Presentation to Southern California Business Coalition on SCAQMD and 2016 AQMP

"**AQMD Must Reassess Its Air Quality Regulations**"

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The South Coast Air Quality Management District (AQMD), one of the most powerful regulatory agencies in the United States, has just proposed tightening its regulations. During the past 40 years it has implemented strong air quality regulations in the 11,000 square-mile South Coast Air Basin (SCAB), which includes the 17 million people who live in the populated areas of Los Angeles, Orange, Riverside, and San Bernardino counties. These increasingly aggressive and costly regulations have impacted all sectors of the economy, from utility power plants, oil refineries, the ports, and all manufacturers to restaurants, dry cleaners, printers, and auto repair shops. While these regulations have improved air quality substantially, they have been excessive and have contributed to the loss of more than half of the manufacturing jobs in Southern California.

The regulation of fine particulate matter ($PM_{2.5}$), ozone (O_3), and nitrogen oxides (NO_x) has been largely justified on a cost-benefit basis by the claim that air pollution causes 5,000 premature deaths per year in the SCAB. This claim relies on the implausible and unproven hypothesis that inhalation over a lifetime of about one teaspoon of $PM_{2.5}$ (particles less than 2.5 microns in diameter) causes premature death. For perspective, inhaling this amount of $PM_{2.5}$ is roughly equivalent to smoking two cigarettes a year, certainly not a lethal dose. Moreover, there is overwhelming epidemiological evidence, including two large 2011 AQMD-funded epidemiological studies, that air pollution does not cause any premature deaths in California. Furthermore, the SCAB has an age-adjusted total death rate that is lower than the death rate in every state except Hawaii. It has a similarly low total cancer death rate.

Regarding exposures, the average ambient levels of 8-hour ozone and 24-hour $PM_{2.5}$ in the SCAB, as measured by AQMD monitors, are below the current Environmental Protection Agency (EPA) National Ambient Air Quality Standards (NAAQS) for ozone and $PM_{2.5}$. Furthermore, the average personal exposures to ozone and $PM_{2.5}$ among SCAB residents are much lower than the ambient levels measured by AQMD monitors. These average personal exposure levels are far below the levels associated with adverse health effects. Air pollutants are now at record low levels and close to natural background levels. The last Stage 3 smog alert was in 1974 and the last Stage 2 smog alert was in 1988. Much of the remaining SCAB pollution comes across the Pacific Ocean from China, which ignores air pollution regulations and which does much of the manufacturing that used to be done here.

Unfortunately, the AQMD staff, led since 1997 by Executive Officer Barry R. Wallerstein, has ignored the extremely positive air quality evidence above. Instead of acting in the best public health and socioeconomic interest of the SCAB residents, AQMD staff has implemented scientifically unjustified regulations in conjunction with the EPA, the California Air Resources Board, and powerful environmental activist groups (like Coalition for Clean Air, American Lung Association, Natural Resources Defense Council, and Sierra Club). The AQMD Board justifiably fired Wallerstein on March 4. There is now an opportunity for the remaining AQMD staff to work with numerous qualified experts like myself in order to reassess the scientific validity of all their regulations. The REgional CLean Air Incentives Market (RECLAIM), the Multiple Air Toxics Exposure Study (MATES), and the 2012 Air Quality Management Plan (AQMP) all need to be reassessed. These reassessments must be made before the 2016 AQMP is finalized and, if they are not made, the AQMD Board should not approve the 2016 AQMP. It is time to stop unjustified regulations in Southern California and to bring manufacturing jobs back.

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Subject: Important Request re November 17 SCAQMD STMPR AG Agenda

November 16, 2015

Joe Cassmassi
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SCAQMD 2016 Air Quality Management Plan (AQMP)
Scientific, Technical & Modeling Peer Review (STMPR) Advisory Group
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Dear Mr. Cassmassi,

I am submitting these written public comments to the STMPR Advisory Group and to the SCAQMD staff members who are presenting at the November 17, 2015 Modeling Session Meeting. I make four basic points that are highly relevant to the preparation of the 2016 AQMP, although none of these points are on the Modeling Session Agenda. I request that all four of my points be addressed by the STMPR Advisory Group and SCAQMD staff as soon as possible.

1) There is overwhelming evidence that the ambient levels of 8-hour ozone and 24-hour fine particulate matter (PM2.5) throughout the South Coast Air Basin (SCAB), as measured by SCAQMD (http://www.aqmd.gov/home/library/air-quality-data-studies), are substantially below the current USEPA NAAQS of 75 ppb for 8-hour ozone and of 35 μg/m³ for 24-hour PM2.5 (http://www3.epa.gov/ttn/naaqs/criteria.html). For instance, on November 15, 2015, the entire SCAB had an ambient 8-hour maximum ozone exposure of 53 ppb. The November 15, 2015 forecast for ambient 24-hour PM2.5 exposure at 38 monitoring stations throughout the SCAB ranged from 10 to 21 μg/m³, with an average of 12.9 μg/m³.

2) There is overwhelming evidence that personal exposure to ozone and PM2.5 among the residents of the SCAB is much lower that the ambient exposure levels cited above. For instance, from June 1995 to May 1996 the average personal exposure of school children was 11.4 ppb in Upland and 13.9 ppb in mountain towns between Crestline and Running Springs (http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1637960/pdf/envhper00304-0121.pdf).

3) There is strong evidence that China is the source of a significant portion of the ozone (http://news.sciencemag.org/earth/2014/09/china-blamed-u-s-ozone) and PM2.5 (http://blogs.wsj.com/chinarealtime/2010/12/01/california-pollution-made-in-china/) in the SCAB and throughout California. Sources of ozone and PM2.5 that are outside of the SCAB need to be addressed in the 2016 AQMP.

4) Public hearings need to be held as soon as possible before the SCAQMD Board regarding the latest report and peer review on "the health impacts of particulate matter air pollution in the South Coast Air Basin," in accordance with California Health and Safety Code Section 40471(b)

(http://www.leginfo.ca.gov/cgi-bin/displaycode?section=hsc&group=40001-41000&file=40460-40471). Such hearings have been mandated every three years since 2001, but they have never been held before the SCAQMD Board Members. There is strong evidence that the health impacts of particulate matter in the SCAB are very minimal, as I have repeatedly stated to SCAQMD during the past decade.

In order to understand the importance of my request, please read recent comments critical of EPA, CARB, and SCAQMD (http://www.scientificintegrityinstitute.org/BC110115091215.pdf). These comments address both ozone and PM2.5 and have been published in the Wall Street Journal, the Los Angeles Daily News, the Bakersfield Californian, and the San Bernardino Sun. They include an op-ed by an SCAQMD Board Member and statements of concern by San Joaquin Valley Air Pollution Control Officer Seyed Sadredin.

Thank you very much for your prompt attention to my request.

Sincerely yours,

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The Harvard Southern California Chronic Ozone Exposure Study: Assessing Ozone Exposure of Grade-School-Age Children in Two Southern California Communities

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The Harvard Southern California Chronic Ozone Exposure Study measured personal exposure to, and indoor and outdoor ozone concentrations of, approximately 200 elementary school children 6–12 years of age for 12 months (June 1995–May 1996). We selected two Southern California communities, Upland and several towns located in the San Bernardino mountains, because certain characteristics of those communities were believed to affect personal exposures. On 6 consecutive days during each study month, participant homes were monitored for indoor and outdoor ozone concentrations, and participating children wore a small passive ozone sampler to measure personal exposure. During each sampling period, the children recorded time–location–activity information in a diary. Ambient ozone concentration data were obtained from air quality monitoring stations in the study areas. We present ozone concentration data for the ozone season (June–September 1995 and May 1996) and the nonozone season (October 1995–April 1996). During the ozone season, outdoor and indoor concentrations and personal exposure averaged 48.2, 11.8, and 18.8 ppb in Upland and 60.1, 21.4, and 25.4 ppb in the mountain towns, respectively. During the nonozone season, outdoor and indoor concentrations and personal exposure averaged 21.1, 3.2, and 6.2 ppb in Upland, and 35.7, 2.8, and 5.7 ppb in the mountain towns, respectively. Personal exposure differed by community and sex, but not by age group. *Key words*: children, chronic, exposure, ozone, personal, sampler, Southern California. *Environ Health Perspect* 108:265–270 (2000). [Online 4 February 2000]
http://ehpnet1.niehs.nih.gov/docs/2000/108p265-270geyh/abstract.html

Almost three decades ago, in response to the Clean Air Act of 1970, the U.S. Environmental Protection Agency promulgated National Ambient Air Quality Standards (NAAQS) (*1*) for six air pollutants: ozone, total suspended particles, nitrogen dioxide, sulfur dioxide, carbon monoxide, and lead. At that time, it was generally believed that only residents of Southern California were at risk for exposure to high ozone concentrations. Now almost every statistical metropolitan area in the United States has reported violations of the 1979 ozone standards of 0.12 ppm for 1 hr during a single year. In 1995, 50 cities across the United States exceeded the air quality standard one or more times (*2*). In 1997, the NAAQS for ozone was changed to an 8-hr integrated value of 0.08 ppm. Compliance will be based on 3 years of monitoring, where the fourth highest 8-hr average in a calendar year cannot exceed 0.08 ppm. Analysis in anticipation of the new standard indicates that even more Americans will be living in areas that exceed healthy levels (*3*).

Chamber studies and other acute exposure studies suggest that short-term effects of ozone on respiratory function and sensory irritation are reversible. However, only a few investigations have studied the chronic effects of ozone exposures over months and years. Using ambient ozone data collected from local monitoring sites, Schwartz et al. (*4*) reported highly significant ozone-associated reductions in lung function for people living in areas where annual ozone concentrations exceeded 40 ppb. Time–series analysis of daily mortality in Los Angeles showed an association with ozone concentration that was significant for both respiratory and cardiovascular-related deaths (*5*). Further, the work of Burnett et al. (*6*) in Ontario, Thurston et al. (*7*) in New York (*7*), and White et al. (*8*) in Atlanta are consistent in showing an association among contemporary measures of ambient ozone and hospital admissions, particularly for asthma.

Although these studies suggest a chronic effect for ozone, they are still limited by a lack of understanding of the relationship between ambient measurements and personal exposures. Several questions about chronic ozone exposure remain unanswered. The relationship between ambient ozone and personal exposures of individuals living in a community has not been adequately addressed, and the interpersonal variability in ozone exposures that are expected because of behavior, housing characteristics, and spatial differences in ozone concentrations has not yet been quantified.

Until recently, collecting personal ozone exposure information has been difficult. Only ultraviolet (UV) photometric or chemiluminescence continuous ozone monitors have been available for ozone concentration measurements and they are too heavy and cumbersome to be carried around by individuals for personal monitoring purposes. Small lightweight passive ozone exposure monitors, however, are now available. These monitors make personal and microenvironmental monitoring feasible (*9–11*). The Harvard passive ozone sampler is one such device that depends on the reaction between ozone and the nitrite ion for ozone concentration measurement (*11*). Over the last several years, short-term personal ozone exposure studies have been carried out by several researchers using this monitor (*12–15*). These studies demonstrated the feasibility of monitoring personal exposure of both children and adults for periods of up to 1 week.

The purpose of this study was to profile personal exposure to ozone over a time period that would provide information for the discussion of potential chronic effects of exposure to ozone. Data obtained from this work will be used to develop a model for estimating annual personal ozone exposure. The study was designed to measure exposure over a time period that would capture seasonal variations in ambient ozone concentrations and in locations which would demonstrate the impact of geographical location on exposure. The Harvard Southern California Chronic Ozone Exposure Study measured personal exposure to, and the indoor and outdoor ozone concentrations of, elementary school children for 12 months (June 1995–May 1996). Two

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Lowering the Ozone Standard Will Not Measurably Improve PUBLIC HEALTH

by Bryan Shaw, Sabine Lange, and Michael Honeycutt

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The Texas Commission on Environmental Quality argues that the thoughtful integration of scientific data does not support the assumption that tightening the ozone standard will result in measurable health benefits.

The Texas Commission on Environmental Quality (TCEQ) strives to protect our state's public health and natural resources consistent with sustainable economic development. In accordance with this mission, the State of Texas alone has spent >$1 billion since 2001 striving to achieve the 1997 0.08 parts per million (ppm) ozone standard. Most of Texas' air quality areas recorded their lowest ozone values ever in 2014. The Houston and Dallas/Fort Worth areas, for example, have seen ozone levels reduced 29% and 21%, respectively, during the past 15 years, while the population has increased 34% and 29%, respectively. We will continue to expend resources to achieve the 2008 75 parts per billion (ppb) ozone standard, which has yet to be fully implemented by the U.S. Environmental Protection Agency (EPA). However, as the concentration of ambient ozone decreases, it becomes exponentially more difficult, and expensive, to attain further reductions. EPA is poised to lower the standard further. While cost cannot be considered in setting the standard, the high cost of further lowering the standard necessitates that this be a sound policy decision and will result in measurable health benefits.

EPA bases its proposal to lower the ozone standard on three key health-related endpoints: premature mortality, respiratory morbidity (i.e., asthma exacerbation, emergency department visits, and hospital admissions), and lung function (i.e., primarily FEV_1 [Forced Expiratory Volume in 1 second, a measure of lung function] decrements). We agree that respiratory effects can occur at the high ozone concentrations that were measured in the 1980s and 1990s. The pertinent question is whether lowering the ozone standard from 75 ppb to 70 or 65 ppb will result in a measurable reduction in these effects. In this short review, we consider some important concerns with EPA's conclusions about the health effects of ambient ozone concentrations. We conclude that EPA has not demonstrated that public health will measurably improve by decreasing the level of the ozone standard.

Ecological Epidemiology Studies, Not Adequate for Setting Standard

EPA relies heavily on ecological epidemiology studies for its assessment of premature mortality and respiratory morbidity. These studies have been very inconsistent in their findings, and flaws, biases, and unusual characteristics of the data have made them difficult to interpret. One unusual and as-yet unexplained characteristic of the epidemiological associations between short-term ozone exposure and mortality is regional heterogeneity. This heterogeneity means that different cities have different associations between short-term exposure to ozone and mortality, and very few of those associations are positive.[1-4]

For example, Smith et al.[1] found that only 7 of the 98 cities investigated showed a statistically significant positive association between 8-hr ozone concentrations and mortality (this is very close to the 5% that would be expected purely by chance). Additionally, there was no association between the estimated effect of ozone on mortality for a city and the concentration of ozone in that city (see Figure 1 on page 28). EPA[5] estimates short-term mortality impacts based on Zanobetti and Schwartz[4] and the Smith et al. study.[1] However, the concentration response functions (CRFs) vary from negative to positive for the same city, depending on study selection, ozone averaging time, model specifications, and ozone season. In fact, most of these estimates are indistinguishable from zero. EPA uses a pooled nationwide estimate for their risk calculations, but the substantial heterogeneity between cities that ranges from positive to null or even negative (i.e., higher ozone concentrations correlated with reduced mortality) makes this nationwide estimate misleading and overestimates ozone risk.

The relationship between long-term ozone exposure and mortality has been investigated in at least 12 epidemiology studies.[6-17] When considering other potential causes of mortality, such as other air pollutants, only one of those studies[15] showed a statistically significant (but very small) effect of ozone on respiratory mortality. Interestingly, the effect only occurred at temperatures above 82 °F. It is known that very warm or very cold temperatures are associated with increased mortality.[18] Paradoxically, the increased mortality was not observed in U.S. regions with the highest ozone concentrations (e.g., Southern California) nor in areas with the highest number of respiratory deaths (e.g., the Northeast and industrial Midwest). Therefore, long-term mortality studies also demonstrate unexplained regional heterogeneity and mostly don't show associations between ozone and long-term mortality.

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Ozone ecological epidemiology studies suffer from severe exposure measurement error.

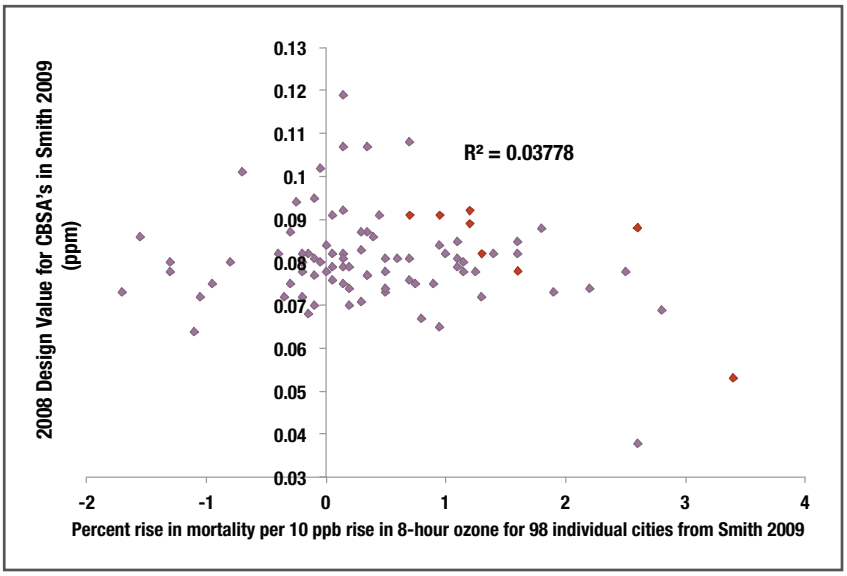

Figure 1. Association between 2008 Ozone Design Values and 8-hr Effect Estimates of Cities.[1]

Notes: Approximate mortality effect estimates (in percent rise per 10-ppb increase in 8-hr ozone) from different cities in Smith et al. (2009)[1] are plotted against the 2008 ozone design values (the 4th highest ambient ozone concentration, averaged over the years 2006–2008) for the matched core-based statistical area (CBSA). Purple points represent cities where mortality was not statistically associated with ambient ozone concentration and red points represent cities where mortality was statistically associated with ambient ozone concentration. The correlation coefficient for the relationship between the mortality effect estimates and the ozone design values (R^2) is given. If ozone and mortality were associated, one would expect an increase in mortality as ozone concentrations (design values) increase.

Ecological epidemiology studies suffer from severe exposure measurement error, because they assume that people are continuously exposed (i.e., 24 hours a day, 7 days a week) to the pollutant concentrations measured at the ambient monitors. In the case of ozone, this error is even more egregious because of the nature of ozone as a pollutant. Ozone is primarily an outdoor pollutant, with ventilation and indoor structures scavenging it and removing it from indoor air. The average American adult, senior citizen, and child will spend only 5.3%, 5.8%, and 7.9% of their time outdoors, respectively,[19] and so they will often not be exposed to ozone.

Studies[20,21] that have investigated ozone personal exposure and compared it to ambient concentrations have found that personal exposure is much lower than ambient exposure (i.e., approximately 10% of the measured ambient level), and that there may not even be a correlation between personal and ambient concentrations.[22,23] Even outdoor workers—whom EPA considers to be an at-risk population—experienced personal ozone concentrations that were only 60% of ambient concentrations.[24] Because of this personal exposure issue, the use of ambient ozone concentrations as a proxy for ozone exposure concentrations grossly overestimates their exposure, and therefore risk. This is particularly true of the short-term mortality data, where the subjects of the study (who are mostly elderly) are within days of death when the ambient concentrations are measured, and so are even less likely to be outdoors.

Altogether, this means that it is highly unlikely that the measured associations between ozone and respiratory mortality/morbidity are plausible, because the ozone exposures of the people in the population are so low. Were *all* of the hundreds of thousands of people in the epidemiology studies outside for 8 hours the day immediately before their deaths? In fact, this concern was raised by the Clean Air Scientific Advisory Committee (CASAC) ozone review panel, EPA's scientific advisors, in a June 5, 2006 letter[25] to EPA: "The Ozone Staff Paper should consider the problem of exposure measurement error in ozone mortality time-series studies. It is known that personal exposure to ozone is not reflected adequately, and sometimes not at all, by ozone concentrations measured at central monitoring sites…Therefore, it seems unlikely that the observed associations between short-term ozone concentrations and daily mortality are due solely to ozone itself." This difference between ambient ozone concentrations and personal exposures is critical for interpreting both epidemiological studies as well as clinical exposure studies.

Lung Function Decrements Unlikely to Be Adverse Below Current Standard

The TCEQ agrees with EPA that the ozone clinical data are best for setting the ozone standard. The American Thoracic Society (ATS) defines adversity as a significant decrease in FEV_1 with a significant increase in symptoms.[26] The ATS notes that FEV_1 decrements can vary by as much as 5% in healthy adults within a single day and by 15% or more from year to year. EPA defines a 10% FEV_1 decrement in a sensitive population as an appropriate adverse effect to protect against because it is mild and reversible. EPA asserts that two clinical studies, by Kim et al.[27] and Schelegle et al.,[28] justify lowering the current 75-ppb standard.

The Kim study reported statistically significant FEV_1 decrements (1.71%) in healthy young adults after 6.6 hours of 60-ppb ozone exposure while exercising heavily for 50 minutes out of every hour. However, these decrements are within normal variation and are not adverse by either the ATS criteria (i.e., because they were not statistically

associated with symptoms), or by EPA's criteria (i.e., because they were less than 10%).

The Schelegle study reported statistically significant FEV_1 decrements—5.34%, 7.23%, and 11.42%, respectively—associated with symptoms in healthy young adults after 6.6 hours exposure to 72-, 81-, and 88-ppb ozone, but not 63-ppb ozone, while exercising heavily for 50 minutes out of every hour. For 72-, 81-, and 88-ppb ozone, this exposure meets the ATS criteria for adversity, but at 72- and 81-ppb, it does not meet EPA's criteria of adversity until 88-ppb, which is above the current standard.

To claim that the lung effects at 60- and 72-ppb from the Kim study and the Schelegle study are adverse, even though the group mean FEV_1 decrements were not adverse, EPA notes that at 60-ppb, 3 of 59 study subjects had FEV_1 decrements greater than 10%, and at 72-ppb 5 of 31 individual participants had FEV_1 decrements greater than 10%. EPA is essentially basing its assertion of adverse effects occurring at concentrations lower than the current standard on these eight individual measurements.

On the other hand, 5 of 31 individual participants had *increases* in FEV_1 after 72-ppb exposure. The remaining participants showed little, if any, change in FEV_1, altogether confirming the known large inter-individual variability in lung function responses. Lung function returned to baseline for all of the participants within 1–4 hours after cessation of exposure.[28] As noted by Folinsbee et al.[29] and McDonnell et al.,[30] the exposure regimens used in the Kim and Schelegle studies simulate work performed during a day of heavy manual labor in outdoor workers. This is an unrealistic exposure scenario for sensitive subpopulations, such as asthmatic children and elderly chronic obstructive pulmonary disease patients. In addition, these lung function decrements would be transient, reversible, would not interfere with normal activity, and would not result in permanent injury or respiratory dysfunction.[31]

Further, EPA evaluated these effects based on exposure concentration, not *dose* (i.e., a function of exposure concentration, time, and ventilation rate). The healthy young study participants exercised vigorously for the majority of their 6.6 hour

EPA has not demonstrated that lowering the ozone standard from 75-ppb to 70–65-ppb will result in a decrease in adverse lung function effects in the population.

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exposure, dramatically increasing their dose, and therefore response, as compared to a resting or moderate exercise ventilation rate for the same exposure concentration. Given these facts, EPA has not demonstrated that lowering the ozone standard from 75-ppb to 70–65-ppb will result in a decrease in *adverse* lung function effects in the population.

Evidence for Ozone Exacerbation of Asthma Is Insufficient

EPA investigated the epidemiology studies that show effects of ambient ozone concentrations on asthma health outcomes. Keeping in mind that these studies suffer from the same exposure measurement errors as the mortality studies, EPA showed that 21 of the 33 reported associations between ozone and asthma symptoms were not statistically significant, and those that were significant were not consistent with one another.[19] This result is quantified in the regulatory impact analysis,[32] where EPA shows that there is no statistically significant decrease in asthma exacerbations with a decreasing level of the ozone standard. EPA also states that emergency department visits and hospital admissions are robust to co-pollutant confounders, but does not mention investigation of confounding by pollen, which is a known, strong inducer of asthma.[33,34] Also, confounding by race, ethnicity, and household poverty are important considerations, as was shown in a recent study demonstrating that asthma incidence and morbidity is not more associated with urban (more polluted) areas, but rather with ethnicity and poverty.[35] Therefore, EPA should not have drawn the conclusion that ozone enhances asthma morbidity at ambient concentrations based on these data.

In conclusion, the TCEQ thinks the thoughtful integration of the scientific data does not support the assumption that lowering the ozone standard from 75 ppb to 70–65 ppb will result in measurable health benefits. The ecological epidemiology studies are critically flawed due to severe exposure misclassification because personal exposure to ozone is approximately 10% of ambient levels, dramatically reducing the ozone dose people actually receive. The clinical studies do not indicate anything beyond mild, reversible effects below 75 ppb. It is biologically implausible that 8-hr ambient ozone concentrations below 75 ppb would cause mortality when they do not cause mild effects. em

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https://energycommerce.house.gov/hearings-and-votes/hearings/hr-4775-ozone-standards-implementation-act-2016

U.S. House Energy and Commerce Committee

H.R. 4775, Ozone Standards Implementation Act of 2016

Thursday, April 14, 2016 - 10:15am

Location:

2322 Rayburn

H.R. 4775, Ozone Standards Implementation Act of 2016

Subcommittees: Energy and Power (114th Congress)

Video of H.R. 4775, Ozone Standards Implementation Act of 2016

Witnesses

Mr. Misael Cabrera
Director, Arizona Department of Environmental Quality

- Witness Statement
- Witness Invitation
- Truth in Testimony and CV

Mr. Alan Matheson
Executive Director, Utah Department of Environmental Quality

- Witness Statement
- Witness Invitation
- Truth in Testimony and CV

Mr. Ali Mirzakhalili
Director, Division of Air Quality, Delaware Department of Natural Resources and Environmental Control

- Witness Statement
- Witness Invitation
- Questions for the Record
- Response to Questions for the Record
- Truth in Testimony and CV

Mr. Seyed Sadredin
Executive Director/Air Pollution Control Officer, San Joaquin Valley Air Pollution Control District

- Witness Statement
- Witness Invitation
- Truth in Testimony and CV

Dr. Bryan W. Shaw
Chairman, Texas Commission of Environmental Quality

- Witness Statement
- Witness Invitation
- Questions for the Record
- Response to Questions for the Record
- Truth in Testimony and CV

Text of Legislation

- H.R. 4775, Ozone Standards Implementation Act of 2016

Documents

- Hearing Notice
- Background Memo
- Document for the Record - Statement for the Record from EPA Acting Assistant Administrator McCabe
- Opening Statement of Energy and Commerce Committee Chairman Upton
- Opening Statement of Energy and Power Subcommittee Chairman Whitfield
- Document for the Record - Chairman Whitfield - Letters of Support from Members of the San Joaquin Valley Air District Environmental Justice Advisory Group
- Document for the Record - Chairman Whitfield - IECA Letter of Support
- Document for the Record - Chairman Whitfield - ACC Letter of Support
- Document for the Record - Chairman Whitfield - "2015 Ozone Standard Exceedances in National Parks"
- Document for the Record - Chairman Whitfield - ECOS Resolution
- Document for the Record - Chairman Whitfield - AAPCA Survey
- Opening Statement of Energy and Commerce Committee Ranking Member Pallone
- Hearing Attendance
- Document for the Record - Rep. Rush - Letter for the Record from Health Groups
- Document for the Record - Rep. Rush - Letter for the Record from Advocacy Organizations
- Preliminary Transcript
- Opening Statement of Majority Whip Scalise

http://www.epw.senate.gov/public/index.cfm/2016/6/examining-pathways-towards-compliance-of-the-national-ambient-air-quality-standard-for-ground-level-ozone-legislative-hearing-on-s-2882-and-s-2072

US Senate Committee on Environment and Public Works

Hearings

Examining Pathways Towards Compliance of the National Ambient Air Quality Standard for Ground-Level Ozone: Legislative Hearing on S. 2882 and S.2072

Wednesday June 22, 2016 02:30 PM EST

406 Dirksen

The Senate Committee on Environment and Public Works Subcommittee on Clean Air and Nuclear Safety will hold a hearing entitled, "Examining Pathways Towards Compliance of the National Ambient Air Quality Standard for Ground-Level Ozone: Legislative Hearing on S. 2882 and S.2072."

WEDNESDAY June 22, 2016

2:30 PM

Room 406 of the Dirksen Senate Office Building

WITNESSES:

Andrew Chesley, Executive Director
San Joaquin Council of Governments, California

Kurt Karperos, P.E., Deputy Executive Officer
California Air Resources Board

Glenn Hamer, President and CEO
Arizona Chamber of Commerce and Industry

Mark Raymond, Commissioner and Chair, Uintah County, Utah

Dr. Mary B. Rice MD, Vice-Chair, ATS Environmental Health Policy Committee American Thoracic Society, Assistant Professor of Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School BIDMC

By Order of the Chairman

Shelley Moore Capito

Archived Webcast

Majority Statement

- Sen. Jim Inhofe

Minority Statement

- Sen. Tom Carper

  Senator Tom Carper Hearing Statement for the Record.pdf (15.8 KBs)

Panel 1

Glenn Hamer
President and CEO
Arizona Chamber of Commerce and Industry
Hamer Testimony.pdf (1.3 MBs)

Mark Raymond
Uintah County Commissioner and Chair
Uintah County, Utah
Raymond Testimony.pdf (264.7 KBs)

Andrew Chesley
Executive Director
San Joaquin Council of Governments
Chesley Testimony.pdf (563.6 KBs)

Mary B Rice MD
Vice-Chair
ATS Environmental Health Policy Committee American Thoracic Society Assistant Professor of Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School BIDMC
Rice Testimony.pdf (118.5 KBs)

Kurt Karperis P.E.
Deputy Executive Officer
California Air Resources Board
Karperos Testimony.pdf (104.6 KBs)

Related Files

- SPW 062216.pdf (177.8 KBs)

Permalink: http://www.epw.senate.gov/public/index.cfm/2016/6/examining-pathways-towards-compliance-of-the-national-ambient-air-quality-standard-for-ground-level-ozone-legislative-hearing-on-s-2882-and-s-2072

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https://www.govtrack.us/congress/bills/114/hr4775

govtrack.us

1. Congress
2. Bills
3. H.R. 4775

H.R. 4775: Ozone Standards Implementation Act of 2016

Introduced: **Mar 17, 2016**

Status: **Passed House** on **Jun 8, 2016**

This bill passed in the House on June 8, 2016 and goes to the Senate next for consideration.

Sponsor:

Pete Olson Representative for Texas's 22nd congressional district Republican

Text:

IIB

114TH CONGRESS
2D SESSION

H. R. 4775

IN THE SENATE OF THE UNITED STATES

JUNE 9, 2016

Received; read twice and referred to the Committee on Environment and Public Works

AN ACT

To facilitate efficient State implementation of ground-level ozone standards, and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled,

SECTION 1. SHORT TITLE.

Read Text »

Last Updated: Jun 9, 2016

Length: 14 pages

Prognosis: 24% chance of being enacted (details)

Track this bill

Call Congress

About the bill

Full Title To facilitate efficient State implementation of ground-level ozone standards, and for other purposes. *The bill's title was written by its sponsor.*

Read CRS Summary >

History

Mar 17, 016	Introduced This is the first step in the legislative process. Read Text »
May 18, 2016	Reported by Committee A committee has issued a report to the full chamber recommending that the bill be considered further. Only about 1 in 4 bills are reported out of committee. Read Text » See Changes »
Jun 8, 2016	Passed House The bill was passed in a vote in the House. It goes to the Senate next. View Vote » Read Text » See Changes »
	Passed Senate
	Signed by the President

A bill must be passed by both the House and Senate in identical form and then be signed by the President to become law.

Details

Cosponsors 43 cosponsors (40R, 3D) (show)

Committee Assignments

House Energy and Commerce ↪ Energy and Power

Senate Environment and Public Works

The committee chair determines whether a bill will move past the committee stage.

Votes

House Vote on Passage

Jun 8, 2016 5:14 p.m.

Passed 234/177

Show 6 additional vote(s)...

Subject Areas

Environmental Protection

Air quality

Atmospheric science and weather

Congressional oversight

show 7 more

Related Bills

Legislative action may be ocurring on one of these bills in lieu of or in parallel to action on this bill.

S. 2882 (Related)

Ozone Standards Implementation Act of 2016

Referred to Committee
Last Action: Apr 28, 2016

H.Res. 767 (Related)

Providing for consideration of the bill (H.R. 4775) to facilitate efficient State implementation of ...

Agreed To (Simple Resolution)
Jun 8, 2016

show 1 more

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