



Natural Gas Electricity

October 2014

Volume 31
Number 3

THE MONTHLY JOURNAL FOR PRODUCERS, MARKETERS, PIPELINES, DISTRIBUTORS, AND END-USERS

EPA May Go Beyond Law and Science in Setting NAAQS

Lucy Fraiser

Information from highly respected sources, such as the American Cancer Society,¹ indicates that only about 2 percent of cancer deaths in the United States are the result of exposures to carcinogenic agents in the workplace, community, and other settings. But the news about health risks from environmental exposures to air pollutants is almost constant. Although the US air quality has improved dramatically in the last 30 years, each recent review of a National Ambient Air Quality Standard (NAAQS) by the US Environmental Protection Agency (EPA) has resulted in a reduction in existing standards or replacement by a completely different and generally more stringent standard.

Only about 2 percent of cancer deaths in the United States are the result of exposures to carcinogenic agents in the workplace, community, and other settings.

The EPA lowered the eight-hour ozone (O₃) NAAQS from 80 parts per billion to 75 in 2008. The O₃ standard is under review now and, given the tone of the draft Policy Assessment for O₃, additional reductions appear imminent. The EPA established new one-hour NAAQS of 100 parts per billion and 75 parts per billion for nitrogen dioxide (NO₂) and

Lucy Fraiser (lfraiser@zephyrenv.com) is a principal with Zephyr Environmental.

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Natural Gas & Electricity

Associate Publisher: Robert E. Willett Executive Editor: Margaret Cummins

Natural Gas & Electricity (ISSN 1545-7893, Online ISSN 1545-7907 at Wiley Online Library, wileyonlinelibrary.com) is published monthly, 12 issues per year, by Wiley Subscription Services, Inc., a Wiley Company, 111 River Street, Hoboken, NJ 07030-5774. Copyright © 2014 Wiley Periodicals, Inc., a Wiley Company. All rights reserved.

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Subscription price (2014): One year print only: \$1,881 in U.S., Canada, and Mexico; \$1,953 outside North America. Electronic only: \$1,881 worldwide. A combination price of \$2,164 in U.S., Canada, and Mexico, \$2,236 outside North America, includes the subscription in both electronic and print formats. All subscriptions containing a print element, shipped outside U.S., will be sent by air. Payment must be made in U.S. dollars drawn on a U.S. bank. Claims for undelivered copies will be accepted only after the following issue has been received. Please enclose a copy of the mailing label. Missing copies will be supplied when losses have been sustained in transit and where reserve stock permits. Please allow four weeks for processing a change of address. Address subscription inquiries to Subscription Manager, Jossey-Bass, a Wiley Company, One Montgomery Street, Suite 1200, San Francisco, CA 94104-4594; Tel.: (888) 378-2537, (415) 433-1767 (International); E-mail: jbsubs@wiley.com.

Postmaster: Send address changes to Natural Gas & Electricity, Subscription Distribution US, c/o John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030-5774.

Reprints: Reprint sales and inquiries should be directed to Gale Krouser, Customer Service Department, c/o John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030-5774. Tel: (201) 748-8789. E-mail: gkrouser@wiley.com.

Other Correspondence: Address all other correspondence to: Natural Gas & Electricity, Margaret Cummins, Executive Editor, Professional Development Division, c/o John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030-5774.

Indexed by ABI/Inform Database (ProQuest) and Environment Abstracts (LexisNexis).

Editorial Production, Wiley Periodicals, Inc.: Ross Horowitz

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sulfur dioxide (SO₂), respectively, in 2010. In 2006, the EPA retained the annual NAAQS for fine particles less than 2.5 micrometers in diameter (PM_{2.5}) at 15 micrograms per cubic meter but reduced the 24-hour PM_{2.5} standard from 65 micrograms per cubic meter to 35 and then promulgated a more stringent annual PM_{2.5} standard in December 2012, lowering it from 15 micrograms per cubic meter to 12.

Each recent review of a National Ambient Air Quality Standard by the US Environmental Protection Agency has resulted in a reduction in existing standards or replacement by a completely different and generally more stringent standard.

Proponents of more stringent environmental regulation have continually offered the opinion that the significant positive benefits of reducing NAAQS to ever-lower levels are undisputed. However, for some recent decisions on NAAQS, there is good reason to believe that an adequate foundation in scientific merit is lacking.

Proponents . . . have continually offered the opinion that the significant positive benefits of reducing NAAQS to ever-lower levels are undisputed.

WHAT CONSTITUTES AN “ADVERSE HEALTH EFFECT” AND “MARGIN OF SAFETY”

The Clean Air Act (CAA) requires that primary NAAQS that are protective of public health, with an adequate margin of safety, and secondary NAAQS that are protective of public welfare to be established. “Public welfare” is defined as including “effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility and climate.” Examples of what is intended by “public health” are not similarly provided, nor is a definition of what constitutes an “adequate margin of safety.”

The CAA’s silence on the definition of “public health” and “adequate margin of safety” has forced the EPA to make public health decisions without a clear definition of what constitutes an “adverse effect” or the degree of protection intended by the regulation.

Morton Lippmann, a participant in the EPA’s Clean Air Scientific Advisory Committee reviews

of Criteria Documents for NAAQS compounds between 1980 and 1987, opined on the EPA’s interpretation of its statutory authority in setting NAAQS at the time the 1970 amendments to the CAA were enacted.² He believed primary standards are not intended to protect against all identifiable effects, but only those judged by the EPA administrator to be “adverse.” However, because the primary NAAQS were intended by Congress to be precautionary and preventive, the EPA administrator is not free to define only those effects that are clearly harmful or for which there is a medical consensus about the degree of harm as “adverse.” Lippmann further opined that a standard is statutorily sufficient when there is “an absence of adverse effect on the health of a statistically related sample of persons in sensitive groups from exposure to the ambient air.”

The American Thoracic Society (ATS) has provided guidance on the distinction between “adverse” and “non-adverse” health effects. The ATS also acknowledges that the CAA suggests that the adequacy of any standard could be tested in a statistically representative sample of sensitive individuals.³

The ATS guidance indicates that healthy people may sustain transient reductions in pulmonary function with exposure to air pollutants but recommends that reversible loss of lung function only be considered “adverse” when it is accompanied by respiratory symptoms. The ATS further recommends that the following be considered “adverse”: (1) any detectable level of permanent lung function loss attributable to air pollution, (2) air-pollution-related symptoms associated with diminished quality of life or with a change in clinical status (e.g., requiring medical care or change in medication), and (3) any effect on mortality.

While previous ATS guidance⁴ hinged the distinction between “adverse” and “non-adverse” effects on medical considerations, the most recent ATS guidance⁵ places increased emphasis on quality-of-life measures as indicators of adversity and formally acknowledges the concept that minute individual risks may be significant from a population standpoint, even if no individual experiences a level of exposure that is associated with clinically relevant consequences. This guidance seems to have opened the door to considering increasingly benign and isolated effects that occur in very few test sub-

jects in controlled human studies as relevant in the NAAQS setting process.

KEY SUPPORT FOR RECENTLY REDUCED PRIMARY NAAQS

Controlled human exposure studies evaluating effects on lung function provide the primary support for the one-hour NAAQS for SO₂ and the eight-hour O₃ NAAQS.

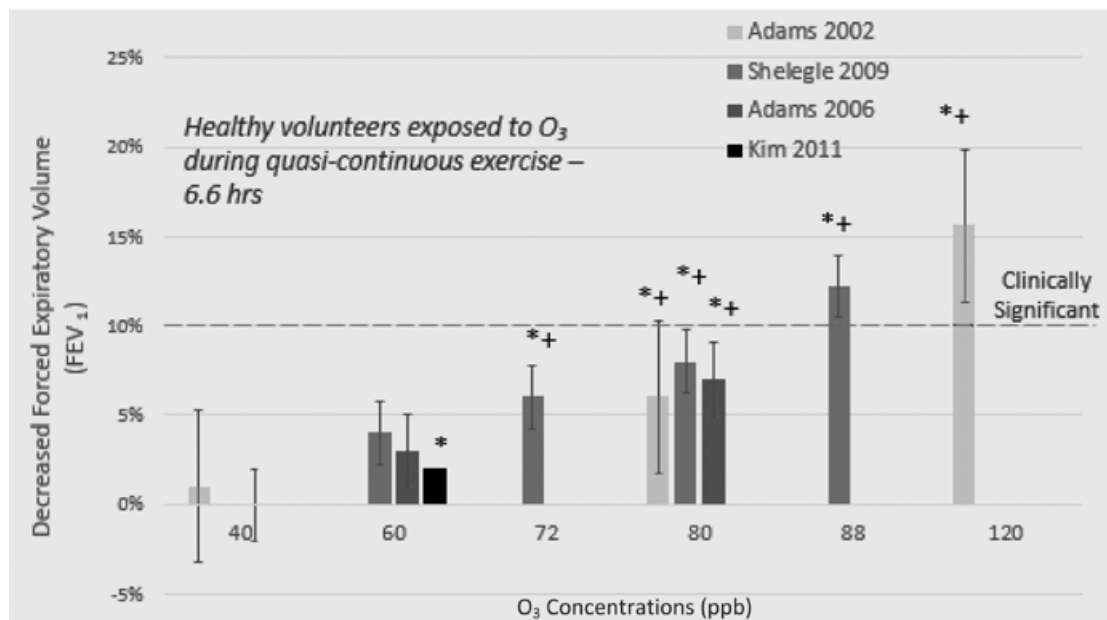
Ozone

The EPA is currently considering lowering the existing eight-hour O₃ NAAQS of 75 parts per billion to a level between 60 parts per billion and 70 parts per billion because of concerns about the adequacy of its health protectiveness. One of the early effects of O₃ exposure is transient lung-function decrements, which has been demonstrated in controlled human exposure studies in young healthy adults exposed to O₃ while engaging in moderate, semicontinu-

ous exercise for more than six hours. **Exhibit 1** summarizes the results of the controlled human studies that the EPA is relying on in its current, ongoing O₃ NAAQS review. There are more short-term O₃ studies than those shown in Exhibit 1, but only four controlled human studies have evaluated short-term O₃ exposures below 80 parts per billion. These are the studies that provide the primary basis for the EPA's questions about the adequacy of the current eight-hour NAAQS of 75 parts per billion.

Forced expiratory volume in one second (FEV₁), a measure of how much air a person can exhale during a forced breath, and respiratory symptoms were selected as critical health endpoints for judging the respiratory effects of O₃. The EPA has outlined a graded classification for lung-function changes in which changes in FEV₁ were graded as mild, moderate, or severe for reductions of less than 10 percent, 10–20 percent, and greater than 20 percent, respectively.

Exhibit 1. Evidence for O₃-Induced Lung-Function Decrements



* statistically significant.
 *+ statistically significant + respiratory symptoms.

Sources: Adams, W. (2002). Comparison of chamber and face-mask 6.6-hour exposures to ozone on pulmonary function and symptoms responses. *Inhalant Toxicology*, 14, 745–764; Schelegle, E., Morales, C., Walby, W., Marion, S., & Allen, R. (2009). Hour inhalation of ozone concentrations from 60 to 87 parts per billion in healthy humans. *American Journal of Respiratory Critical Care Medicine* 180, 265–272; Adams, W. (2006). Comparison of 6.6 h exposures to 0.04–0.08 ppm ozone via square-wave and triangular profiles on pulmonary responses. *Inhalant Toxicology*, 18, 127–136.

A moderate decrease in FEV₁ of greater than or equal to 10 percent has been judged by the EPA to be a clinically relevant change. As shown in Exhibit 1, FEV₁ decrements are not accompanied by respiratory symptoms (required to be considered “adverse”) until O₃ concentrations reach 72 parts per billion and clinically relevant lung-function decrements (decrease of at least 10 percent) do not occur until O₃ concentrations reach 88 parts per billion.

Clinically relevant lung-function decrements . . . do not occur until O₃ concentrations reach 88 parts per billion.

The EPA acknowledges that group mean changes in lung function, such as those shown in Exhibit 1, are small following exposures to O₃ concentrations in the range of 60–70 parts per billion. But the EPA still argues that some sensitive individuals experience clinically meaningful decrements at these levels because in each study summarized in Exhibit 1, one or two subjects experienced greater than or equal to 10 percent decreases in FEV₁. However, this conclusion is entirely dependent upon the assumption that a 10 percent decrease in FEV₁ represents a clinically relevant benchmark.

This assumption is questionable for several reasons. Many studies have demonstrated that there is a high degree of spontaneous day-to-day and diurnal variation in FEV₁ and that a 10 percent drop in FEV₁ is within the range of normal variation, even in healthy individuals that are neither exercising nor exposed to pulmonary irritants.⁶ In its justification of the 10 percent decrease in FEV₁ as the threshold for clinical relevance, the EPA states that some asthmatics would choose to self-limit activities and might require additional or more frequent use of asthma medication. However, there are many studies⁷ that dispute this statement, indicating instead that asthma symptoms and medication use, as well as “quality-of-life” indicators, such as self-reported activity restriction, are poorly correlated with pulmonary function tests such as FEV₁.

Sulfur Dioxide

The most immediate effect of SO₂ exposure on the respiratory system is bronchoconstric-

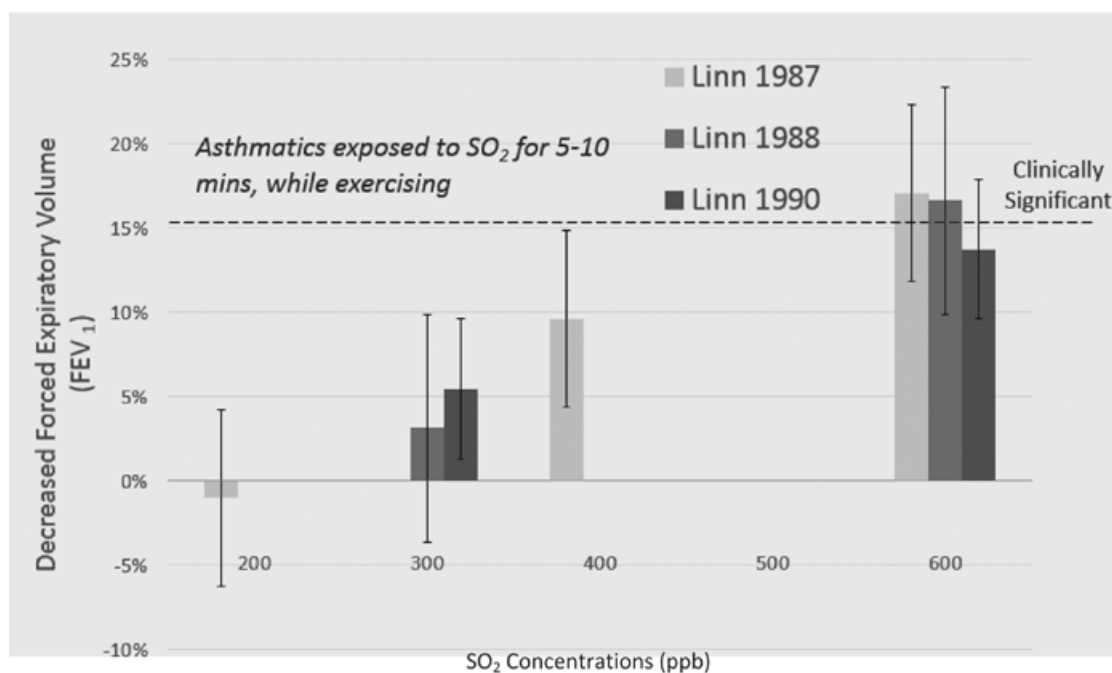
tion. FEV₁ and airway resistance (sRAW) were selected as the primary critical health endpoints for judging the respiratory effects of SO₂. For SO₂, the EPA judges a moderate decrease in FEV₁ to be clinically relevant but considers a 15 percent decrease to be the threshold (as opposed to the 10 percent threshold used for O₃).

Exhibit 2 summarizes the results of several controlled human studies that evaluated the effect of short-term SO₂ exposure in exercising asthmatics on lung function, as measured by decrease in FEV₁, and that were relied upon by the EPA in establishing the one-hour SO₂ NAAQS in the 2010 NAAQS review. There are other controlled human studies that evaluated SO₂-induced decreases in FEV₁, but those in Exhibit 2 represent the ones for which the EPA’s raw data were made available. The studies in Exhibit 2 are representative of the others in terms of results. None of the Linn studies reported whether the decrements were statistically significant or whether they were accompanied by respiratory symptoms, but as the exhibit shows, the group mean decreases in FEV₁ did not reach the 15 percent threshold level required to be considered clinically relevant in any of the studies until SO₂ concentrations reached 600 parts per billion.

In addition to classifying FEV₁ changes, the EPA has also outlined a classification system for sRAW, in which increases in sRAW of less than 100 percent, 100–200 percent, and greater than 200 percent are graded as small, moderate, and large, respectively. The EPA considers a moderate increase in sRAW as the threshold for clinical relevance. **Exhibit 3** summarizes the results of controlled human studies on the effect of short-term SO₂ exposure in exercising asthmatics on lung function measured as an increase in sRAW. As shown in the exhibit, a statistically significant and clinically relevant group mean increase in airway resistance (greater than 100 percent) was observed at a concentration of 500 parts per billion, but not below. A statistically significant increase in airway resistance large enough to be considered clinically relevant (greater than 100 percent) that was also accompanied by respiratory symptoms (required to be considered adverse) was only reported in one study, and it was not observed until the SO₂ concentration reached 1,000 parts per billion.

Surprisingly, on the basis of these studies, the EPA concluded that respiratory effects were

Exhibit 2. Evidence for SO₂-Induced Lung-Function Decrements, Measured as Decreases in FEV₁



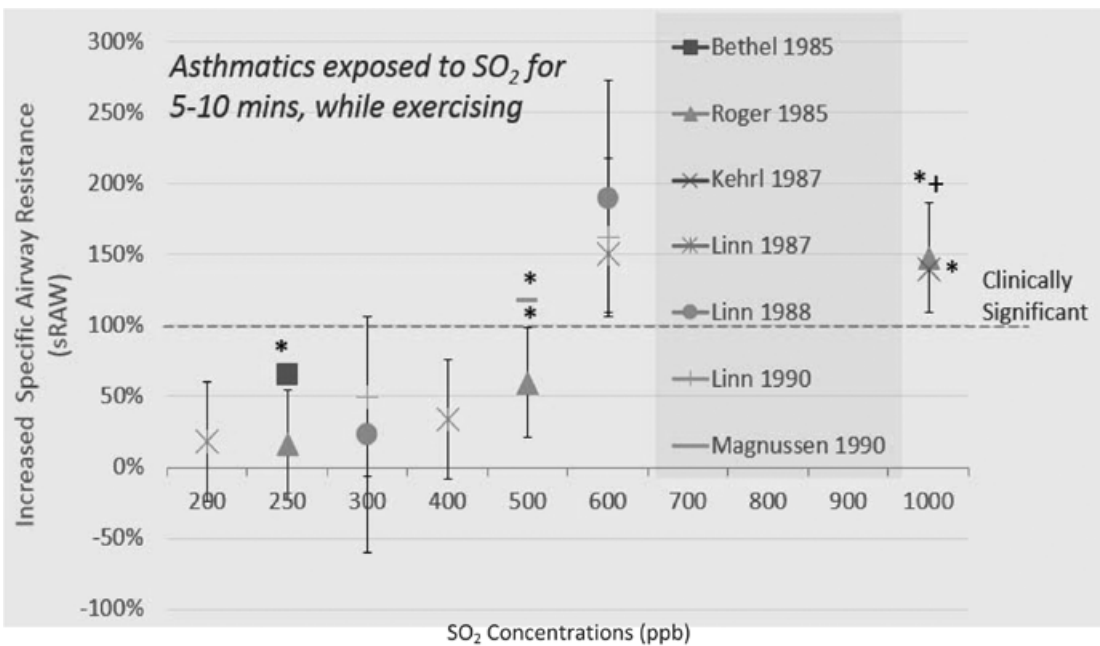
Sources: Linn, W., Avol, E., Peng, R., Shamoo, D., & Hackney, J. (1987). Replicated dose-response study of sulfur dioxide effects in normal, atopic, and asthmatic volunteers. *American Review of Respiratory Diseases*, 136, 1127–1134; Linn, W., Avol, E., Shamoo, D., Peng, R., & Hackney, J. (1988). Effect of metaproterenol sulfate on mild asthmatics' response to sulfur dioxide exposure and exercise. *Archives of Environmental Health*, 43, 399–406; Linn, W., Shamoo, D., Peng, R., Clark, K., Avol, E., & Hackney, J. (1990). Responses to sulfur dioxide and exercise by medication-dependent asthmatics: Effect of varying medication levels. *Archives of Environmental Health*, 45, 24–30.

consistently observed following five-to-ten-minute exposures to SO₂ at concentrations of more than 200 parts per billion in asthmatics engaged in moderate to heavy levels of exercise. Again, while the group mean lung-function decrements were small, the EPA argued that a few sensitive individuals experienced clinically relevant decreases in FEV₁ (greater than or equal to 15 percent) or increases in sRAW (greater than 100 percent). From that five-to-ten-minute 200 parts per billion SO₂ level, the EPA calculated an hourly concentration of 75 parts per billion and set that level as the one-hour SO₂ NAAQS. However, the symptoms observed at 200 parts per billion were mild and occurred in only a small number of participants (i.e., effect was not consistently observed).

Studies have shown that, like FEV₁, sRAW varies substantially between individuals, which

can lead to isolated effects that occur in only a few individuals and are most likely unrelated to exposure. Bethel⁸ reported that baseline (i.e., pre-exercise and pre-exposure) sRAW values collected on two consecutive days from 28 volunteers varied by as much as 53 percent and that 11 of the 28 volunteers experienced differences of greater than or equal to 25 percent. The range of variability in baseline sRAW values provides further evidence that lung function can vary appreciably due to factors unrelated to exposure to air pollutants. Linn and his coauthors⁹ reevaluated results from their earlier study¹⁰ and found FEV₁ decreases of greater than or equal to 15 percent in five subjects, *increases* of greater than or equal to 15 percent (opposite direction expected) in five other subjects, and an overall group mean response that was not different from the control group.

Exhibit 3. Evidence for SO₂-Induced Lung-Function Decrements, Measured as Increases in sRAW



* statistically significant.

* + statistically significant + respiratory symptoms.

Sources: Bethel, R., Sheppard, D., Geffroy, B., Tam, E., Nadel, J., & Boushey, H. (1985). Effect of 0.25 ppm sulfur dioxide on airway resistance in freely breathing, heavily exercising, asthmatic subjects. *American Review of Respiratory Diseases*, 131, 659–661; Roger, L., Kehrl, H., Hazucha, M., & Horstman, D. (1985). Bronchoconstriction in asthmatics exposed to sulfur dioxide during repeated exercise. *Journal of Applied Physiology*, 59, 784–791. Kehrl, H., Roger, L., Hazucha, M., & Horstman, D. (1987). Differing response of asthmatics to SO₂ exposure with continuous and intermittent exercise. *American Review of Respiratory Diseases*, 135, 350–355; Linn, W., Avol, E., Peng, R., Shamoo, D., & Hackney, J. (1987). Replicated dose-response study of sulfur dioxide effects in normal, atopic, and asthmatic volunteers. *American Review of Respiratory Diseases*, 136, 1127–1134; Linn, W., Avol, E., Shamoo, D., Peng, R., & Hackney, J. (1988). Effect of metaproterenol sulfate on mild asthmatics' response to sulfur dioxide exposure and exercise. *Archives of Environmental Health*, 43, 399–406; Linn, W., Shamoo, D., Peng, R., Clark, K., Avol, E., & Hackney, J. (1990). Responses to sulfur dioxide and exercise by medication-dependent asthmatics: Effect of varying medication levels. *Archives of Environmental Health*, 45, 24–30.

Comparable changes in sRAW occurred with a similar frequency.

Just as the lung-function improvements seen in a few individuals exposed to 200 parts per billion do not indicate a beneficial effect of SO₂, the small lung-function decrements after low-level SO₂ exposure do not represent an “adverse” effect of SO₂.

CONCLUSIONS

There have been recent efforts to formalize processes for characterizing the weight of the scientific evidence and judging the strengths and limitations of individual studies that may affect the overall interpretation of the results.

Nevertheless, there has been inconsistency in the way the EPA has interpreted health effects studies used to support its decisions on setting NAAQS. The examples given here are but a few of those inconsistencies.

[There has been inconsistency in the way the EPA has interpreted health effects studies.](#)

Clearly, Congress did not intend that only healthy persons would be protected by the NAAQS. The legislative history of Section 109 of the CAA indicates that a primary standard is to be set at “the maximum permissible ambi-


ent air level . . . which will protect the health of any [sensitive] group of the population,” and that for this purpose “reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group.”¹¹ Based on this interpretation and similar interpretations by Lippmann¹² regarding the statutory authority given to the EPA by the CAA in setting NAAQS, it is unclear how far the EPA should go in terms of protecting “hyper-susceptible individuals” and against effects that are not clearly “adverse.” Nonetheless, the trend has been for the EPA to consider increasingly benign and isolated effects that occur in very few test subjects as relevant in the NAAQS-setting process.

The trend has been for EPA to consider increasingly benign and isolated effects that occur in very few test subjects as relevant.

In contrast to the ATS’s recommendations,¹³ the EPA often considers a moderate decrease in lung function or symptoms alone to be “adverse” in setting NAAQS. Presumably, this policy is at least partly based on the EPA’s assertion that these effects would interfere with normal activities and/or require additional or more frequent use of asthma medication. This tendency may be related to the ATS’s recently increased focus on “quality-of-life” measures as indicators of adversity. However, as previously discussed, there are many studies disputing that lung-function tests (e.g., FEV₁) are good predictors of “quality-of-life” indicators, symptoms, or asthma medication use.¹⁴

Focusing on the response of hypersensitive individuals in the test group . . . appears to go beyond the original intent of the CAA.

In addition, by focusing on small decreases in FEV₁ or increases in sRAW that only occur in a few sensitive individuals, justifications for lowering the NAAQS are being based on what most likely represents normal variation in pulmonary function tests rather than clearly “adverse” effects associated with exposure to air pollutants. Furthermore, focusing on the response of hy-

persensitive individuals in the test group, rather than responses in a representative sample of sensitive groups, appears to go beyond the original intent of the CAA. It is clear from the inconsistencies described in this article that more objective and specific criteria for weighing the scientific evidence and judging the strengths and limitations of individual studies need to be established so that judgments about reducing NAAQS are made based on truly “adverse” health effects in representative samples of sensitive subpopulations. 

NOTES

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11. S.Rep. No. 91–1196, 91st Cong., 2d Sess. 10 (1970) as referenced in 71 Fed. Reg. 2620–2708 (January 17, 2006).
12. See Note 2.
13. See Notes 3 and 4.
14. See Note 7.