Statement of the American Lung Association on EPA's

Second Draft Integrated Science Assessment for Sulfur Dioxide
Docket ID No.: EPA-HQ-ORD-2006-0260
and
First Draft Risk and Exposure Assessment to Support the Review of the SO_2 Primary National Ambient Air Quality Standards
Docket ID No.: EPA-HQ-OAR-2007-0352

Clean Air Scientific Advisory Committee
July 30-31, 2008

The current National Ambient Air Quality Standards (NAAQS) for sulfur dioxide (SO_2) were set in 1971 based on a Criteria Document (CD) published in 1969 by the Department of Health Education and Welfare. The scientific articles considered in the original CD predate 1968. In 1971, the newly established EPA set the first primary SO_2 NAAQS: an annual average standard of 0.03 ppm (80 µg/m^3); and a 24-hour standard of 0.14 ppm (365 µg/m^3).[1] The current standards are based on 40 plus year old science.

Following the promulgation of the initial SO_2 standards, a series of clinical chamber studies established that that low level exposures to SO_2 -- for periods as short as five minutes -- can be harmful to the health of people with asthma. EPA completed a review of these studies in a 1982 CD. An addendum to the CD, summarizing 16 additional controlled studies of asthmatics under exercise conditions, was published in a 1986. In 1994, EPA published a supplement to update the CD addendum.

Additionally, in the intervening years we have amassed new epidemiological studies that indicate respiratory effects at levels well below the current standards.

Ultimately, in 1996, the Agency decided not to revise the standards based primarily on the results of the exposure assessment. The exposure assessment relied on numerous assumptions about emissions, ambient concentrations, activity patterns, breathing rates, and even medication use. The cynical reasoning was that very few asthmatics were predicted to be exercising in the vicinity of a power plant plume, and if they were, they could take medication to relieve their symptoms.[2]

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[1] The California Air Resources Board has adopted a 24-hour SO_2 standard of 0.04 ppm. and a 1-hour SO_2 standard of 0.25 ppm.

[2] The discussion of medication availability as a moderating effect in this context is completely without merit. Many people with asthma --including those who use asthma medications -- do not have their

The American Lung Association believes the NAAQS should be set based solely on the medical evidence and rejects reliance on an exposure assessment to deny protection to people with asthma. EPA's reliance on the APEX model of human activity patterns seems to suggest that if people stayed inside all day, there would be no need for air quality standards.

The standards must be based on a judgment of what constitutes a safe air quality level -- not on an estimate of how many people will encounter given concentration levels. That safe level must consider the effects on sensitive populations, like people with asthma whether their asthma is controlled or not, whether they are out of doors or not. Under the Clean Air Act, the NAAQS must also provide a margin of safety, because protecting public health requires that we take precautions against what we cannot know yet with certainty. If the studies show adverse health effects at a certain level, the standards must be set below that level. That is the only way to ensure public health protection from ambient air pollution.

The Clean Air Act never contemplated that NAAQS would be based on an imperfect assessment of the numbers of people at risk, much less base our assessments on flawed assumptions about what those people are doing. Quite the opposite: the Act directed that we would protect those people at risk. The NAAQS sets that level of protection.

The American Lung Association challenged the Agency's failure to set a short-term standard in federal court, and in 1999, the court ruled in the American Lung Association's favor and sent the rule back to EPA for a rewrite.

In the wake of the remand, EPA asked states to report more 5-minute monitoring data on a voluntary basis. However, not a lot of additional data was generated, and where five-minute data was provided, monitors may not have been optimized for short-term data collection.

Ten years later, we still lack the short-term SO_2 standard to protect against peak exposures that can result from start-up, shutdown, upset, malfunction, downwash, and inversion conditions. For existing sources, emission limits in permits are tagged to the ambient standards. We lack the means to compel monitoring of peak concentrations in hotspots. We lack an up-to-date standard to ensure that new sources will not extend the health threat for asthmatics to other communities.

SO_2 control programs such as the acid rain program and the now defunct Clean Air Interstate Rule (CAIR) are trading programs that allow some utility sources to forgo controls by buying credits from other, so-called "over controlled" sources. Such trading programs fail to protect the local population nearest to the source, who face the greatest, continuing exposure. Thus, in the absence of a short-term standard for SO_2, there is no

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condition under control, as evidenced by numerous emergency room and urgent care visits, particularly among children.

way to ensure that people are protected from breathing short term spikes that can be harmful. At minimum, we need the backstop measure of a short-term standard to accompany further trading programs. We need a new standard to drive a targeted monitoring program to detect these local exposures in time and space.

In 2006, EPA directed states to consider decommissioning SO_2 monitors as part of the National Monitoring Strategy revisions. This was based on the premise there were few if any exceedances of the 40-year old standard -- without consideration of the fact that that the standard needed to be updated in light of more recent studies.

Meanwhile, the CAIR program which would have reduced power plant SO_2 emissions by fifty percent was just recently struck down by the courts. Major smelters such as Asarco in Texas are starting up again. Pollution from ocean-going vessels and other sources in ports demand attention. According to the Department of Energy, there are 45 coal-fired power plants in the pipeline and another 76 coal-fueled power plants on the drawing boards.[3] There is massive oil and gas development underway in the Rocky Mountain West. Up to date air quality standards are needed to ensure that the appropriate level of health protection is achieved with future control efforts.

We would like to offer some comments on the second draft Integrated Science Assessment (ISA).

There is a lot to like about the second draft ISA. We commend the authors, and appreciate the careful review and substantive comments on the first draft provided by CASAC.

We applaud the improvements in data presentation, especially the graphics you have included in Chapter 4 to show data from key older controlled human exposure studies, e.g. Figures 3-1, 4-2 and 4-3.

Another good example is the visual presentation of the epidemiological studies in summary charts, for example Figures 5-1 and 5-2.

Your emphasis on quantification of the levels at which effects were observed in the human clinical and animal toxicology studies, Tables 3-1, 5-1 and 5-2 helps to focus the discussion.

We welcome how you have drawn key conclusions about various health endpoints, and provided comparisons to previous documents, Table 5-3.

The carefully focused charge questions you developed should greatly assist the CASAC and public review of these documents.

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[3] National Energy Technology Laboratory. Tracking New Coal-Fired Power Plants, February 18, 2008.

We have some suggestions for improvements. Given that the current administration has eliminated the Staff Paper, we hope that the ISA will include even more of the analyses that used to be found in the Staff Paper.

The discussion in chapter 2 of monitor siting does not address network design. This is a key concern which carries forward into the exposure assessment. How many, and which of the monitors in the network are source-oriented monitors sited to capture maximum concentrations? If monitors were sited for other purposes, they are not likely to be relevant to the question of peak 5-minute exposures, or for the calculation of peak to mean ratios. The monitor objective and scale provide some insight into this question, but these data fields are not always reported to the Air Quality System database.

The ISA should not gloss over the results of earlier controlled human exposure studies finding adverse effects at low concentrations. Two figures below, drawn from an Australian assessment, are very informative in graphically indicating that risks to asthmatics begin to increase at 0.2 ppm.[4] These figures are derived from data provided by Linn et al (1987). They show average changes in FEV_1 and total symptom scores after exposure for approximately 15 minutes for three groups of exercising subjects - those with no asthma, those with mild asthma and those with severe asthma.

As figure 2 indicates, a mean FEV_1 decline of approximately 6-8 percent was observed between 0.2 and 0.4 ppm for the mild and the moderate/severe asthma groups.

The symptoms score illustrated in figure 3 represents a sum of a range of symptoms measured on a scale of 0 to 40 where 0= not present and 40 = incapacitating. The symptoms used in the score included cough, sputum, substernal irritation, wheeze, chest tightness, shortness of breath, throat irritation, nasal discharge/congestion, fatigue, headache, eye irritation, and other) (Linn et al, 1983).

[4] National Environment Protection Council. National Environment Protection (Ambient Air Quality) Measure. Review of the Practicability of a 10 Minute Sulfur Dioxide Standard. Issues Paper, March 2004.

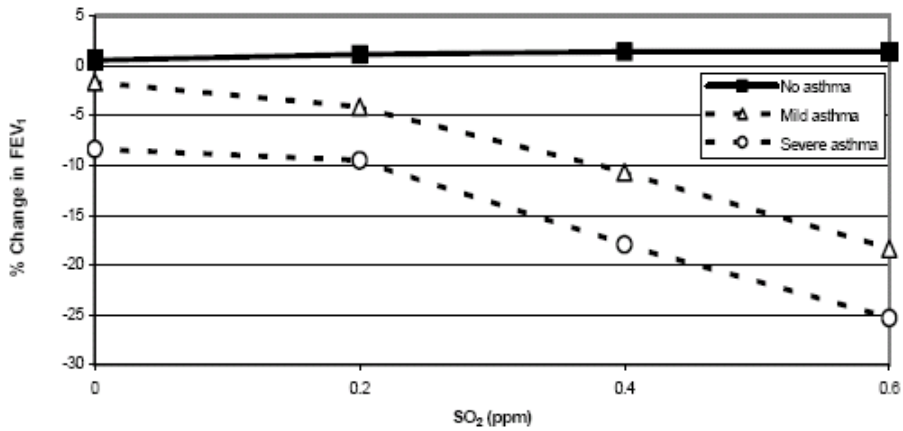

Figure 2: Mean percentage change in FEV_1 associated with sulfur dioxide exposures for approximately 15 minutes

Source: Health Impacts of Ozone and Sulfur Dioxide, Woolcock Institute of Medical Research, 2003 (using data provided in Linn et al, 1987)

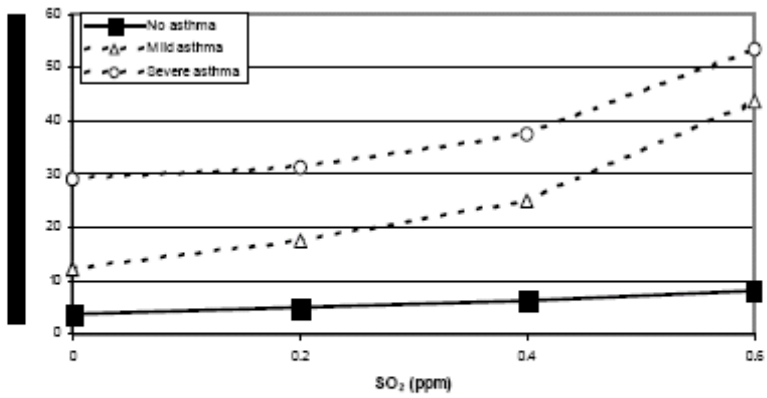

Figure 3: Mean absolute change in symptom scores with sulfur dioxide exposures for approximately 15 minutes

Source: Health Impacts of Ozone and Sulfur Dioxide, Woolcock Institute of Medical Research, 2003 (using data provided in Linn et al, 1987)

These or similar figures and discussion should be included in the ISA, and the effects at concentrations of 0.2 ppm should be highlighted in Table 5-1 of the ISA, and summarized in the key findings in Table 5-3.

Also, the ISA should note that there is evidence of changes in airway resistance at concentrations as low as 100 ppb when combined with exercise for a period of several minutes. A controlled human exposure study examined changes in specific airway resistance in seven exercising subjects with mild asthma. Investigators reported that 2 of

the 7 subjects experienced increased airway resistance after inhalation of 0.10 ppm.[5] SO_2 in this study was administered by mouthpiece. This study should also be highlighted in Table 5-1 of the ISA, and summarized in the key findings in Table 5-3. A subsequent study reported that both oral and oronasal breathing of low concentrations of SO_2 during exercise can cause significant bronchoconstriction in people with asthma.[6]

In evaluating the controlled human exposure studies, it is important to consider the responses by individual subjects as well as the group mean responses. According to one of the nation's leading experts, the controlled human exposure studies provided clear evidence that responsive asthmatics exposed to concentrations of SO_2 above 0.25 ppm for 3-5 minutes while exercising, experienced symptomatic asthma attacks.[7]

Several additional principles to keep in mind when interpreting the chamber studies for standard-setting purposes are:

- Severe asthmatics and children were not studied.
- Due to the small number of subjects included in any one study, the most sensitive people may not have been included.
- In the real world, people breathe sulfur dioxide under different atmospheric conditions than in the laboratory. For instance, chamber studies are usually conduced at room temperature; some asthmatics experience increased response when sulfur dioxide is administered in cold dry air.[8]
- Exposures in the laboratory are to sulfur dioxide alone, not in combination with sulfates and other fine particles as people breathe in real world atmospheres.

On p. 5-12, line 14, the draft ISA states that there is "some evidence of respiratory effects at concentrations as low as 0.2 in the most sensitive asthmatics." We take issue with language about only exceptionally sensitive responders ("the most sensitive asthmatics") in the conclusion. It is likely that the "most sensitive asthmatics" were not subjects in chamber studies. In any event, studies with seven or nine or 20 subjects are highly unlikely to have captured the full range of inter-individual variability and most likely do not reflect the most sensitive subjects.

As noted in the ISA, epidemiological studies find effects at lower concentrations than in the chamber studies. Community health studies provide convincing evidence of adverse

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[5] Sheppard D, Saisho A, Nadel JA, Boushey HA.. Exercise increases sulfur dioxide-induced bronchoconstriction in asthmatic subjects. *Am Rev Respir Dis* 1981; 123: 486-491.
[6] Kirkpatrick MB, Sheppard D, Nadel JA, Boushey HA. Effect of the oronasal breathing route on sulfur dioxide-induced bronchoconstriction in exercising asthmatic subjects. *Am Rev Respir Dis* 1982; 125: 627-631.
[7] Statement of Dean Sheppard, M.D. to Clean Air Scientific Advisory Committee, 4/12/94.
[8] Sheppard D et al. Magnitude of the interaction between the bronchomotor effects of sulfur dioxide and those of dry (cold) air. *Am Rev Resp Dis* 1984; 130: 52-55.

effects of SO_2 exposures at current ambient concentrations, well below the level of the NAAQS.

Additionally, several recent intervention studies that examine the health impact of declining SO_2 concentrations are reviewed in the ISA. These studies demonstrate that real world improvements in public health have resulted from reductions in sulfur dioxide emissions.

There is a new 8-city study of asthmatic children and the effects of SO_2 which should be included in the ISA.[9] The study reported that short-term increases in air pollution at concentrations below the National Ambient Air Quality Standards were associated with adverse respiratory health effects, and found that inner-city children with asthma may be particularly vulnerable to the adverse effects of air pollution. The study focused on 860 children ages 5-12 who lived in low-income areas of Boston, the Bronx, Chicago, Dallas, New York, Seattle and Tucson. The children in the study had moderate to severe asthma, and most were Black or Hispanic. The study involved a larger number of kids and a more comprehensive evaluation of respiratory health effects than earlier studies.

Over two years, the researchers regularly monitored the children's asthma symptoms, breathing function, and school absences, and obtained daily outdoor pollution measurements from the EPA's monitoring network. Every six months, they tested lung function twice-daily over a two-week period. They also asked the children's parents for observations of their children's symptoms.

Results revealed that children had significantly decreased lung function following exposure to higher concentrations of sulfur dioxide, fine particles, and nitrogen dioxide. Higher nitrogen dioxide levels and higher levels of fine particles also were associated with school absences related to asthma, and higher nitrogen dioxide levels were associated with more asthma symptoms.

Studies such as this are important in evaluating the environmental justice implications of alternative standards. Dr. Larson, in comments on the draft methodology for the exposure and risk assessment, pointed out that people living in more industrial neighborhoods have lower incomes, less access to medical care for asthma, yet the greatest exposure to peak concentrations.[10]

The estimates of asthma prevalence in Table 4-2 seem somewhat low relative to estimates produced by the American Lung Association based on data from the National Center for Health Statistics. The estimates in the Figures below most likely reflect an

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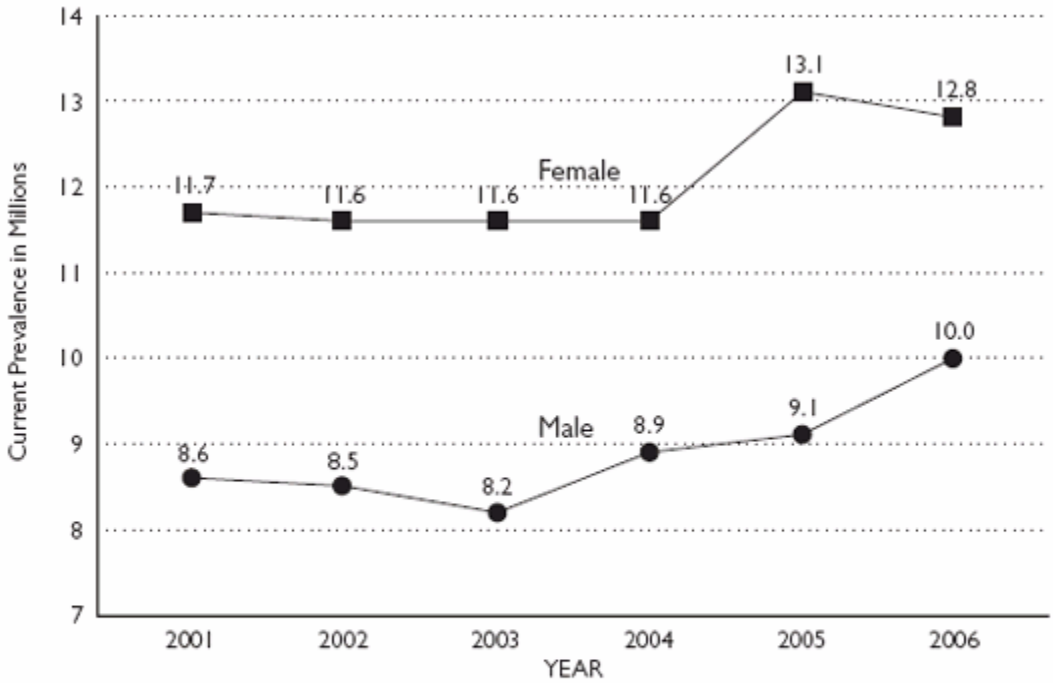
[9] O'Connor GT, Neas L, Vaughn B, Katttan M, Mitchell H, Crain EF, Evans III R, Gruchalla R, Morgan W, Stout J, Adams GK, Lippmann M. Acute respiratory health effects of air pollution on children with asthma in US inner cities. *J Allergy Clin Immunol* 2008; Article in press doi: 10.1016/j.jaci.2008.02.020.

[10] Henderson, Rogene, Chair, Clean Air Scientific Advisory Committee. Clean Air Scientific Advisory Committee's (CASAC) Consultation on EPA's *Sulfur Dioxide Health Assessment Plan: Scope and Methods for Exposure and Risk Assessment* (November 2007 Draft), EPA-CASAC-08-006, January 9, 2008. Attachment B: Compilation of Individual Panel Member Comments.

underestimate of true asthma prevalence, since studies have shown that there are many individuals with undiagnosed asthma.[11]

Figure 1: Asthma—Trend in Current Prevalence by Sex, U.S., 2001–2006[1,*]



Source: Centers for Disease Control and Prevention. National Center for Health Statistics. National Health Interview Survey, 2001–2006. Analysis by the American Lung Association, Research and Program Services Division using SPSS and SUDAAN software.

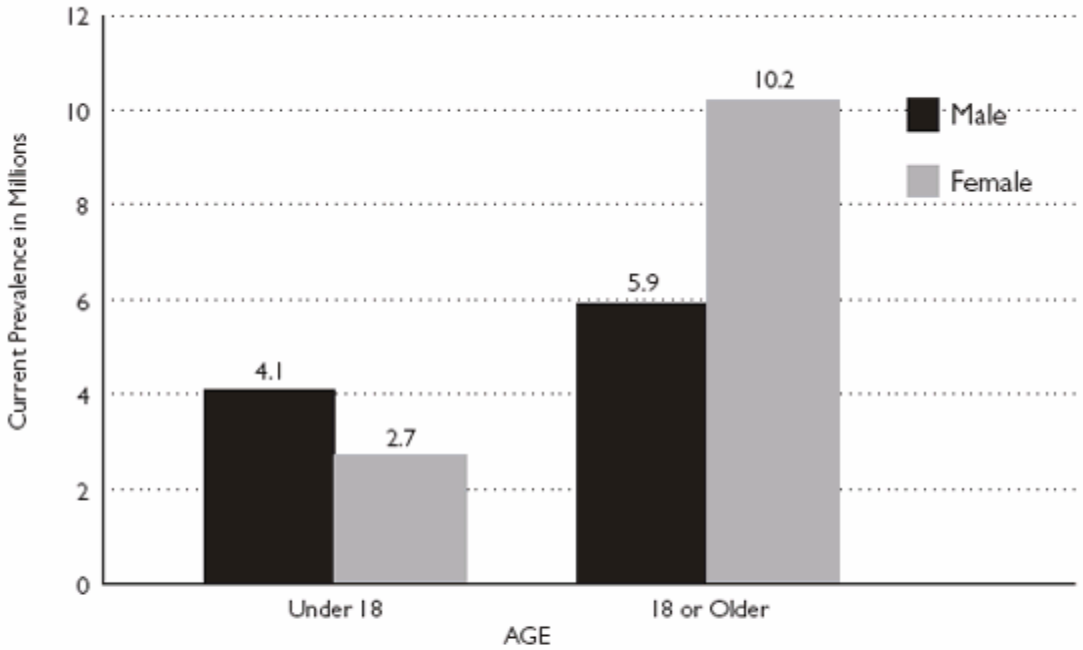
Notes:

1. Current prevalence is defined as answering yes to both "Have you EVER been told by a doctor or other health professional that you had asthma?" and "Do you still have asthma?"

* **Comparisons should only be made between groups and diseases using rates, not number of cases, as these do not take into account differences which may exist in population size or demographics.**

[11] American Lung Association Epidemiology and Statistics Unit. Trends in Asthma Morbidity and Mortality. November 2007.

Figure 2: Asthma—Current Prevalence by Age and Sex, U.S., 2006[1,*]



Source: Centers for Disease Control and Prevention. National Center for Health Statistics. National Health Interview Survey, 2006. Analysis by the American Lung Association, Research and Program Services Division using SPSS and SUDAAN software.

Notes:

1. Current prevalence is defined as answering yes to both "Have you EVER been told by a doctor or other health professional that you had asthma?" and "Do you still have asthma?"

* **Comparisons should only be made between groups and diseases using rates, not number of cases, as these do not take into account differences which may exist in population size or demographics.**

With respect to the first draft Risk and Exposure Assessment (REA), we have a number of concerns.

We find the warnings of Dr. Crawford-Brown on the first draft REA to be prescient:

> "…I worry that it may hide a false sense of confidence in these results, which I take to be quite uncertain. There are many, many assumptions built into the assessment. At the moment, I think of the results as a kind of scenario analysis, and not necessarily an accurate reflection of actual exposures and risks in the U.S. population. The methods may be pushing the current analytic ability too far."[12]

We have reviewed the comments on individual CASAC members on the Draft Scope and Methods for the SO_2 Exposure and Risk Assessment.[13] A number of these concerns have not been adequately accounted for in the current draft.

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[12] 7-23-08 Sox REA - 1st Draft -- Preliminary Individual Comments from Clean Air Scientific Advisory Committee (CASAC) Sulfur Oxides Primary National Ambient Air Quality Standards (NAAQS) Review Panel.

[13] Henderson, Rogene, Chair, Clean Air Scientific Advisory Committee. Clean Air Scientific Advisory Committee's (CASAC) Consultation on EPA's *Sulfur Dioxide Health Assessment Plan: Scope and*

A great many CASAC members supported use of the epidemiological studies reviewed in the ISA in the quantitative assessment of risk. This concern was raised by Drs. Thurston, Gordon, Sheppard, Balmes and Hattis in their comments to EPA. For instance, Dr. Balmes indicated support for the use of emergency department asthma visit data in the risk assessment, and noted that hospitalization data only captures the tip of the iceberg. Dr. Gordon concurred that respiratory hospital admissions and emergency room visits should be included in the quantitative risk assessment. Dr. Sheppard suggested that the criterion that time series study estimates come from the same cities where they are applied is too strict, while raising concerns about the complex nature of the pollution mixture. Yet EPA proposes not to do a quantitative risk assessment based in the epidemiological studies.

A number of panel members including Drs. Speizer, Sheppard, Hattis and Thurston also raised concerns about how exposures of concern are defined. While EPA has lowered the benchmark concentrations somewhat, the benchmark concentrations in the current draft of 0.4-0.6 ppm are inadequate based on studies in ISA. EPA must be concerned with protecting the health of sensitive populations. The Lung Association believes the benchmark exposures of concern should be defined as no greater than 200 ppb, based on the controlled human exposure studies alone, and much lower, when considering the epidemiological studies.

Dr. Hattis presented much information on inter-individual variability and suggested a quantitative approach for assessing human variability which has not been factored into the first draft. Dr. Hattis' comments clearly indicate that effects are observed in people well below the levels of concern proposed by EPA.

In the view of the American Lung Association, the exposure assessment should also consider alternative forms of the standard, and should analyze the impact of changing the from a block form to a rolling average form.

Numerous members of the CASAC panel raised concerns about limitations of the monitoring data to drawing conclusions about exposures. These concerns have not been adequately accounted for in the current draft. Dr. Sheppard indicated that monitor siting features will affect the usefulness of the data. Dr. Siegneur mentioned that the existing monitoring network may not adequately characterize industrial sources or emissions from ships. Dr. Kenski pointed out that monitor proximity to sources such as shipping, industrial coal use coking, metal processing and paper mills needs to be considered and accounted for. Mr. Avol stated that the assertion that current ambient monitoring siting captures peak exposures may not be true for ports and shipping lanes in coastal areas. Dr. Samet indicated that given the limited set of monitors reporting 5-minute concentrations, analyses directed at representativeness are needed.

Methods for Exposure and Risk Assessment (November 2007 Draft), EPA-CASAC-08-006, January 9, 2008. Attachment B: Compilation of Individual Panel Member Comments.

Dr. Larson emphasized that the historical monitoring data may capture a different mix of sources than currently exists, especially with respect to tall stacks vs. near ground level releases.

Dr. Hattis pointed out that realistic future scenarios that project increased SO_2 emitting economic activities of all kinds should be modeled when considering approaches to simulating air quality just meeting current SO_2 standards.

We urge extreme caution in the derivation of peak to mean ratios. The 2:1 peak to mean ration does not seem realistic given the range of emissions scenarios and atmospheric conditions that drive peak concentrations, including start up, shutdown, upsets, malfunctions, downwash, and inversions. Further, the peak to mean ratio may not relevant for non-utility sources. An analysis of short-term monitoring data performed by A.S. L. & Associates concluded that "No relationship could be found between the hourly maximum 5-minute and hourly maximum SO_2 values."[14]

As Dr. Russell points out, the peak to mean ratios are gong to be highly dependent upon how close one is to the source and on the coexistence of many sources in a region. There may be considerable exposure error in measuring average ambient air quality, and even more so in estimating personal exposures. Dr. Sheppard also indicates that using different peak to mean ratio distributions for different features, such as proximity to source, may be called for.

Dr. Sheppard also questions why short-term peaks in the vicinity of SO_2 sources won't be considered. EPA has excluded the results from the Caribou, ID monitor from the analysis, because it is a stationary source-oriented monitor sited within close proximity to a chemical plant. However, this is exactly what is needed to predict peak concentrations. And the people who live nearest these plants are usually those who have the greatest need of our protection.

[14] LeFohn, Allen S. A.S.L. & Associates. Assessing the Potential for the Occurrence of Hourly Maximum 5-Minute Concentrations $\geq$ 0.5 ppm at SO_2 Emission Sources in the United States. Prepared for Clean Air Task Force. March 22, 1999.