12 ppm, asthmatics' responses to ozone exposure, in terms of both lung function decrements and respiratory symptoms, are small, transitory, and comparable to the types of responses that asthmatics commonly experience. As explained by numerous physicians who specialize in treating and studying patients with asthma, these types of responses are not deleterious to the asthmatics' health. Responses at lower ozone levels would be expected to be even less. Overall, EPA has not presented any significant new clinical information on asthmatics that would warrant a reduction in the standard level.
3. EPA asserts that epidemiological asthma panel studies show "robust" associations between 8-hour ozone concentrations and lung functions effects, respiratory symptoms, and medication usage in asthmatic children, even at ozone levels below the current standard. The studies themselves, however, do not support EPA's assertion, because:

- Most were conducted in areas with ozone levels higher than the current standard;
 - These studies involve considerable uncertainties as to whether the measured ozone concentrations adequately represent personal exposures;
 - At least some of these studies used pulmonary function measurements considered to be unreliable;
 - These studies do not show consistent associations between ozone and effects and cannot attribute the effects observed to ozone (as opposed to other pollutants or factors); and
 - These studies are inconsistent with other recent studies which have found no associations between ozone concentrations and asthma symptoms.
4. EPA argues that other time-series epidemiological studies show associations between short-term ozone concentrations and other health effects, including respiratory emergency room visits and hospital admissions and (in multi-city studies) premature non-accidental mortality, even at ozone levels below the current standard. These studies do not support EPA's claims for many of the same reasons noted above – i.e.:
- Most were conducted in areas or included a large number of cities that would not have met the current standard;
 - It cannot be assumed that the ambient ozone concentrations measured in these studies adequately reflect people's exposures;
 - EPA has failed to adequately consider model selection;
 - The studies have inconsistent results and cannot implicate ozone (as opposed to other pollutants or factors) as the cause of the reported effects; and
 - The studies on associations between mortality and short-term ozone exposure contain implausible findings and are inconsistent with the studies of long-term ozone

exposure, which EPA concedes have shown no consistent relationship between such exposure and mortality.

5. EPA relies on its exposure and risk assessments as indicating significant risks if the current standard were just met and thus supporting the need to lower the standard. However, EPA has failed to take into account the significant uncertainties in these assessments. These include:

- Uncertainties in the exposure estimates;
- Reliance on health effects endpoints which have not been shown to be adverse to health or to be causally associated with ozone exposures;
- Uncertainties regarding the concentration-response relationship and regarding a causal link between ozone exposures and effects, particularly at levels below 0.08 ppm;
- Underestimates of “policy-relevant background”; and
- Uncertainties about whether standards below the current level would be attainable, such that the asserted health benefits could be realized.

Given these factors, EPA’s exposure and risk estimates are too speculative to support lowering the primary standard level.

In addition to the lack of adequate support for lowering the level of the primary standard, EPA has not presented a sufficient justification for changing the specification of the standard from two to three decimal places at this time, given that: (a) EPA’s air monitoring reference methods specify a precision of two decimal places for ozone; (b) there is still uncertainty about the precision of the third decimal place, particularly in field measurements; and (c) specifying the standard to the third decimal place implies a degree of precision in the underlying health effects data that does not exist, due to the uncertainties in those data.

Secondary NAAQS must specify a level of air quality requisite to protect the public welfare from any known or anticipated adverse effects. The current secondary standard was set to be identical to the primary standard. In the Proposed Rule, EPA concludes that that standard is inadequate to protect the public welfare from known and anticipated adverse effects on vegetation; and it proposes to replace that standard with one of two options: either (a) a cumulative seasonal standard (with a complex form); or (b) a standard identical to the proposed revised primary standard. As summarized in these comments, EPA's proposal has failed to take account of the numerous uncertainties that remain about the degree to which and levels at which ozone produces adverse effects on the public welfare and about the extent to which the proposed revised secondary standard (under either option) would protect the public welfare from such effects. These uncertainties need to be addressed before adopting a new and more stringent standard that would produce substantial and widespread adverse impacts on businesses and communities, which are also part of the public welfare.

For these reasons, the scientific evidence does not demonstrate that a reduction in the standard is needed to protect the public health and welfare. Rather, the Commenters believe that the current standard meets the statutory requirements and should be retained as both the primary and the secondary standard.

II. BACKGROUND

A. Requirements for NAAQS

Under the CAA, EPA must review NAAQS at least every five years and revise them “as may be appropriate” in accordance with Sections 108 and 109(b) of the Act (CAA § 109(d)(1)). Primary NAAQS must be set at a level “requisite to protect the public health” with “an adequate margin of safety” (CAA § 109(b)(1)). Secondary NAAQS must specify a level of air quality “requisite to protect the public welfare from any known or anticipated adverse effects” (CAA § 109(b)(2)). As stated by the Supreme Court in *Whitman v. American Trucking Ass'ns*, 531 U.S. 457, 476 (2001), “requisite to protect” means “not lower or higher than is necessary.” The comments below demonstrate that the existing standards are “requisite to protect” the public

health and welfare, and that EPA's proposed revisions are not necessary to meet that objective and therefore are not justified.

B. Historical Context

In 1997, EPA revised the primary NAAQS for ozone from a one-hour average standard of 0.12 ppm (with one allowable exceedance per year) to the current 8-hour standard of 0.08 ppm, based on the annual 4th highest daily maximum 8-hour average concentration over a three-year period (62 Fed. Reg. 38856, July 18, 1997). In doing so, the Administrator concluded that “[t]he 8-hour averaging time is more directly associated with health effects of concern at lower O₃ concentrations than is the 1-hour averaging time,” and that “an 8-hour standard would limit both 1- and 8-hour exposures” (*id.* at 38861). With regard to the level of the standard, the Administrator first acknowledged that, as increasingly stringent standards were evaluated, including an 8-hour standard of 0.07 ppm, the estimated risks decreased for respiratory functional and symptomatic effects and for hospital admissions for respiratory causes (*id.* at 38864). Additionally, the Administrator acknowledged that there might be no ozone level “below which absolutely no effects are likely to occur” (*id.* at 38863). Notwithstanding these observations, the Administrator determined that a standard more stringent than 0.08 ppm was “not requisite to protect the public health with an adequate margin of safety” (*id.* at 38868). The Administrator therefore promulgated a primary 8-hour standard of 0.08 ppm.

With respect to the secondary standard, EPA recognized in 1997 that it had considerable evidence on the effects of ozone on vegetation. It also acknowledged that “the available scientific information supports the conclusion that a cumulative seasonal exposure index . . . is more biologically relevant than a single event or mean index” (*id.* at 38875). Nevertheless, the Administrator chose to set the secondary standard equal to the new 8-hour primary standard (*id.* at 38877). Specifically, the Administrator decided not to set a seasonal secondary standard due to the “substantial uncertainties” in the estimates of the increased welfare protection that a seasonal standard would provide (*id.* at 38877-78). The Administrator concluded that adoption of the 8-hour primary standard as the secondary standard would result in “significant improvements in public welfare” (*id.* at 38877).

The primary and secondary standards promulgated in 1997, which were identical, were not challenged in court as less stringent than necessary. Rather, they were challenged as overly stringent, but were ultimately upheld against those challenges. See *American Trucking Ass'ns v. EPA*, 283 F.3d 355, 378-80 (D.C. Cir. 2002), *upon remand from* 531 U.S. 457, 476 (2001).

C. EPA's Proposed Rule

In the Proposed Rule, EPA proposes a number of revisions to both the primary and secondary standards for ozone. For the primary standard, EPA proposes to retain: (a) use of O₃ as the most appropriate surrogate for ambient photochemical oxidants; (b) the 8-hour averaging time to provide protection against both short-term and prolonged exposures; and (c) the current form of the standard, which is the 4th highest daily maximum 8-hour average. However, EPA proposes to reduce the level of the 8-hour standard to a level within the range of 0.070 to 0.075 ppm, and to specify that level to the nearest thousandth of a ppm. At the same time, EPA solicits comment on alternative levels down to 0.060 ppm and up to and including the current level of 0.08 ppm (which is effectively 0.084 ppm due to rounding) (72 Fed. Reg. at 37818).

EPA summarizes in several places the basis for its proposal to lower the primary standard level (e.g., 72 Fed Reg. at 37870, 37878-79, 37880). As summarized by EPA, that proposal is based on the following principal lines of evidence:

1. Clinical evidence in healthy people exposed to ozone levels at and above 0.080 ppm of lung function decrements, respiratory symptoms, pulmonary inflammation, and other airway responses (deemed "medically significant" by EPA), with some indication of lung function decrements and respiratory symptoms at lower levels;
2. Clinical evidence that EPA believes shows that asthmatics are likely to experience larger and more serious effects than healthy people;
3. Epidemiological evidence of lung function decrements, respiratory symptoms, and increased medication usage in asthmatics associated with ozone exposures;

4. Epidemiological evidence of ozone associations for a variety of serious health effects, including respiratory emergency room and hospital admissions and premature mortality; and
5. EPA's estimates of exposures and risks associated with just meeting the current standard.

With respect to the secondary standard, EPA concludes that the current standard (which is identical to the primary standard) is “inadequate to protect the public welfare from known and anticipated adverse O₃-related effects on vegetation and the ecosystem,” notably, visible foliar injury, seedling and mature tree biomass loss, and crop reductions (72 Fed. Reg. at 37899). Further, EPA concludes that a complex cumulative seasonal form of the standard is more “biologically relevant” than the current 8-hour standard, although the current 8-hour form “can also provide substantially improved protection to vegetation when set at an appropriate level” (*id.* at 37899; see also *id.* at 37905). EPA thus proposes to replace the current standard with one of two options: either (a) a cumulative seasonal standard; or (b) a standard identical to the proposed revised primary standard (*id.* at 37818, 37899, 37905).

D. Implications of EPA's Proposed Rule

Under Section 109 of the CAA, EPA does not have authority to base NAAQS on the economic costs of compliance. However, as recognized by Justice Breyer in *Whitman v. American Trucking Ass'ns*, 531 U.S. at 494-95, Section 109 “does not require the EPA to eliminate every health risk, however slight, at any economic cost, however great”; and it gives the Administrator the flexibility to consider various contextual factors, including the severity of a pollutant's effects, the number of those affected, the distribution of the effects, the uncertainties in the estimates, and comparative health and welfare consequences, in establishing the “requisite” primary and secondary standards. These comments demonstrate that, based on the current scientific evidence, the proposed changes to the primary and secondary NAAQS for ozone are not necessary or justified to meet the statutory standard of being requisite to protect the public health and welfare.

Before doing so, however, we note briefly, for context, that a reduction in the level of the primary and secondary NAAQS would have substantial adverse impacts on the regulated community and on States and local communities. Currently, States are still in the process of executing implementation plans to meet the current standard (established in 1997, but delayed due to litigation). These implementation plans include new restrictions and controls that are expected to significantly reduce ground-level ozone emissions, as EPA recognizes (EPA, 2007b). Adopting a more stringent standard before the current standard has been fully implemented would put a substantial new burden on the States to adopt new implementation plans before the health and environmental benefits of the current standard can be evaluated.

Moreover, the reduction of the primary standard to the level proposed by EPA, 0.070 to 0.075 ppm, would put a large number of additional areas in the United States into non-attainment, while the adoption of a standard at the lower alternative level identified by EPA, 0.060 ppm, would likely result in the majority of the country being so designated when background ozone levels are taken into consideration (see discussion at end of Section IV below). Non-attainment designations have significant adverse consequences for areas so designated. For example, businesses may be unable to locate new operations or expand existing operations in non-attainment areas due to the increased costs, delays, and uncertainties associated with the restrictive permitting regimes required in such areas. Similarly, businesses and consumers within non-attainment areas may be subjected to increased costs as a result of special requirements for vehicles and fuels sold in those areas.

These substantial and widespread adverse impacts can and should be avoided because the scientific evidence does not demonstrate the need for the proposed revisions of the standard as being “requisite to protect” the public health and welfare, as discussed in these comments.

E. Scope of Comments

The Commenters support EPA’s proposal to retain the current indicator (O_3) and averaging time (8 hours) of the primary standard. These items are not discussed further in these comments.

The Commenters strongly oppose any reduction in the level of the primary standard and support the alternative of retaining the current standard level. In support of this position, Section III demonstrates that the clinical and epidemiological evidence upon which EPA relies does not justify its proposed reduction of the standard level. Section IV shows that EPA's exposure and risk assessments likewise do not justify such a reduction. Section V presents the Commenters' position that changing the current expression of the standard to add a third decimal place is not warranted at this time.

With respect to the secondary standard, Section VI briefly describes the Commenters' position that, due to the substantial remaining uncertainties regarding the occurrence of adverse effects on public welfare at levels below the current standard, as well as the extent to which a revised standard would provide protection from such adverse effects, the current scientific information is insufficient to justify a change in the secondary standard.

III. THE HUMAN CLINICAL AND EPIDEMIOLOGICAL STUDIES DO NOT JUSTIFY LOWERING THE PRIMARY STANDARD LEVEL.

As noted above, EPA asserts that the body of human clinical and epidemiological studies demonstrates that the existing primary standard of 0.08 ppm is not adequate to protect the public health. Specifically, EPA asserts that: (1) there is "strong" clinical evidence of "medically significant" effects in healthy people at exposure levels of 0.080 ppm and above, with "some indication" of effects at lower levels; (2) there is "substantial" clinical evidence that asthmatics "are likely to experience larger and more serious effects than healthy people"; (3) there is likewise "substantial" epidemiological evidence of such effects in asthmatics due to ozone (including at levels below the current standard); and (4) epidemiological studies also show significant associations of ozone exposure with "a wide range of serious health effects, including respiratory emergency room visits and hospital admissions, and premature mortality, at and below 0.080 ppm" (72 Fed. Reg. at 37879). In reaching these conclusions, as shown below, EPA has substantially overinterpreted the findings and implications of the existing human clinical and epidemiological studies. While in some places EPA recognizes certain uncertainties and limitations associated with the data, in the end it plays down those uncertainties and limitations, ignores others, and consistently interprets or characterizes the data beyond what the data can

actually support, in an effort to show that the current standard is not protective. As a result, the Commenters believe, EPA's conclusions are not scientifically valid (see *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993)).

We recognize that the Clean Air Scientific Advisory Committee (CASAC) likewise concluded that the existing primary standard is not protective, and recommended an even more drastic reduction in the standard level (to a range of 0.060 to 0.070 ppm), relying on the same basic lines of evidence on which EPA relies (CASAC, 2006b, 2007). However, as also discussed below, a careful review of the evidence indicates that CASAC similarly overinterpreted the data and reached conclusions that cannot be supported by an appropriate evaluation of the evidence.

A. Clinical Evidence on Healthy Subjects

EPA states that several studies (e.g., Folinsbee et al., 1988, 1994; Horstman et al., 1990; Adams, 2002, 2003, 2006) have reported statistically significant lung function responses and increased respiratory symptoms in healthy subjects from prolonged exposures (4-8 hours) to ozone levels in the range of 0.08 to 0.16 ppm (72 Fed. Reg. at 37828-29). With the exception of the Adams studies, these studies were available at the time of the last standard review in 1997 and thus provide no new information. EPA relies in particular on the recent studies by Adams (2002, 2006), since they are the only available human clinical studies that investigated such effects at levels below the current standard. These studies investigated effects of ozone on healthy subjects at levels of 0.08, 0.06, and 0.04 ppm, using both square-wave (steady-state) and triangular (increasing to a peak and then decreasing) exposures during a period of 6.6 hours involving considerable exercise. EPA observes that, in these studies, the author reported no statistically significant group effects on forced expiratory volume in one second (FEV₁) or total symptom score from exposure to any levels lower than the current standard. However, it notes that a reanalysis by EPA of the Adams (2006) data did show statistically significant group FEV₁ responses at the 0.06 ppm ozone exposure level, that the studies showed statistically significant lung function decrements in a small percentage of the individual subjects, and that the author's analysis also showed some statistically significant respiratory symptom responses toward the end of the exposure period (72 Fed. Reg. at 37828, 37864, 37875).

Regardless of the statistical significance of the reported responses, the effects shown in these clinical studies at levels of 0.08 ppm and below are not medically significant. Both the functional responses and symptoms reported in these studies were small, transient, and reversible. For example, the mean functional changes reported by Adams (2002, 2006) from pre- to post-exposure included FEV₁ decrements of around 4-6% at 0.08 ppm and 1-2% at 0.06 ppm (with no reduction at 0.04 ppm). EPA itself recognizes that such functional changes would not interfere with normal activities and would not constitute adverse health effects (72 Fed. Reg. at 37850). Similarly, the total mean symptoms scores reported by Adams (2002, 2006) were 8-10 at 0.08 ppm, and 2-4 at 0.06 and 0.04 ppm out of a possible score of 160. As noted in the comments of Dr. McFadden (2007) (copy attached as Exhibit A, excluding his curriculum vitae), these scores indicate that the symptoms were small and not medically significant.

EPA puts greater reliance on the reported effects in a few individual subjects. It notes that, based on data from the earlier studies (e.g., Folinsbee et al., 1988; Horstman et al., 1990), as interpreted by McDonnell et al. (1991), about 26%, 18%, and 8% of the subjects had FEV₁ decrements > 10%, 15%, and 20%, respectively, with exposures to 0.08 ppm ozone (72 Fed. Reg. at 37828; EPA, 2007, p. 5-22). EPA observes further that, in the Adams (2002, 2006) studies, with exposure to 0.08 ppm, 17% of the subjects experienced FEV₁ decrements > 10% when the results were not corrected for the effect of exercise in clean air and 23% experienced such effects after such correction, and that in the Adams (2006) study, with exposure to 0.06 ppm, 7% of the subjects experienced such effects after correction for the effect of exercise in clean air (72 Fed. Reg. at 37828). CASAC likewise relied on such findings, noting that “26% of the subjects had greater than 10% decrements [in FEV₁], which can be clinically significant” (CASAC, 2006b, p. 4).

However, these reported responses in a few individual subjects do not support a reduction in the standard level. To begin with, based on current data, it is unknown whether these individuals are representative of any identifiable segment of the population. Indeed, as stated by one CASAC member (Sverre Vedal), placing great reliance on the occurrence of effects in such a small number of subjects is “a dangerous precedent – especially in this case where we are looking at small effects in 3 of 30 vs. 1 of 30, a pitiful number on which to attempt to base

policy” (Vedal, 2007). Moreover, even the individual effects that have been reported at 0.08 ppm and below are transient responses that have not been shown to be adverse to health. Contrary to the opinion expressed in the CASAC (2006b) letter, the occurrence of FEV₁ decrements in the range identified by EPA and CASAC (~ 10-20%) in healthy subjects is not of medical concern. As discussed in the next section, numerous physicians have expressed the opinion that reversible FEV₁ decrements up to approximately 20% (or more in some cases) are not adverse health effects even in asthmatics. It follows that such changes in healthy individuals are of no consequence to their health. Additionally, the studies do not provide evidence of serious respiratory symptoms in any specific subset of the subjects.

EPA concludes that evidence of lung function decrements and respiratory symptoms in some healthy individuals at the 0.06 ppm exposure level is “too limited to support a primary focus at this level” (72 Fed. Reg. at 37878). In fact, an appropriate interpretation of the data is that the evidence on such responses in healthy individuals does not support *any* reduction in the level of the current standard.

B. Clinical Evidence on Asthmatic Subjects

EPA also claims that the evidence from human clinical studies indicates that individuals with asthma are likely to experience larger and more serious effects than healthy people (72 Fed. Reg. at 37826, 37864, 37878). In this regard, EPA presents a fairly lengthy discussion of the response profile of asthmatics in an effort to demonstrate why this is so (*id.* at 37846-47). CASAC likewise stated that asthmatics “have been found to be more sensitive and to experience larger decrements in lung function in response to ozone exposures than would healthy volunteers” (citing the Mortimer et al. (2002) epidemiological study discussed in the next section); and it speculated that if asthmatics had been included in the clinical studies of Adams, the percentage with lung function declines > 10% “would most likely be considerably greater” (CASAC, 2006b, pp. 4, 10).

These arguments do not take into account the actual clinical studies on asthmatics exposed to ozone, most of which were included in the 1997 Criteria Document and Staff Paper. Those studies included exposures to ozone at levels of around 0.12 ppm and higher for various

time periods (ranging from 1-2 hours to over 7 hours), generally combined with exercise (e.g., Linn et al., 1994; Weymer et al., 1994; Horstman et al., 1995; Kreit et al., 1989; McBride et al., 1994; Koenig et al., 1985, 1987, 1988). There do not appear to be any clinical studies of asthmatics exposed to ozone at 0.08 ppm and below. However, even at exposure levels of around 0.12 ppm, the functional changes and symptomatic effects observed in these studies are small and not of health significance, as discussed below.

The functional changes seen in these studies at ozone levels of around 0.12 ppm consisted of mean FEV₁ decrements well below 10% (and not statistically significant in most cases). EPA agrees that, even for asthmatics, such changes are not adverse (72 Fed. Reg. at 37850). As EPA notes (*id.* at 37846), Horstman et al. (1995) did report that, with exposure to 0.16 ppm ozone, the asthmatic subjects had a mean reduction in FEV₁ of 19%, compared to 10% in the healthy subjects. EPA asserts that FEV₁ decrements of 10-20% would constitute adverse health effects in asthmatics (72 Fed. Reg. at 37850); and CASAC (2006b, p. 12) asserted that “in asthmatic children, a 10% change [in FEV₁] is indicative of adverse effects.”

The findings of Horstman et al. (1995) at 0.16 ppm were available to EPA in its last review in 1997 and do not support lowering the current standard level below 0.08 ppm. Moreover, EPA’s (and CASAC’s) conclusion that FEV₁ reductions of 10-20% in asthmatics are adverse health effects is not well supported. Numerous physicians who specialize in studying and treating asthma patients have made clear that reductions in FEV₁ of up to 20% are not deleterious to the health of asthmatics. For example, as explained in the comments of Dr. McFadden (2007) (Exhibit A hereto), such transient decreases in FEV₁ of 10-20% commonly occur in asthmatics (especially overnight), are comparable to the kinds of bronchoconstrictive responses routinely induced in pulmonary function tests to diagnose asthma, and are not significant or meaningful to asthmatics. Many statements by other asthma physicians making similar points regarding FEV₁ changes of this magnitude were initially submitted to EPA in 1995 by the National Mining Association in connection with comments on the NAAQS for sulfur oxides (NMA, 1995, Appendix A), and copies were re-submitted as exhibits to the September 18, 2006 comments of a group of trade associations that included most of the present Commenters on EPA’s second draft of the Staff Paper on the NAAQS for ozone (Alliance of

Automobile Manufacturers et al., 2006). Excerpts from those comments are provided in Exhibit B to the present comments.

Similarly, the types of symptoms reported in the clinical studies of asthmatics exposed to ozone levels at around 0.12 ppm, while possibly greater than those observed in non-asthmatics, were small and do not indicate a health risk. For example, as discussed by McFadden (2007), the total symptoms scores reported by Linn et al. (1994) (at 0.12 ppm ozone) and by Weymer et al. (1994) (at 0.10 and 0.25 ppm ozone) are not indicative of significant symptoms.

Thus, the clinical studies on asthmatics exposed to ozone levels around 0.12 ppm, most of which were available at the time of the last review, do not indicate that such exposures produce effects of health concern to asthmatics. One would expect that the responses of asthmatics at levels of 0.08 ppm and below would be even less than those reported in these studies. Such responses are transitory and comparable to the types of responses that asthmatics commonly experience, and tolerate well, in response to many non-specific stimuli, such as exercise, cold air, stress, vigorous laughter, and exposure to common irritants.

EPA's statements in its preamble that are based on mechanistic reasons relating to asthmatics' response profile are speculative and do not take into account the actual studies on asthmatics' exposures to ozone. In his comments, Dr. McFadden (2007) explains that those statements represent assumptions about the pathogenesis of asthma, but are not supported by data demonstrating that the types of inflammatory responses discussed lead to clinical manifestations in terms of exacerbations of asthma or other adverse health effects. Further, he points out that, even if they did, the data do not address the question of the level at which ozone may have such effects and, in particular, whether ozone would have such effects at levels at and below the current standard. In fact, the clinical studies discussed above do not indicate that ozone exposures at such levels would have adverse health effects. Thus, EPA's statements do not provide a sound quantitative basis for reducing the standard level.

C. Epidemiological Asthma Panel Studies

EPA asserts that its conclusions about the greater effects of ozone exposure on asthmatics are further supported by the epidemiological studies showing that 8-hour average ambient ozone

concentrations are associated, at certain lag times, with measures of lung function, daily symptoms, and increased medication usage in asthmatic children (72 Fed. Reg. at 37828-29, 37847, 37865, 37878). These studies consist of asthma panel studies – notably, the large U.S. multi-city panel studies by Mortimer et al. (2002) (the National Cooperative Inner-City Asthma Study [NCICAS]) and by Gent et al. (2003), as well as a number of smaller, single-city studies. EPA recognizes that these studies do not provide “direct evidence of a causal link between exposure to ozone and the occurrence of the effects” (72 Fed. Reg. at 37879). However, it claims that the studies show “robust” associations between ozone concentrations and lung function effects, symptoms, and medication usage in asthmatic children (*id.* at 37829, 37865) and provide “strong evidence” of such effects (*id.* at 37847), thus supporting its proposal to lower the standard. (As noted above, CASAC (2006b) also relied on these studies, notably that of Mortimer et al. (2002).) In fact, a review of these studies reveals that they do not show such “robust” associations or “strong” evidence and thus do not support a lowering of the standard. The reasons are discussed below.

First, as EPA recognizes (72 Fed. Reg. at 37865), most of these studies were conducted in areas that would not have met the current ozone standard. Consequently, the results of such studies cannot be reliably used to support a reduction in the level of the current standard. EPA notes that Mortimer et al. (2002) reported statistically significant associations between 8-hour average ambient ozone concentrations and lung function decrements even on days when ozone levels above the current standard were excluded (72 Fed. Reg. at 37865, 37876). That study, however, is subject to the other serious limitations discussed below.

Second, there are serious questions about whether the ambient ozone measurements used in the studies adequately characterize individuals’ ozone exposure. On this subject, EPA provides a discussion that attempts to discount this problem and, in fact, argues that because ambient concentrations generally overestimate true personal exposures, the effects occur at lower levels than indicated by the ambient measurements (*id.* at 37838-39). EPA fails to recognize, however, that the assumption that ambient measurements can be used to represent personal exposures has not been sufficiently validated or demonstrated, and that the uncertainty regarding this assumption creates uncertainty about whether the associations reported in the studies in fact

show any effect of ozone at all, particularly since these studies involved other pollutants as well. EPA does not take into account a number of studies that have found that ambient concentrations of gaseous pollutants (such as ozone) do not correlate well with personal exposures to those pollutants (e.g., Koutrakis et al., 2005; Sarnat et al., 2006; Linaker et al., 2000; Liu et al., 1997). CASAC recognized these problems in the context of estimating ozone exposure in time-series studies (CASAC, 2006a, p. 3; 2006b, p. 10), as discussed in Section III.D. The same problems create uncertainty in the asthma panel studies.

Third, the pulmonary function measure used in many of the panel studies that EPA relies on, including the study by Mortimer et al. (2002), used self-reported peak expiratory flow rate (PEFR). Self-reported PEFR measurements are known to demonstrate high variability and are generally considered unreliable. The problems include low compliance rates, low reproducibility of the results, and missing and self-invented data (Kamps et al., 2001). These concerns were noted by EPA in its draft Integrated Science Assessment (ISA) for nitrogen oxides. In that document, EPA stated that “[r]eliable data are notoriously difficult to come by using portable peak flow measuring devices” (EPA, 2007c, p. 3-16). For this reason, EPA dismissed the results from all nine studies that failed to demonstrate an association between ambient NO₂ and reduced PEFR. Among those nine studies is the study by Mortimer et al. (2002). In any case, the very small changes in PEFR reported by Mortimer et al. (2002) (<2%) are not of clinical significance.

Fourth, the asthma panel studies, which were multi-pollutant studies, do not show consistent effects of ozone and cannot reliably attribute the effects observed to ozone, as opposed to other pollutants or other potential causal factors. For example, Mortimer et al. (2002) attribute the associations with decreased lung function, increased symptoms, and increased medication usage to general “summer-time air pollution,” rather than ozone in particular. Indeed, in that study, when all the pollutants studied – including sulfur dioxide (SO₂), nitrogen dioxide (NO₂), and particulates less than 10 micrometers in diameter (PM₁₀), as well as ozone – were considered, there was no significant association of effects with ozone (see, e.g., Exponent, 2006; Annapolis Center for Science-Based Public Policy, 2007). Moreover, this study found associations only in the morning; no associations were found with evening pulmonary function

or symptoms. As stated by Moolgavkar (2007, p. 20), if the association with ozone were causal, “one would [expect] to detect this association in the evening in view of the fact that children are outdoors during the day and exposed to ozone.”

The Gent et al. (2003) study has similar limitations, as explained by Moolgavkar (2007). That study considered only ozone and fine particles; it did not consider NO₂, which has been found to be associated with pulmonary function decrements and respiratory symptoms in other studies, and it did not control for relative humidity. Further, that study was conducted using a non-standardized questionnaire to record symptoms. Also, the study did not show a dose-response relationship between ozone levels and bronchodilator usage, and the associations reported for bronchodilator usage are inconsistent with those reported for more subjective symptoms.

Finally, the asthma panel studies upon which EPA relies are inconsistent with other recent studies, not mentioned by EPA, which have found no consistent associations between ozone concentrations and asthma symptoms. For example, Schildcrout et al. (2006) investigated associations between exposure to various air pollutants and asthma exacerbations (daily symptoms and use of inhalers) among asthmatic children in eight U.S. cities and found no associations of ozone with these effects. Additionally, while EPA mentions an earlier study by McConnell et al. (2002) on the onset of asthma (72 Fed. Reg. at 27834), a later study of associations of air pollution and asthma symptoms by McConnell et al. (2003) in southern California showed no significant associations of ozone with such symptoms in multi-pollutant models when NO₂ and organic carbon were also considered (see Moolgavkar, 2007).

In short, contrary to EPA’s statements in its preamble, the asthma panel studies show inconsistent and uncertain, not “robust,” associations between ozone concentrations and effects in asthmatics and certainly do not provide “strong” evidence of such effects due to ozone. In response to EPA’s request for comments on the degree to which these associations “reflect causal relationships between important health endpoints and exposure to O₃ alone at ambient O₃ levels below the current standard” (72 Fed. Reg. at 37878), it is apparent that the asthma panel

studies do not present reliable evidence of such causal relationships and thus do not support a reduction in the standard level.

D. Epidemiological Studies on Hospital/Emergency Room Admissions and Mortality

EPA also relies on other time-series epidemiological studies as evidence of an association between short-term ozone exposure and a wide variety of serious health effects, including respiratory emergency room visits and hospital admissions (72 Fed. Reg. at 37832, 37865-66, and 37876) and premature non-accidental mortality (*id.* at 37835-36, 37844, 37866, and 37876). CASAC (2006b, p. 12) also supported the reliance on these studies. While EPA claims that some of these studies support causal associations between health effects and short-term ozone exposure even at concentrations below the level of the current primary standard, these studies do not support a lowering of the standard, for the reasons discussed below.

First, as EPA recognizes, many of the hospitalization studies and nearly all the emergency room visit studies were conducted in areas that likely would not have met the current standard (EPA, 2007a, pp. 6-12, 6-13; 72 Fed. Reg. at 37865, 37866). Similarly, the multi-city mortality studies generally included a large number of cities that were not in attainment with the current ozone standard and nearly all single-city studies that showed statistically significant associations with mortality had 98th percentile ozone concentrations indicative of ozone levels that likely would not have met the current standard (EPA, 2007a, p. 6-16). In these circumstances, the results of these studies cannot be reliably used to support a reduction in the level of the current standard. While EPA notes that an analysis by Bell et al. (2006) of a subset of the data from the multi-city National Mortality, Morbidity and Air Pollution Study (NMMAPS) continued to show mortality associations even when only days with a maximum 8-hour average ozone concentration below 0.061 ppm were included (72 Fed. Reg. at 37876), that study is flawed in many significant respects, as described below, and cannot be relied on to support a reduction in the primary standard level.

Second, the epidemiological studies on hospital/emergency room admissions and mortality erroneously assume that measurements from one or more fixed monitors accurately represent individuals' ozone exposure. As noted above, EPA contends that, in the absence of

available data on personal ozone exposure, ambient measurements represent a good surrogate for personal exposures and may actually overestimate personal exposures (72 Fed. Reg. at 37838-39). In fact, however, there is considerable uncertainty about the validity of using ambient measurements to reflect personal exposures. For example, Sarnat et al. (2006) found that the ability of ambient gas monitors to represent personal exposure to such gases is quite limited, and they concluded that their results “suggest that time-series health studies based on 24-hour ambient concentrations may not be able to identify the effects of gases on health, and better exposure surrogates are needed.” Similarly, Koutrakis et al. (2005) demonstrated, based on studies in Boston and Baltimore, that ambient concentrations of gaseous pollutants such as ozone do not correlate well with personal exposure measures of those pollutants. Similar findings were reported by Liu et al. (1997) (for ozone), Linaker et al. (2000) (for NO₂), and Brauer et al. (2002) (for PM_{2.5}). In the context of estimating ozone exposure in time-series studies, CASAC (2006a) stated (at p. 3):

“It is known that personal exposure to ozone is not reflected adequately, and sometimes not at all, by ozone concentrations measured at central outdoor monitoring sites. Typically, personal exposures are much lower than the ambient concentrations, and can be dramatically lower depending on time-activity patterns, housing characteristics, and season. In addition, and of particular importance for the ozone time-series studies, there can be no correlation between personal concentrations of ozone measured over time and concentrations measured at central outdoor sites.”

In its later letter, CASAC (2006b) reiterated this problem, noting (at p. 10) that “Panel members have little insight as to what we would find if we had actual exposure measurements.” Thus, despite the fact that measured ambient concentrations may generally be higher than personal exposures, the relationship between the two is so variable and uncertain that ambient measurements cannot serve as a reliable surrogate for personal exposures in attempting to attribute any effects seen in the time-series studies to people’s ozone exposure, particularly when other pollutants are involved.

Third, EPA has failed to adequately consider and critically evaluate model selection in the epidemiological studies on which it relies. As Moolgavkar (2007, p. 11) points out: “It has become abundantly clear that choice of model, particularly adjustment for weather and temporal trends and choice of lag structure, can significantly influence the conclusions drawn from time-

series studies of air pollution.” Moreover, where the biologically correct models are unknown, as in the case of air pollution epidemiology, residual bias may occur (Moolgavkar, 2007, pp. 10-11). Similarly, Heuss (2007, pp. 47-49) notes that failure to account for model uncertainty in the time-series analyses leads to an overestimation of the certainty of the results of such analyses. In support of this view, Heuss (2007) cites a study by Koop and Tole (2004), who used Bayesian model averaging to evaluate model uncertainty in time-series analyses using an extensive set of pollutants and meteorological variables from Toronto, Canada, and concluded:

“Point estimates of the effect of numerous air pollutants all tend to be positive, albeit small. However, when model uncertainty is accounted for in the analysis, measures of uncertainty associated with these point estimates became very large. Indeed they became so large that the hypothesis that air pollution has no effect on mortality is not implausible. On the basis of these results, we recommend against the use of point estimates from time series data to set regulatory standards for air pollution exposure.”

EPA has not heeded the advice of Koop and Tole, instead relying on time-series studies without adequately considering the importance of model selection.

Fourth, the time-series studies reporting associations between ozone concentrations and hospital admissions or emergency room visits have inconsistent results and cannot clearly distinguish the effects of ozone from other potentially causal factors, including both other pollutants and non-pollutant variables. EPA’s Staff Paper recognized that “inherent limitations in time-series epidemiological studies raise questions about the utility of such evidence to inform judgments about a NAAQS for an individual pollutant such as O₃ within a mix of highly correlated pollutants, such as the mix of photochemical oxidants, especially at ambient O₃ concentrations below levels at which O₃-related effects have been observed in controlled human exposure studies” (EPA, 2007a, p. 6-6). In the Proposed Rule, however, EPA has not taken the cautious approach indicated by these limitations, but instead has overstated the likelihood of a causal relationship between exposures to ozone, especially at lower concentrations, and hospital admissions or emergency room visits for respiratory disorders. As discussed by the Annapolis Center for Science-Based Public Policy (2007, p. 4) and Heuss (2007, p. 51), Anderson et al. (1998) evaluated numerous studies of air pollution and daily asthma emergency room visits or admissions, and concluded that while there is evidence that the mix of air pollutants, including

SO₂, NO₂, fine particles, and ozone, may have an effect on such visits or admissions, there is no consistency in determining the specific pollutant responsible. The same remains true today, particularly at concentrations below the current ozone standard.

Fifth, the multi-city mortality studies – namely, the NMMAPS study by Bell et al. (2004, 2006), the European study by Gryparis et al. (2004), and the meta-analyses by Ito et al. (2005), Levy et al. (2005), and Bell et al. (2005) – do not provide reliable or consistent evidence implicating ozone exposures as a cause of mortality. The reasons for this include the following:

- These studies combined the results from many cities to show an effect when, in fact, the results from some cities did not show a significant positive association between ozone and mortality or showed a negative relationship. For example, the NMMAPS study by Bell et al. (2004) combined estimates of associations between ozone and mortality taken in 95 urban areas to arrive at a national average relative risk for mortality, notwithstanding the fact that the results from the individual cities ranged from strongly negative to strongly positive (see Annapolis Center for Science-Based Public Policy, 2007, p. 42). As shown by Moolgavkar (2007, p. 14) and McClellan (2007, p. 11), the data from this study show that only 7 of the 95 cities had statistically significant positive associations between ozone and mortality, and about a third of the cities had negative coefficients.
- The multi-city mortality studies and meta-analyses did not sufficiently account for other pollutants or other potential causal factors. For instance, while Bell et al. (2004) provided an adjustment for the potential confounding by PM₁₀, they did not have the necessary air quality data to provide similar and adequate adjustments for the potential confounding effects of fine particles (PM_{2.5}), which EPA has concluded also cause mortality (see Annapolis Center for Science-Based Public Policy, 2007, p. 42).
- The NMMAPS studies contain several findings that are inconsistent or implausible. For example, as explained by Moolgavkar (2007, p. 15), the original NMMAPS study showed a negative association between ozone and mortality in winter. By contrast, while the extended NMMAPS study by Bell et al. (2004) did not report a separate analysis for

winter, it found a larger effect estimate for the full-year analyses than for analyses during the summer months. This is at odds with most time-series studies of ozone that looked at seasonal effects and implies that there must have been a greater effect in winter than in summer, contrary to the original finding. Moreover, the Bell et al. (2004) study reported premature mortality at such low levels as to imply that ozone-related mortality is occurring at levels well below natural background, which is not biologically plausible (see Hayes, 2006, p. 5; Annapolis Center for Science-Based Public Policy, 2007, p. 42).

- The meta-analyses of ozone and mortality commissioned by EPA (Ito et al., 2005; Levy et al., 2005; Bell et al., 2005) cannot be considered to be independent from each other, since there is considerable overlap in the cities used in these three meta-analyses. In any case, these meta-analyses confirm the existence of publication bias in reporting such results and, despite the overlap, show a high degree of unexplained heterogeneity in the reported results (see Moolgavkar, 2007, p. 15).
- The findings of acute cardiopulmonary mortality are not coherent with the studies of hospital admissions, which report associations for respiratory but not cardiac-related admissions.
- Finally, EPA's conclusion that the mortality studies on which it relies suggest that there is an elevated risk of total non-accidental mortality associated with acute exposure to ozone is inconsistent with studies of long-term ozone exposure, which EPA admits have found no consistent association between long-term ozone exposure and mortality (72 Fed. Reg. at 37837). The absence of an association between long-term exposure to ozone and mortality undercuts the conclusion that short-term exposures cause premature mortality.

In sum, contrary to EPA's assertions, the time-series epidemiological studies cited in the Proposed Rule do not provide convincing evidence of causal associations between exposure to ambient ozone and either morbidity or mortality. Therefore, these studies do not support a reduction in the primary standard level.

IV. EPA'S EXPOSURE AND RISK ESTIMATES DO NOT JUSTIFY LOWERING THE PRIMARY STANDARD LEVEL.

In addition to the human clinical and epidemiological studies described in the preceding section, EPA relies on its exposure and risk assessments as support for its proposal to lower the existing primary standard of 0.08 ppm (72 Fed. Reg. at 37851-62, 37866-68, 37870-71). EPA acknowledges, however, that there are significant uncertainties and limitations associated with these assessments (e.g., *id.* at 37852, 37856, 37858, 37859, 37876, 37880). Given these uncertainties and limitations, as described in greater detail below, the exposure and risk estimates do not support lowering the primary standard level.

First, there are substantial uncertainties in the exposure estimates. EPA simulated ozone population exposure using the Air Pollutants Exposure Model (APEX), the human inhalation exposure model within the Total Risk Integrated Methodology framework (*id.* at 37852). While EPA expresses confidence in the model structure (i.e., the algorithms in APEX designed to simulate the processes that result in people's exposures to ozone), EPA acknowledges "important uncertainties" with respect to the model inputs, including those related to: (a) the modeling of human activity patterns over an ozone season; (b) the modeling of variations in ambient concentrations near roadways; (c) the modeling of air exchange rates that affect the amount of ozone that penetrates indoors; and (d) the characterization of energy expenditure for children engaged in various activities (*id.*). Uncertainty with respect to energy expenditure is particularly important, as it "carries over to the uncertainty of the modeled breathing rates, which are important since they are used to classify exposures occurring at moderate or greater exertion which are the relevant exposures since O₃-related effects observed in clinical studies only are observed when individuals are engaged in some form of exercise" (*id.*).

In addition to the uncertainties associated with the exposure estimate model inputs, there was substantial variability observed across the 12 urban areas for which exposure estimates were conducted with respect to the percent of the population subgroups estimated to experience ozone exposures of concern. As an example, EPA notes that when 2002 ozone concentrations were simulated to just meet the current primary ozone standard, the aggregate 12 urban area estimate was that 18% of all school age children were estimated to experience ozone exposures (at levels

≥ 0.070 ppm), while the exposure estimates in the 12 urban areas considered separately ranged from 1 to 38% (*id.* at 37855). This variability raises significant concerns about the extent to which the 12 urban areas used in the exposure estimate and, consequently, the risk assessment, are representative of other cities.

Second, there are substantial uncertainties about the concentration-response relationship, particularly at levels below 0.08 ppm. Thus, the extrapolations assumed in the risk assessment may not be valid. EPA acknowledges as much: “key uncertainties include ... uncertainties associated with the shape of the exposure-response relationship, especially at levels below 0.08 ppm, 8-hour average, where only very limited data are available down to 0.04 ppm and there is an absence of data below 0.04 ppm” (*id.* at 37856). In addition to uncertainties relating to the shape of the concentration-response relationship, EPA identified the following uncertainties pertaining to the concentration-response relationship for that portion of the risk assessment based on effects reported in epidemiological studies: (a) those surrounding estimates of the O₃ coefficients for concentration-response relationships used in the risk assessment; (b) those “related to the extent to which concentration-response relationships derived from studies in a given location and time when O₃ levels were higher or behavior and/or housing conditions were different provide accurate representations of the relationships for the same locations with lower air quality distributions and/or different behavior and/or housing conditions”; and (c) those “concerning the possible role of co-pollutants, which also may have varied between the time of the studies from which the concentration-response relationships were derived and the current assessment period” (*id.* at 37856).

Third, the risk estimates based on lung function decrements use a benchmark of FEV₁ declines of $\geq 15\%$ for healthy individuals and $\geq 10\%$ for asthmatics. As shown above, these types of changes are transient, routinely experienced and well-tolerated, and not of adverse health consequence for either healthy or asthmatic individuals.

Fourth, the risk estimates based on respiratory symptoms, hospital admissions, and non-accidental mortality are based on epidemiological studies which, as shown above, do not reliably

implicate ozone, and certainly not ozone at any specific level, as the causal agent for the effects reported.

Fifth, EPA itself notes that while the risk assessment assumes a causal link between ozone exposures and the reported effects, the evidence for such a causal relationship is uncertain at lower levels:

“The Administrator particularly notes that as lower standard levels are modeled, including a standard set at a level below 0.070 ppm, the risk assessment continues to assume a causal link between O₃ exposures and the occurrence of the health effects examined, such that the assessment continues to indicate reductions in O₃-related risk upon meeting a lower standard level. . . . [H]owever, the Administrator recognizes that *evidence of a causal relationship between adverse health effects and O₃ exposures becomes increasingly uncertain at lower levels of exposure.*” (*id.* at 37880, emphasis added).

To the extent that this assumption regarding a causal link at lower ozone levels is invalid, estimated reductions in O₃-related risk are artificially inflated.

Sixth, the estimated risks are for ozone levels in excess of “policy-relevant background” (PRB) (*id.* at 37857). PRB levels were predicted solely by modeling using the GEOS-CHEM model (*id.*). This model underestimates PRB. As shown in detail by Lefohn (2006, pp. 2-1 to 2-48), the modeled estimates of PRB are inconsistent with the empirical ozone monitoring data from Trinidad Head, California, which have been shown by detailed back-trajectory analyses to be more representative of PRB in the West. Those empirical data show the occurrence of numerous hourly average concentrations ≥ 0.05 ppm and indicate that PRB levels are in fact higher than predicted by EPA’s model. By underestimating PRB, EPA’s risk assessment *overestimates* sickness and premature deaths (see, e.g., CRA International, 2006, p. 7-9). EPA’s own sensitivity analysis confirms this overestimation of health effects: “estimates assuming higher PRB levels results [*sic*] in decreased estimates of non-accidental mortality incidence per 100,000 that are 50% or greater less than the base case estimates” (EPA, 2007a, p. 5-82).

In addition, EPA’s PRB estimates inappropriately excluded certain emissions contributions that should be included – notably, anthropogenic precursor emissions from Canada and Mexico (72 Fed. Reg. at 37857). This approach artificially inflates the risk estimates and is

unjustified because, notwithstanding treaties that the U.S. may have with Canada and Mexico, emissions from those countries cannot be directly controlled by U.S. emissions regulations; and, in any event, it is unreasonable to assume zero anthropogenic emissions in those countries. Therefore, anthropogenic precursor emissions from Canada and Mexico should be considered a part of PRB.

Seventh, in modeling the impact of alternative standards, EPA used a Quadratic Rollback approach to adjust hourly ozone concentrations (*id.* at 37853). This approach may overstate the air quality improvements resulting from emissions reductions and thus overstate the benefits of stricter standards. EPA has acknowledged that mid-level ozone concentrations do not necessarily decline at the same rate as peak ozone concentrations, a factor taken into account by the Quadratic Rollback method. However, as explained and demonstrated with actual data by Lefohn (2006, pp. E-9 - E-10 & 4-1 - 4-11), the Quadratic Rollback approach, which applies uniformly over the ozone season, will not capture observed month-to-month variations in the rate of ozone reduction, which are often significant. Therefore, the Quadratic Rollback approach will overstate improvements in mid-range air quality in many cases.

Finally, in considering the health benefits of lowering the standard level, it is important to consider whether such a standard would be attainable. EPA's focus on the mean background, and failure to consider the extremes of background, resulted in a failure on the part of the Agency to adequately evaluate whether the lower primary standard proposed by EPA is attainable, even with complete elimination of controllable man-made emissions. Due to the extremes of background, a standard at the lower level proposed by EPA, 0.070 ppm, would likely be unattainable in many areas of the country, including areas that are far removed from any significant man-made sources; and a standard at the lower alternative level identified by EPA and recommended by CASAC, 0.060 ppm, would likely be unattainable in most areas of the country because the background levels would leave too little room for any man-made emissions (see Annapolis Center for Science-Based Public Policy, 2007 pp. 25-30; Heuss, 2007, pp. 3-6). When EPA established the current primary standard for ozone, the Administrator specifically rejected a standard of 0.070 ppm as being too close to background levels (62 Fed. Reg. at 38868). The same remains true today. Adoption of such a standard, the attainment of

which is dubious for much of the country in light of extremes of background, would make the development of rational state implementation plans difficult, if not impossible. And if the lowered standard is not achievable, that calls into question the validity of the asserted health benefits of adopting such a standard.

Overall, EPA's exposure and risk estimates are based on a number of hypothetical assumptions that are highly uncertain. As a result, they are too speculative to support lowering the primary standard level.

V. CHANGING THE CURRENT ROUNDING PROCEDURE FOR THE STANDARD IS NOT WARRANTED AT THIS TIME.

The ozone standard currently specifies the level to the nearest hundredth of a ppm (two decimal places). EPA is proposing to change the standard to specify the level to the nearest thousandth of a ppm (three decimal places) – i.e., 1 part per billion (ppb). This proposal is based on EPA's conclusion, in which CASAC (2006b, p. 5) concurred, that "current monitoring technology allows accurate measurement of O₃ to support specifying the 8-hour standard to this degree of precision" (72 Fed. Reg. at 37875). However, the technical capability of current monitoring technology does not answer the question of the appropriate rounding procedure to use in evaluating attainment of the standard.

First, while current technology may allow precision in the ppb range, there remains uncertainty about the third decimal place. For example, although (as EPA notes) the California Air Resources Board has adopted an 8-hour ozone standard specified to the nearest thousandth of a ppm (0.070 ppm), it has recognized that "ozone measurements are uncertain in the third decimal place, to the degree of plus or minus 0.0003 ppm" (California Air Resources Board, 2005, p. 6-3). Similarly, Langstaff (2007) has reported bias estimates for individual monitors in Boston varying from -7% to +11% in 2003-04. Further, as noted by Langstaff (2007), interferences from other pollutants and humidity effects can lead to errors in field measurements. Indeed, EPA's ambient air monitoring reference methods specify a precision of two decimal places (0.01 ppm) for ozone concentrations (40 C.F.R. § 53.20, Table B-1).

Moreover, specifying the standard to the third decimal place implies a degree of precision in the underlying health effects data that does not exist. EPA recognized this when it adopted the current standard in 1997. It stated, at that time, that it was specifying the standard to two decimal places “in part to reflect uncertainties in the health effects evidence upon which the proposed standard is based,” including “the measurement uncertainty and representativeness inherent in the reported O₃ concentrations used in field and epidemiological studies and the uncertainty in the exposure estimates upon which the quantitative risk estimates have been based” (62 Fed. Reg. at 38886). As discussed in prior sections of these comments, those uncertainties remain. EPA has not explained why those uncertainties should not lead to the same conclusion reached in 1997.

Given that (a) EPA’s current monitoring requirements specify precision to only two decimal places, (b) there is still uncertainty about the precision of the third decimal place, particularly in field measurements, and (c) there are considerable measurement uncertainties in the underlying health effects studies, as well as uncertainties in the exposure and risk estimates, the Commenters believe that it would not be appropriate, at the present time, to make attainment determinations in the real world based on the implied precision of a standard expressed to three decimal places. Hence, the Commenters support the retention of the current expression of the standard as 0.08 ppm.

VI. THERE REMAIN TOO MANY UNCERTAINTIES TO JUSTIFY ADOPTING A SECONDARY STANDARD THAT IS DIFFERENT FROM THE PRIMARY STANDARD.

In reviewing the data on the effects of ozone on vegetation, EPA concludes that the current secondary standard, which is identical to the primary standard, is inadequate to protect the public welfare from adverse effects. EPA claims, in particular, that the current standard would allow ozone levels to remain that “are sufficient to cause visible foliar injury, seedling and mature tree biomass loss, and crop reductions to degrees that could be considered adverse” (72 Fed. Reg. at 37899). To justify this change from its 1997 conclusion, EPA relies on recent field-based evidence on biomass loss in seedlings, saplings, and mature trees and on foliar injury incidence, as well as exposure and risk analyses that predict adverse effects on crops and

seedlings under scenarios that would meet the current standard (*id.* at 37893-99). In addition, EPA concludes that a cumulative seasonal standard is more appropriate than an 8-hour standard (*id.* at 37899, 37900, 37905).

As with the health effect evidence, EPA has overstated the conclusions that can be drawn from the vegetation data. There remain considerable uncertainties about the degree to which and levels at which ozone produces adverse effects on the public welfare, as well as the extent to which the proposed revised secondary standard (under either proposed option) would protect the public welfare from such effects. In fact, CASAC (2006b, p. 6) recognized that the evidence “linking specific ozone concentrations to specific vegetation/ecological effects must continue to be characterized as having high uncertainties,” although it then failed to take adequate account of those uncertainties in recommending a more stringent, separate secondary standard. While the present Commenters will leave a detailed discussion of the uncertainties to other comments, it is noted that these uncertainties include the following:

- There are significant uncertainties about the extent to which the new field-based research upon which EPA places heavy reliance is reflective of impacts on natural ecosystems. For example, it is unknown whether the individual tree growth effects reported in the AspenFACE studies in Wisconsin would translate into adverse impacts on the multiple tree species that compose an ecosystem, given competitive factors and natural ecosystem succession (see Irving Consulting, 2006, pp. 7-9). Moreover, the EPA Staff Paper acknowledges the “unknown and unquantifiable sources of uncertainty” in the TREGRO model used to estimate mature tree risks (EPA, 2007a, p. 8-2).
- The Staff Paper likewise recognizes that “[l]inking visible foliar injury to other plant effects is still problematic” (EPA, 2007a, p. 8-7).
- The estimates of crop losses continue to be based on concentration-response functions derived from the National Crop Loss Assessment Network (NCLAN) studies performed in the 1980s (see EPA, 2007a, p. 7-52), which were available and considered in the 1997 decision. The uncertainties relating to those estimates, which were recognized by EPA at that time and continue to be recognized (EPA, 2007a, pp. 7-52 - 7-53), remain.

- “[V]ast rural areas of the U.S., where important crops and natural vegetation occur, still do not have O₃ monitor coverage” (EPA, 2007a, p. 7-24). As a result, there are insufficient data to validate or judge the model predictions. EPA acknowledges the “inherent uncertainties in the interpolation that must rely on sparse data that, for the most part, are representative of urban and near-urban areas” (EPA, 2007a, p. 8-8).
- The same underestimates of natural background ozone levels and failure to adequately consider extremes of background that were discussed above with respect to the primary standard lead to overestimates of the improvements in vegetative growth that may result from reducing the secondary standard (see Irving Consulting, 2006, p. 6-7). In fact, the proposed secondary standard may be within the range of uncontrollable background and thus not produce the benefits claimed (see Heuss, 2007, pp.12-14).
- The extent to which the proposed cumulative seasonal form of the standard, known as W126, would provide increased protection of vegetation is also uncertain. For example, as shown by Lefohn (2006), use of that measure by itself will not reliably predict vegetation effects because the same values can lead to very different amounts of foliar injury and damage depending on the number of peak concentrations ≥ 0.10 ppm. Further, EPA notes that one of the reasons for selecting the W126 measure is that, since the new estimates of PRB are lower than those in the last review, this measure would not be significantly influenced by concentrations within the range of PRB (72 Fed. Reg. at 37900). This rationale, however, is unjustified because EPA’s model-predicted PRB is underestimated, as shown in Section IV above.
- In estimating the air quality that would result from just attaining the proposed standard, EPA used the Quadratic Rollback approach to adjust hourly ozone concentrations. That approach is problematic for the same reasons discussed in Section IV.
- The extent to which the predicted effects on vegetation from ozone exposure at levels below the current standard would be adverse to the public welfare depends on the intended use of the vegetation and its significance to the public. These factors have not been adequately explored. Moreover, in terms of evaluating the overall impact of the

current standard versus the proposed standard on the public welfare, the effects on vegetation need to be weighed against the adverse effects on local businesses and communities that would result from adoption of the revised standard.

- The current monitoring network is not appropriate for evaluating vegetation exposure in terms of spatial coverage and monitor height, and it is unclear whether adequate monitoring and modeling tools are available to implement a new standard in a different form from the primary standard (see Heuss, 2007, pp. 17-18).

In the Proposed Rule, EPA “recognizes that there remain significant uncertainties in determining or quantifying the degree of risk attributable to varying levels of O₃ exposure, the degree of protection that any specific cumulative, seasonal standard would produce, and the associated potential for error in determining the standard that will provide a requisite degree of protection – *i.e.*, sufficient but not more than what is necessary” (72 Fed. Reg. at 37905). As noted above, CASAC likewise recognized the existence of “high uncertainties” in the evidence on the vegetation/ecological effects of ozone. However, in its conclusions and proposal, EPA does not take into account those uncertainties or the others listed above. These uncertainties need to be addressed before adopting a new and more stringent standard that would produce substantial and widespread adverse impacts on businesses and communities with only uncertain benefits to the public welfare. In this situation, the Commenters believe that the current information is insufficient to justify adopting either of the proposed secondary standards, and that the current standard should be retained while additional research is conducted to address the many remaining uncertainties.

VII. CONCLUSION

For the reasons discussed above, the tremendous costs and other adverse impacts to businesses and communities that would result from tightening the standard are not necessary because the scientific evidence does not demonstrate that a reduction in the standard is needed to protect the public health and welfare. Accordingly, the Commenters urge EPA to retain the current standard as both the primary and the secondary standard.

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EXHIBIT A

Comments of E. R. McFadden, Jr., M.D., on Health Significance of Asthmatics' Responses Reported in Clinical Studies of Short-Term Ozone Exposure

September 14, 2007

My name is Regis McFadden. I am the Argyll J. Beams Professor of Medicine and the Vice-Chairman for Clinical Research, Department of Medicine, MetroHealth Medical Center, Case Western Reserve University School of Medicine. My curriculum vitae is attached. I have had long experience in treating and studying patients with asthma and have published extensively on exercise-induced asthma, airway reactivity, and the effects of various stimuli on airway physiology in asthmatics. I have been particularly interested in examining the relationship between lung function abnormalities and the development and resolution of acute episodes of asthma.

I have been asked by a group of industry trade associations and companies to provide my views on some of the potential issues raised by the United States Environmental Protection Agency (EPA) in its current review and proposal regarding the National Ambient Air Quality Standards for ozone (EPA, 2007). These issues relate to the health significance for asthmatics of the types of responses that have been reported in clinical studies of short-term exposures to ozone at levels close to the current ozone standard of 0.08 ppm. More specifically, I have been asked to comment on: (1) the health significance for asthmatics of functional changes involving FEV₁ decrements $\geq 10\%$ but $< 20\%$ and/or symptomatic responses (discussed by EPA, 2007, p, 37850); (2) whether the existing clinical studies on healthy subjects exposed to ozone levels at, above, and below 0.08 ppm have shown medically significant effects (see EPA, 2007, pp. 37878-79); and (3) whether the clinical evidence indicates that people with asthma are likely to experience larger and more serious effects than healthy people (see EPA, 2007, pp. 37878-79).

General Discussion of Functional and Symptomatic Changes

For a number of reasons, transient decreases in FEV₁ of 10-20% are not by themselves significant or meaningful to asthmatics. Such changes are an expected part of the asthmatic diathesis and commonly occur, particularly overnight. In fact, until most recently this type of variation was sought as one of the diagnostic criteria to establish the presence of the disease. These responses are spontaneously reversible and short-lived, with rapid onset and prompt abatement. Further, they are exactly the kinds of quantitative and qualitative bronchoconstrictive responses that are deliberately provoked in laboratories during challenges with various airway antagonists such as methacholine, histamine, exercise, and numerous other stimuli, in order to diagnose whether a

patient has asthma. These are not major effects in themselves and cannot be extrapolated to longer-term effects. It has been my experience from examining and studying thousands of patients for both clinical and research purposes that asthmatics typically will not begin to sense bronchoconstriction until their FEV₁ falls about 50% from normal. By way of comparison, asthmatic patients presenting to a physician or emergency room for treatment of acute exacerbations of their illness often have decreases in FEV₁ of 65-70% from normal.

Similarly, relatively minor complaints, such as wheezing, shortness of breath, or chest tightness, which are short-lived and disappear promptly when the stimulus is removed or are eliminated by use of a bronchodilator, are similar to the kinds of reactions that asthmatics experience frequently and tolerate well. It is important to appreciate that the presence and intensity of symptoms are a function of acute changes in physiology as filtered by the patient's sensory perception. Failure of perception is rare, whereas heightened sensitivity in asthmatics is extremely common. I would regard symptoms such as those described above as more in the nature of temporary annoyances rather than adverse health effects.

On the other hand, when the functional changes are not promptly reversible and/or are accompanied by more severe or long-lasting symptoms, they may be regarded as adverse.

In the present context, in considering whether to reduce the level of the current ozone standard, the key question is whether exposure to ozone at levels at and below the current standard produces the latter types of effects.

Review of Clinical Evidence

In evaluating this question, I have first reviewed the recent clinical studies by Adams (2002, 2003, 2006) on the effects of ozone exposures at 0.12, 0.08, 0.06, and 0.04 ppm on healthy subjects. In these studies, the functional changes reported at ozone levels ≤ 0.08 ppm were quite minimal and likely non-detectable by the subjects. I would not regard such events as medically significant. The symptoms reported were barely discerned and the maximum effect represented only a 6% increase over control. For example, the total mean symptom scores on the scale reported by Adams (2006) were only 8-10 units at 0.08 ppm and 2-4 units at 0.04 and 0.06 ppm out of a possible total score of 160.

The next question is whether asthmatics would likely have larger and more serious effects at these ozone levels than those observed in the healthy subjects. Unfortunately, conjecture will not answer this question and one must base conclusions on data.

To my knowledge there are no data on the effect of ≤ 0.08 ppm of ozone on asthmatics. However, to provide some insight into the likely magnitude of the problem, I reviewed a number of earlier studies that examined the effects on asthmatics of ozone exposures at levels of ~ 0.12 ppm and higher (Linn *et al.*, 1994; Weymer *et al.*, 1994; Horstman *et al.*, 1995; Kreit *et al.*, 1989; McBride *et al.*, 1994; Koenig *et al.*, 1985, 1987, 1988). These studies do not show any major effects. Both the functional changes and symptoms (where reported) from ozone exposure at levels of around 0.12 ppm are small. For example, in the Linn *et al.* (1994) investigation, the mean total symptom scores for asthmatics at an ozone concentration of 0.12 ppm were in the range of 20-40 (compared to around 20-30 in clean air) (Fig. 4), while in the Weymer *et al.* (1994) investigation, the mean total symptom scores after exposure to ozone at 0.10 and 0.25 ppm were in the range of 13-17 units (compared to ~ 8 in clean air) (Table 4). Given the large total possible symptom score ranges, these scores are not indicative of significant symptoms. The total possible symptom score in the Linn *et al.* trial (1994, p. 437) appears to have been 480 and that in the Weymer *et al.* investigation (1994, Table 4 note) 520.

Overall, these studies on asthmatics indicate that ozone exposures at ~ 0.12 ppm do not produce medically significant functional changes and are right around the inflection point where one begins to see an increase in symptoms; however, that increase is small. Based on this information, it would be expected that asthmatics' responses to ozone exposure at levels ≤ 0.08 ppm would be even less. It is my view that the available data are not sufficiently robust to indicate that such exposures would present a significant health concern even to sensitive people like asthmatics.

I have also reviewed the general statements which EPA relies upon to support its conclusion that asthmatics would be expected to have larger and more serious effects than healthy people (EPA, 2007, pp. 37826 and 37846-47). As a group, these statements represent a series of assumptions about the pathogenesis of asthma detailing the purported events leading to alterations in physiology and subsequent symptomatic manifestations. While appealing, it must be remembered that they are indeed only assumptions. There simply are no data to support the sequence described or to establish how the type and/or magnitude of inflammation relates to signs, symptoms, and/or the pathogenesis of asthma. Further, the translation between these types of responses and the onset or worsening of asthma or an increase in effects of health significance to asthmatics has not been established. Thus, the assumption that these responses would lead to clinical manifestations in terms of exacerbations of asthma or other adverse health effects remains unproven theory.

Moreover, even if the hypothesized relationships were true, they would apply to the effects of various stimuli, including ozone *generally*, but they do not address the specific question of the level at which ozone would have such effects. Much

of the data discussed represent compilations derived from a series of studies in a variety of models with different ozone exposure concentrations. Unfortunately, the applicability and/or precision of such information is not yet established. Thus, these statements do not tell us whether ozone would have such effects at concentrations at and below the current standard level. As discussed above, the actual clinical studies on asthmatics exposed to ozone do not indicate that ozone exposure at such low concentrations produces effects that are harmful to asthmatics' health. For these reasons, I do not think that EPA's statements provide reliable support for lowering the ozone standard.

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EXHIBIT B

Excerpts from Statements of Other Physicians Specializing in Asthma on Significance of Reductions in FEV₁ of up to 20 Percent

Note: These Statements were initially submitted to EPA in 1995 by the National Mining Association in connection with comments on the NAAQS for sulfur oxides (NMA, 1995, Appendix A). Full copies of these experts' Statements, including the curricula vitae of the experts, were also provided as exhibits to the September 18, 2006 comments of a group of trade associations that include the present Commenters on EPA's second draft of the Staff Paper on the NAAQS for ozone (Alliance of Automobile Manufacturers et al., 2006).

- Dr. Jeffrey Drazen (1995): “[T]here is ample precedent that transient changes in the FEV₁ on the average of 20% are well tolerated by patients with asthma and place them at no medical risk.”
- Dr. Richard Lemen (1995): “[C]hanges of 15-10% in FEV_{1.0} reported in the EPA-cited studies could be largely within the ‘noise level’ of pulmonary function tests. . . . [Such responses] are not indicative of medically significant effects. Unlike real asthma attacks, the changes in FEV_{1.0} and SRaw observed in the SO₂ studies, even in the more responsive subjects, are relatively modest in magnitude and are transient and quickly reversible, returning to normal after a short period. For comparison, asthma patients who seek medical treatment for asthma attacks in emergency rooms or hospitals generally have much larger decrements in lung function . . . , and these changes are not transient but have often persisted for hours or days.”
- Dr. Sheldon Spector (1995): “The changes in pulmonary function reported in the studies are relatively small, and do not appear to lead to any late phase effect which often indicates that an inflammatory process is involved. In my experience, using FEV₁ as a measurement, a fall of 20-50% usually does not produce significant symptoms.”
- Dr. Edward Eden (1995): “The asthmatics who engage in such exercise tolerate readily a drop in pulmonary function of the type shown in the SO₂ studies (i.e., a mean decrease in FEV₁ of around 10-30% with greater changes in the more sensitive subjects).”
- Dr. Stuart Brooks (1995): “The clinical significance of a 20% to 40% fall in FEV-1 for a mild asthmatic capable of vigorous exercise is doubtful. An asthmatic may experience such a decrement not infrequently, and suffer no serious consequences or be put at any increased risk for future detrimental outcomes.”
- Dr. Nicholas Gross (1995): “[B]ronchial challenge (provocation) tests with methacholine, for example, that are routinely performed on asthmatics take a 20% decline in FEV₁ as their endpoint, which means of course that greater decreases will in fact be produced in all hyperresponsive subjects. Such tests are performed in thousands of clinics and doctors’ offices every day and are regarded as safe.”

- Dr. Adam Wanner (1995): “A [10-25%] fall in FEV₁ during bronchoconstriction induced by inhaled antigen, or non-allergenic bronchoconstricting stimuli . . . is rarely associated with cough, wheezing, shortness of breath or other asthma symptoms This may be one of the reasons why a 20% fall in FEV₁ has been chosen by convention as the target bronchoconstriction in bronchial provocation testing.”
- Dr. Peter Hamm (1995): “In my experience, . . . such a drop [in FEV₁ of 30% or more] will not yield a clinically relevant outcome in an [asthmatic] individual with sufficient pulmonary reserve.”
- Dr. Byron Cooper (1995): “The EPA staff regarded changes in FEV₁ as severe if they exceeded 20%. In fact the 20% figure is the minimum considered significant in a pulmonary function lab and is deliberately provoked when doing methacholine challenge. Asthmatics with a 20% reduction from normal are considered mild.”