American Lung Association ● American Public Health Association
Amercan Thoracic Society ● Asthma and Allergy Foundation of America
Trust for America's Health

November 30, 2011

The Honorable Lisa P. Jackson
Administrator
U.S. Environmental Protection Agency
EPA Docket Center
Air and Radiation Docket, Mail Code 28221T
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Re: Oil and Natural Gas Sector: Reviews of New Source Performance Standards and National Emissions Standards for Hazardous Air Pollutants. Docket ID No. EPA-HQ-OAR-2010-0505

Dear Administrator Jackson:

On behalf of our nation's medical and public health groups, we urge the U.S. Environmental Protection Agency to adopt the strongest possible standards to reduce harmful emissions from the production wells, processing plants, transmission pipelines, and storage units within the oil and natural gas industry. As public health groups and medical societies, we are keenly aware of the harmful health effects of these air pollutants. Research has shown that these pollutants can harm the circulatory, respiratory, nervous, and other essential and vital life systems. These emissions can even cause cancer, developmental disorders, and premature death. The cleanup of air pollution from oil and natural gas wells is necessary for the protection of public health, appropriate for the EPA to undertake, and of growing importance. We applaud EPA's efforts to respond to this growing source of air pollution and appreciate the opportunity to provide comments.

Air Pollution from Oil and Natural Gas Sectors

We agree with the strong evidence the EPA provides to support their decision that action under Sections 111 and 112 of the Clean Air Act is both appropriate and necessary to protect public health. The Clean Air Act requires that EPA review and revise standards to see if they adequately protect public health from new sources of pollution and from hazardous air pollutants. The expanding oil and natural gas production in the United States represents sources of such emissions that must be addressed under the law.

Emissions can occur during the extraction, production, processing, flaring, transportation and distribution of oil and natural gas. Those emissions can be vented to the atmosphere (intentionally or unintentionally) and impact air quality and must be adequately addressed under the proposed rules. Additionally, the large engines used in drilling and production processes burn fossil fuels and produce emissions. Although those impacts are not covered under these proposed rules, they can particularly add to the air pollution burden affecting local communities.

The rapid development of high volume/horizontal drilling in conjunction with hydraulic fracturing ("fracking") technologies to recover natural gas from shale formations has driven the expansion of new sources, as it rapidly expanded the nation's supply of natural gas resources. As recently as 2001, shale gas made up less than two percent of U.S. production of natural gas. It tripled to six percent in 2006 and doubled again to 12 percent in 2008. As of July 2011, estimates place shale gas at 29 percent of natural gas produced outside of Alaska and Hawaii. (SEAB, 2011). Natural gas production is also expanding into new areas of the country, including highly populated areas, such as western Pennsylvania. In 2009, Pennsylvania had only one percent of natural gas development but now has one of the largest expansions of shale gas production underway with 1,650 wells reportedly producing as of June 30, 2011 (SEAB, 2011; Olson, 2011). EPA's own estimates project shale gas to make up 45 percent of all natural gas production by 2035 (EPA, 2011e).

The expansion of oil and gas production has led to astonishing and unhealthy concentrations of ozone in unexpected areas. In Sublette County, Wyoming, 8-hour ozone concentrations in February 2008 reached as high as 122 ppb (EPA Air Data, 2008). Sublette County currently has a design value of 78 ppb for 2008-2010, well in excess of the 2008 national ambient air quality standards (EPA, 2011d).

Overall Comments

We recommend EPA include both new and existing sources of air pollution related to oil and natural gas production, not just new ones. EPA should require cleanup of all existing equipment—especially compressors, pneumatic controllers and valves—throughout the process. Stronger limits on emissions are in place in Wyoming, California, and Colorado and should be incorporated into EPA's final rule. For example, Wyoming's current rule requires 98 percent control of volatile organic compounds (VOCs) and hazardous air pollutants from storage vessels, not 95 percent as the EPA rule proposes. Also, some local air quality control districts in California require that well cellars, sumps, and pools of oil be covered to prevent VOC emissions.

We applaud EPA's efforts to bring more modern systems into the measures and monitoring, including the use of better monitoring tools to detect leaks or fugitive emissions. We urge EPA

to incorporate these requirements into the rules for existing facilities, as Colorado is using in ozone nonattainment areas. EPA can help the producers save valuable energy that is being lost and help reduce methane emissions. However, we do urge EPA to target methane reductions specifically and not just including them as a co-benefit of the VOC reductions.

Malfunctions. We appreciate EPA's proposal to apply the Section 111 and 112 provisions at all times, including startup and shutdown. However, we urge EPA to review the exposure estimates to recognize that significant air emissions are likely to result from malfunctioning equipment as well. EPA needs to acknowledge that malfunctions are likely events which must be factored into the exposure estimates. EPA's language implies that these are rare events, but such is not the case, as recent reports have shown. For example, the Louisiana Bucket Brigade released a report last week that calculated one accidental release per day in Louisiana for the past six years. (Louisiana Bucket Brigade, 2011) The historical industry evidence should provide more than adequate information on likely malfunctions and exposures.

Unfortunately, the proposed "affirmative defense" option creates a loophole that will not likely reduce the risk of malfunctions, providing facilities with a way to avoid penalties that could provide incentives to reduce malfunctions. The EPA needs to close this loophole as they develop standards under Section 111 and 112 of the Clean Air Act to limit emissions during all phases of operation, including when equipment fails to work properly.

Major source determination. We urge that the guidance clearly require that the threshold of a "major source" be based on evaluations of both the emissions in a single source within the facility and in a facility-wide assessment of all similar sources. If more than one source would not meet the individual requirements, then the total emissions from those sources in a facility should be evaluated to determine if they collectively meet the threshold. We are concerned about the possibility that a facility that has multiple "non-major" sources all of which fall just below the threshold for classification as a major source could avoid having to comply with the requirements. While we support looking at the emissions from each source in a facility, we also do not want to have multiple smaller sources ignored because they fit under the threshold.

Water ponds. We also support measures to require permits and controls on "produced" water ponds used in the extraction process. These ponds can emit large amounts of volatile organics. A number of air quality districts in California have long required controls on volatile emissions from produced water ponds. For example, the Santa Barbara County, California Air Pollution Control District requires controls for emissions from covered ponds (SBC APCD, 1994).

Risks to populations likely greater than estimated in proposal

As oil and gas extraction have expanded, emissions from those sectors have increased. Emitted into the air during oil production and natural gas production (both conventional and hydraulically

fractured) are sulfur dioxide and volatile organic compounds (VOCs), which includes gases considered "air toxics" and methane. Using 2005 data, EPA estimates that hydraulically fractured wells are the source of 500,000 tons of VOC emissions each year. EPA also acknowledges that the oil and natural gas sector as a whole emits significant amounts of air pollutants that seriously threaten human health. In EPA's proposed rule, they report that the industry emits 2.2 million tons of VOCs, 130,000 tons of air toxics, and 16 million tons of greenhouse gases (methane) each year (40% of all methane emission in the U.S.). The industry is one of the largest sources of VOCs and sulfur dioxide emissions in the United States (EPA, 2011a; 2011b).

We applaud the EPA's intent to incorporate an "ample margin of safety" approach to determining risk exposure to the air toxics. That approach recognizes that the scientific knowledge can never be exact and the intent of the Clean Air Act is to protect health from such dangerous emissions. However, EPA's tools incorporated in the review have some significant limitations that need addressing in the final rule.

First, the inventory of emissions used to develop this assessment is woefully out of date. The EPA used the 2005 version of the National Emissions Inventory (NEI), a six-year-old inventory which does not cover all toxics that are detected at oil and natural gas wells and does not account for the recent rapid growth in hydraulic fracturing in the industry. In the final rule, EPA should at least use a much more up-to-date estimate of the emissions, as even the 2008 inventory—newer than the one referenced in this rule—misses the current and projected reality. A better choice would be to use actual measured emissions data, since EPA has the authority under Section 114 of the Clean Air Act to request it.

The problems with these data create other limitations in understanding and assessing the potential risk as well, especially risks to children, older adults, and in the environmental justice analysis. EPA has worked hard to assess the potential for disproportionate harm on different demographic groups who may be exposed to these hazardous pollutants. However, EPA had to rely on data drawn from the 2000 Census, now 11 years old, which is missing the significant changes in the composition, distribution, and economic status that have occurred since then. With expanded well production comes expanded exposure to different and changing populations. Because of this, EPA needs to incorporate a wider margin for assessing risk provides a more appropriate basis for evaluating the threats.

The discussion of risk to children needs to include a better assessment of the current evidence about the risk to their health. Children face quite different risks from air pollutants than adults. The lungs and their alveoli are not fully grown until children become adults (Dietert et al., 2000). Biological defenses that help adults fight off infections are still developing in young bodies (WHO, 2005). Furthermore, children don't behave like adults, and their behavior also affects

their vulnerability. They are outside for longer periods and are usually more active when outdoors. Consequently, they inhale more polluted outdoor air than adults typically do (AAP, 2004).

Toxic substances may put children more at risk than adults. For example, the California Environmental Protection Agency explored improved methodologies to determine susceptibility to carcinogens *in utero* and childhood after finding in 2001 that the existing approaches did not adequately reflect the risks to children. Their subsequent research found that the children generally display greater sensitivity to environmental carcinogens than did adults (CEPA, 2009). They recommended a more protective adjustment to risk assessments to reflect that greater risk. We urge EPA to examine and use the most current research on these and other cumulative impacts for children and adults in developing the "ample margin of safety."

Communities of color and poorer people also appear to face higher risk, underscoring the need to properly assess this margin of safety. Research indicates that minorities live in greater concentrations both in areas that do not meet federal air quality standards and in areas with above average numbers of air-polluting facilities (NAS, 1999). Both African Americans and Hispanics have been found to be more likely than Caucasians to live in areas with high levels of air toxics (Morello-Frosch and Lopez, 2006).

- A study in Maryland found that the risk of cancer related to air toxics was greatest in areas with the largest African American population proportions and lowest among those with the smallest African American population proportions. In addition, the estimated cancer risk decreased for every 10 percent increase in the percentage of Caucasians living in an area. Having a low income also increased the risk among African Americans more so than among Caucasians (Apelberg BJ et al., 2005).

- In Houston, Houston is home to one of the world's largest petrochemical complexes, researchers found that the risk of cancer in an area increased along with the proportion of the population that was Hispanic and as measures of social disadvantage increased (Linder et al., 2008).

- Socioeconomic position has been more consistently associated with greater harm from air pollution. Recent studies show evidence of that link. Low socioeconomic status consistently increased the risk of premature death from fine particle pollution among 13.2 million Medicare recipients studied in the largest examination of mortality associated with particulate matter levels nationwide (Zeger et al., 2008).

Risks from specific emissions and pollutants

Not only is there clear evidence of harm directly from emissions of sulfur dioxide, nitrogen oxide and VOCs, but from ozone and fine particulate matter ($PM_{2.5}$) as well. VOCs, nitrogen oxides

and sulfur dioxide are precursors to ozone and fine particulates, also pose a significant threat to human health. These pollutants can cause or increase risk of cardiovascular, respiratory, and other acute and chronic systemic damage, and may increase risk of cancer. The standards will help reduce ozone and fine particulate matter levels in areas where oil and gas production occurs and downwind. The air toxics standards for oil and natural gas wells will also reduce hazardous air pollutants, including the risk of benzene and formaldehyde, both carcinogens, in the oil and gas production process and for transmission and storage. The discussion below summarizes the evidence that these pollutants pose serious threats to health and must be reduced.

Sulfur Dioxide (SO_2)

Sulfur dioxide (SO_2) a gaseous air pollutant composed of sulfur and oxygen. Sulfur dioxide is emitted during the production and processing operations for some sulfur-containing fuels. The sulfur dioxide standard will help reduce this harmful pollutant, long recognized for its harm to health. Sulfur dioxide causes a range of harmful effects on the lungs, including wheezing, coughing, shortness of breath and chest tightness, and other problems, especially during exercise or physical activity. Continued exposure at high levels aggravates asthma, increases respiratory symptoms, and reduces the ability of the lungs to function. Short exposures to peak levels of SO_2 in the air can make it difficult for people with asthma to breathe when they are active outdoors. Rapid breathing during exercise helps SO_2 reach the lower respiratory tract, as does breathing through the mouth. SO_2 pollution increases the risk of hospital admissions or emergency room visits, especially among children, older adults, and people with asthma (EPA, 2009a). In addition, sulfur dioxide poses another threat to human health by reacting in the air to form sulfates (SO_4) which exist as aerosolized fine particulate matter ($PM_{2.5}$), another harmful air pollutant discussed below (EPA, 2009b).

Nitrogen Oxides (NO_x)

Nitrogen oxides (NO_x) are a class of gaseous air pollutants composed of nitrogen and oxygen. NOx is emitted during the combustion of natural gas in engines, turbines, heaters, and boilers during production and processing operations for oil and gas wells. NOx is also emitted during pit flaring of VOC emissions from well completions. The pollutant itself can inflame the airways and reduce lung function, worsened cough and wheezing, increase asthma attacks and hospital visits; and increase risk of respiratory infection (EPA, 2008). EPA's own review of the science found that exposure to NOx can increase the risk of hospitalization by up to 20 percent (EPA, 2008). Nitrogen oxides are also precursors to nitrates (NO_3) which also are recognized as aerosolized fine particulate matter ($PM_{2.5}$) and discussed below. (EPA 2009b)

Fine Particulate Matter ($PM_{2.5}$)

Reductions in sulfur dioxide and nitrogen oxides through the final oil and natural gas wells standards would provide a crucial collateral benefit: reduction in secondary fine particulate matter. Sulfates formed from sulfur dioxide comprise the majority of fine particulate matter in

much of the United States, especially in the summer months. Nitrates from nitrogen oxides are also a major source of fine particulate matter in the fall, winter and spring (EPA, 2011c). $PM_{2.5}$ is made up of microscopic particles, including aerosols, that can bypass the body's natural defenses and lodge deep within the lungs (EPA, 2004, 2009b). Fine particles elevate risk of heart attacks and strokes (Dominici et al., 2002; Hong et al., 2002; Franklin et al., 2007; D'Ippoliti et al., 2003; Miller et al., 2007); stunt lung function and development (Gauderman et al., 2002; Gauderman et al., 2004); inflame and damage lung tissue and airways (Ghio et al., 2000; Churg et al., 2003); increase hospital visits for respiratory and cardiovascular problems (Dominici et al., 2006; Tsai et al., 2003); and aggravate asthma attacks (Lin M et al., 2002; Norris et al., 1999; Tolbert et al., 2000; Slaughter et al., 2003; Lin et al., 2002b). The evidence shows that $PM_{2.5}$ causes cardiovascular harm and is likely to cause respiratory harm. More seriously, $PM_{2.5}$ can cause premature death from lung cancer and cardiovascular effects and is likely to cause death from respiratory effects as well (Pope et al., 2002; Pope et al., 2004).

The most vulnerable populations, including children, teens, senior citizens, people with low incomes and people with chronic lung disease, such as asthma, chronic bronchitis, and emphysema, are at risk of being sickened by fine particulate matter. People with diabetes, heart disease, high blood pressure, coronary artery disease, and congestive heart failure, are also at risk (EPA, 2004, 2009b). The evidence suggests that long-term exposure to $PM_{2.5}$causes reproductive and developmental effects as well as cancer, mutagenicity and genotoxicity (EPA, 2009b).

Volatile Organic Hazardous Air Pollutants

Volatile organic hazardous air pollutants are specific toxic gases that react easily with other gases and particles. These take in a host of carcinogens and other toxins. According to the EPA's Regulatory Impact Assessment, six organic hazardous air pollutants dominate the mass from oil and natural gas wells and can most harm human health: benzene, toluene, carbonyl sulfide, ethylbenzene, mixed xylenes, and n-hexane. Other major hazardous air pollutants from wells include formaldehyde, ethylene glycol, methanol, and 2,2,4-trimethylpentane.

Many of these toxic air pollutants can cause cancer, but they can also irritate the eyes, skin, and respiratory tract, impair lung function, and affect vital organs. Benzene and formaldehyde are recognized as known human carcinogens, while ethylbenzene is considered a probable carcinogen (HHS, 2011). Long-term exposures to benzene can cause leukemia, a blood cancer, and other blood disorders such as anemia and depressed lymphocyte count in blood. Exposure to formaldehyde can also cause chronic bronchitis and nasal epithelial lesions. A recent review of the research found evidence that formaldehyde may increase the risk of asthma, particularly in the young (McGwin et al., 2010). Non-cancer effects associated with exposure to these organics range from irritation of the skin, eyes, nose, throat, and respiratory tract, and dizziness, nausea, and vomiting. These compounds can also cause difficulty in breathing, impaired lung function and respiratory symptoms, damage to the liver and kidneys, and stomach discomfort. They may

also cause developmental disorders, adverse effects to the nervous system, impairment of memory and neurological function, and slow response to visual stimuli. These pollutants can also affect hearing, speech, vision, and motor coordination (ATSDR, 1999a, 1999b, 2000, 2007a, 2007b, 2010).

Volatile Organic Compounds as Precursors to Ozone (O_3)

One of the most crucial aspects of the rule is the limit it sets on the amount of volatile organic compounds (VOCs) that are emitted by oil and natural gas wells. Cleaning up VOCs with these standards is critical to protecting human health. As noted above, many VOCs are hazardous air pollutants. However, VOCs are also precursors to the secondary formation of ozone when they react with nitrogen oxides (NOx) in the presence of sunlight. By limiting emissions of VOCs, the proposed oil and natural gas standard will indirectly reduce the amount of secondary ozone formed in the air, human exposure to ozone, and the incidence of ozone-related health effects.

Ozone is a colorless, odorless gas that reacts chemically ("oxidizes") with internal body tissues, such as those in the lung. Some have described the inflammation that ozone causes in the airways as similar to a "sunburn" on the lungs. It acts as a powerful respiratory irritant at the levels frequently found across the nation during the summer months. Breathing ozone may lead to shortness of breath and chest pain (Horstman et al., 1990; McDonnell et al., 1999), wheezing and coughing (Triche et al., 2006); inflammation of the lung lining (Mudway and Kelly, 2004); increased risk of asthma attacks (Mortimer et al., 2002), increased susceptibility to respiratory infections (Hollingsworth et al., 2007), and need for medical treatment and for hospitalization for people with lung diseases, such as asthma or chronic obstructive pulmonary disease (COPD) (EPA, 2006; Lin et al., 2008); and premature death (Bell et al., 2005; Levy et al., 2005; Ito et al., 2005; NRC, 2008).

The most vulnerable individuals, including children, teens, senior citizens, people who exercise or work outdoors, and people with chronic lung diseases like asthma, COPD, and emphysema, are most in danger of being sickened by ozone (Peters, 1997; Delfino et al., 1998; Gauderman et al., 2002; Lin S et al., 2002; Gent et al., 2003,; Desqueyroux et al., 2003; Lin et al., 2008). So-called "responders," otherwise healthy individuals who experience health effects at lower levels of exposure than the average person, are also susceptible to ozone (Devlin, 1993). Children who grow up in areas of high ozone pollution may never develop their full lung capacity as adults. That can put them at greater risk of lung disease throughout their lives (Kunzli et al., 1997).

Methane (CH_4)

Although the health effects of methane have been more commonly addressed as a result of methane's role as a precursor to ozone or as a greenhouse gas, methane itself also poses a serious health risk. Methane is also a VOC, but is an odorless gas that can burn or explode at concentrations of 5 percent to 15 percent by volume of air (ATSDR, 2001). At high

concentrations, methane can also displace enough oxygen to cause a deficiency in the air, leading to unconsciousness and even death by suffocation (NIOSH, 1985). Methane is a major concern especially from an occupational safety and health standpoint for workers at natural gas wells who would be exposed to high volumes of the gas during the hydraulic fracturing process.

In addition to its direct health effects, methane poses another risk as a powerful and potent greenhouse gas that is a cause of climate change. Although the standards do not directly reduce methane, the reductions in VOCs will also cut methane. Greenhouse gases pose multiple long-term threats to human health. Warmer temperatures and changing climates can make ozone levels higher than they would be otherwise. Wildfires and dry soil dust resulting from warmer temperatures can increase concentrations of particle pollution, which can be inhaled deep into the lungs. Pollen and allergens may increase (IPCC, 2007; WHO, 2003).

Although the standards do not directly reduce methane, the reductions in VOCs will also cut methane. The EPA has identified the oil and gas industry as the "single largest contributor to United States anthropogenic methane emissions" (EPA, 2011f). As recently recommended by the U.S. Department of Energy's Advisory Board on Shale Gas Production, the EPA should include methane in the oil and gas standards and directly reduce methane emissions on both new and existing pollution sources (SEAB, 2011). The EPA should require the installation of effective and readily available control technologies on both new and existing equipment.

Preventing the emission of these air pollutants will protect human health

The proposed standards on the oil and gas industry are a good step towards protecting the health of Americans. The VOCs, air toxics, and sulfur dioxide reductions in the proposed rule are expected to improve outdoor air quality, reduce cancer risk from air toxics emissions, and reduce health effects associated with exposure to ground-level smog and fine particle pollution. People most at risk of harm from breathing these air pollutants who are depending on the EPA to take action to clean up air pollution from the oil and natural gas industry include: infants, children and teenagers; older adults; pregnant women; people with asthma and other lung diseases; people with cardiovascular disease; diabetics; people with low incomes; and healthy adults who work or exercise outdoors.

People with chronic diseases, including cardiovascular diseases, respiratory diseases and diabetes, face higher risk regardless of age. Their diseases make them at much higher risk for harm. Current estimates include these groups:

- Asthma - 24.6 million people, including 7.0 million under age 18 (American Lung Association, 2011)
- Cardiovascular diseases – 82.6 million people (Roger et al., 2011)
- Diabetes – 25.8 million people (CDC, 2011)

- Chronic Obstructive Pulmonary Disease (COPD)—12.1 million adults age 18 and older (American Lung Association, 2010)

As adults age, their body's physiological process decline naturally, placing even healthy older adults at risk from airborne pollutants. In addition, many older adults also have one or more chronic diseases that increase their susceptibility (EPA, 2009).

Particularly at risk are people with low incomes and some racial and ethnic groups. Scientists have speculated that there are three broad reasons why disparities may exist. First, pollution sources may be located near disadvantaged communities, increasing exposure to harmful pollutants. Second, low social position may make some groups more susceptible to health threats because of factors related to their disadvantage. Lack of access to health care, grocery stores and good jobs, poorer job opportunities, dirtier workplaces or higher traffic exposure are among the factors that could handicap groups and increase the risk of harm. Finally, existing health conditions, behaviors, or traits may predispose some groups to greater risk. For example, diabetics are among the groups most at risk from air pollutants, and the elderly, Blacks/African-Americans, Mexican-Americans and people living near a central city have higher incidence of diabetes (O'Neill et al., 2003).

Conclusion

According to the EPA estimates, these new standards will result in the following emissions reductions each year (EPA, 2011a):

- VOCs: an industry-wide reduction of 25 percent (540,000 tons)
- Air toxics, or hazardous air pollutants: a reduction of nearly 30 percent (38,000 tons)
- Methane: a reduction of about 26 percent (3.4 million tons).

We support EPA's efforts to greatly reduce emissions, but more can be done.

We urge EPA to do more to protect public health: update the section 111 emission guidelines for existing sources as well as making final the New Source Performance Standards for new or expanded sources; update and incorporate the 2011 existing and projected emissions inventory; follow the lead of states such as Colorado and Wyoming to ensure more protective limits on emissions and stronger control requirements; improve the detection and control of leaks and fugitive emissions; target methane with specific control requirements; and require planning for responding to the all-too-frequent problem of malfunction.

These emissions not only harm human health, they waste money. Essentially, gas products are escaping into the air, and these standards provide a proven approach to protect health and save money. EPA has calculated that these measures to install cleanup technologies will save the

industry $30 million annually even as they cut emissions of benzene and other air toxics, as well as volatile organic compounds, sulfur dioxide, ozone, and fine particulate matter.

The updated standards will level the playing field by relying on existing, cost-effective technology and will institutionalize best practices that are already in place in some states. The technologies and best practices allow oil and gas well operators to capture and sell natural gas that currently escapes into the air, threatening public health and wasting a valuable resource.

The adoption of the safeguards against air pollution from oil and natural gas production, as required under the Clean Air Act, will protect the public from life-threatening pollution. Limiting emissions from oil and natural gas production will yield tremendous benefits and significantly reduce adverse health effects.

The nation needs the EPA to strengthen the oil and natural gas standards to effectively protect the health of our patients and our communities. The standards must be strengthened to keep up with the expansions and the new technology in the oil and gas industry. The EPA has a historic and momentous opportunity to clean the air of notoriously harmful pollutants that endanger human health. Our organizations call on the EPA to adopt strong, final oil and natural gas standards by February 28, 2012, and give our patients and communities the clean air they deserve.

Sincerely,

Janice E. Nolen
Assistant Vice President, National Policy and Advocacy
American Lung Association

Charlotte Collins, J.D.
Vice President of Policy and Programs
Asthma and Allergy Foundation of America

Donald P. Hoppert
Director, Government Relations
American Public Health Association

Rebecca Salay
Director of Government Relations
Trust for America's Health

Gary Ewart
Director, Government Relations
American Thoracic Society

References Cited

Agency for Toxic Substances and Disease Registry (ATSDR). 1999a. Toxicological profile for Formaldehyde. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.Web link: http://www.atsdr.cdc.gov/toxprofiles/tp111-c2.pdf [Accessed: 17 October 2011].

Agency for Toxic Substances and Disease Registry (ATSDR). 1999b. Toxicological profile for *n*-Hexane. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. http://www.atsdr.cdc.gov/toxprofiles/tp113-c2.pdf [Accessed: 17 October 2011].

Agency for Toxic Substances and Disease Registry (ATSDR). 2000. Toxicological profile for Toluene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp56-c2.pdf [Accessed: 17 October 2011].

Agency for Toxic Substances and Disease Registry (ATSDR). 2001. Landfill Gas Primer – An Overview for Environmental Health Professionals: Chapter 3 – Landfill Gas Safety and Health Issues. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/HAC/landfill/html/ch3.html [Accessed: 17 October 2011].

Agency for Toxic Substances and Disease Registry (ATSDR). 2007a. Toxicological profile for Benzene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr
.cdc.gov/ToxProfiles/tp3-c3.pdf [Accessed: 17 October 2011].

Agency for Toxic Substances and Disease Registry (ATSDR). 2007b. Toxicological profile for Xylenes. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp71-c3.pdf [Accessed: 17 October 2011].

Agency for Toxic Substances and Disease Registry (ATSDR). 2010. Toxicological profile for Ethylbenzene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp110-c3.pdf [Accessed: 17 October 2011].

American Lung Association. 2010. Trends in COPD (Chronic Bronchitis and Emphysema): Morbidity and Mortality. Web link: http://www.lungusa.org/finding-cures/our-research/trend-reports/copd-trend-report.pdf.

American Lung Association. 2011. Trends in Asthma Morbidity and Mortality. Web link: http://www.lungusa.org/finding-cures/our-research/trend-reports/asthma-trend-report.pdf.

American Academy of Pediatrics (AAP) Committee on Environmental Health. Ambient Air Pollution: Health Hazards to Children. *Pediatrics* 2004; 114: 1699-1707.

Apelberg BJ, Buckley TJ, White RH. Socioeconomic and Racial Disparities in Cancer Risk from Air Toxics in Maryland. *Environmental Health Perspectives*. June 2005;113(6):693-9.

Bell ML, Dominici F, Samet JM. A Meta-Analysis of Time-Series Studies of Ozone and Mortality with Comparison to the National Morbidity, Mortality, and Air Pollution Study. *Epidemiology* 2005; 16:436-445.

Centers for Disease Control and Prevention. (CDC) 2011. National Diabetes Fact Sheet: National Estimates and General Information on Diabetes and Prediabetes in the United States, 2011. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Churg A, Brauer M, Avila-Casado MdC, Fortoul TI, Wright JL. Chronic Exposure to High Levels of Particulate Air Pollution and Small Airway Remodeling. *Environmental Health Perspectives* 2003; 111:714-718.

Delfino RJ, Murphy-Moulton AM, Becklake MR. Emergency room visits for respiratory illnesses among the elderly in Montreal: Association with low level ozone exposure. *Environmental Research* 1998;76:67-77.

Desqueyroux H, Pujet JC, Prosper M, Le Moullec Y, Momas I. Effects of air pollution on adults with chronic obstructive pulmonary disease. *Archives of Environmental Health* 2002;57:554-560

Devlin RB. Identification of subpopulations that are sensitive to ozone exposure: Use of end points currently available and potential use of laboratory-based end points under development. *Environmental Health Perspectives* 1993;101:225-230.

Dietert RR, Etzel RA, Chen D, et al. Workshop to Identify Critical Windows of Exposure for Children's Health: Immune and Respiratory Systems Workgroup Summary. *Environmental Health Perspectives* 2000; 108 (supp 3); 483-490.

D'Ippoliti D, Forastiere F, Ancona C, Agabity N, Fusco D, Michelozzi P, Perucci CA. Air Pollution and Myocardial Infarction in Rome: A Case-Crossover Analysis. *Epidemiology* 2003;14:528-535.

Dominici F, McDermott A, Zeger SL, Samet JM. On the Use of Generalized Additive Models in Time-Series Studies of Air Pollution and Health. *American Journal of Epidemiology* 2002;156:193-203

Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, Samet JM. Fine Particulate Air Pollution and Hospital Admission for Cardiovascular and Respiratory Diseases. *Journal of the American Medical Association* 2006;295:1127-1134.

Franklin M, Zeka A, Schwartz J. Association Between PM2.5 and All-Cause and Specific-Cause Mortality in 27 U.S. Communities. *Journal of Exposure Science and Environmental Epidemiology* 2007;17:279-287.

Gauderman WJ, Gilliland GF, Vora H, Avol E, Stram D, McConnell R, Thomas D, Lurmann F, Margolis HG, Rappaport EB, Berhane K, Peters JM. Association between Air Pollution and Lung Function Growth in Southern California Children: results from a second cohort. *American Journal of Respiratory and Critical Care Medicine* 2002;166:76-84.

Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, Peters J. The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age. *New England Journal of Medicine* 2004;351:1057-1067

Gent JF, Triche EW, Holford TR, Belanger K., Bracken MB, Beckett WS, Leaderer BP. Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *JAMA* 2003;290:1859-1867.

Ghio AJ, Kim C, Devlin RB. Concentrated Ambient Air Particles Induce Mild Pulmonary Inflammation in Healthy Human Volunteers. *American Journal of Respiratory and Critical Care Medicine* 2000;162:981-988.

Hollingsworth JW, Kleeberger SR, Foster WM. Ozone and pulmonary innate immunity. *Proc Am Thorac Soc* 2007;4:240-246.

Hong Y-C, Lee J-T, Kim H, Ha E-H, Schwartz J, Christiani DC. Effects of Air Pollutants on Acute Stroke Mortality. *Environmental Health Perspectives* 2002;110: 187-191.

Horstman DH, Folinsbee LJ, Ives PJ, Abdul-Salaam S, McDonnell WF. Ozone concentration and pulmonary response relationships for 6.6-hour exposures with five hours of moderate exercise to 0.08, 0.10, and 0.12 ppm. *American Review of Respiratory Disease* 1990; 42:1158-1163.

Intergovernmental Panel on Climate Change (IPCC) (Parry, Martin L., Canziani Osvaldo F., Palutikof, Jean P., van der Linden, Paul J., and Hanson, Clair E. (eds.)). 2007. Climate Change 2007: Impacts, Adaptation, and Vulnerability. Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change., Cambridge University Press, Cambridge, United Kingdom, 1000 pp. Web link: http://www.ipcc-wg2.org/index.html [Accessed 17 October 2011].

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

Kunzli N, Lurmann F, Segal M, Ngo L, Balmes J, Tager IB. Association Between Lifetime Ambient Ozone Exposure and Pulmonary Function in College Freshmen-Results of a Pilot Study. *Environmental Research* 1997; 72: .8-23.

Lin M, Chen Y, Burnett RT, Villeneuve PJ, Krewski D. The Influence of Ambient Coarse Particulate Matter on Asthma Hospitalization in Children: case-crossover and time-series analyses. *Environmental Health Perspectives* 2002;110:575-581.

Lin S, Liu X, Le LH, Hwang S-A. Chronic Exposure to Ambient Ozone and Asthma Hospital Admissions among Children. *Environmental Health Perspectives* 2008;116:1725-1730.

Lin S, Munsie JP, Hwang SA, Fitzgerald E, Cayo MR. Childhood Asthma Hospitalization and Residential Exposure to State Route Traffic. *Environmental Research* 2002b; 88:73-81.

Linder SH, Marko D, Sexton K. Cumulative Cancer Risk from Air Pollution in Houston: Disparities in Risk Burden and Social Disadvantage. *Environmental Science and Technology*. June 2008;42(12)4312-22.

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

Louisiana Bucket Brigade. *Common Ground III: Why Cooperation to Reduce Accidents at Louisiana Refineries is Needed Now*. November 2011. Web link: http://labucketbrigade.org/article.php?id=981

McDonnell WF, Stewart PW, Smith MV, Pan WK, Pan J. Ozone-induced respiratory symptoms: exposure-response models and association with lung function. *European Respiratory Journal* 1999;14:845–853.

Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, Kaufman JD. Long-Term Exposure to Air Pollution and Incidence of Cardiovascular Events in Women. *New England Journal of Medicine* 2007; 56:447-458.

Morello-Frosch R, Lopez R. The Riskscape and the Color Line: Examining the Role of Segregation in Environmental Health Disparities. *Environmental Research*. October 2006; 102(2):181-96.

Mortimer KM, Neas LM, Dockery DW, Redline S, Tager IB. The effect of air pollution on inner-city children with asthma. *European Respiratory Journal* 2002; 19:699-705.

Mudway IS and Kelly FJ. An investigation of inhaled ozone dose and the magnitude of airway inflammation in healthy adults. *American Journal of Respiratory and Critical Care Medicine* 2004;169:1089-1095.

National Academy of Sciences (NAS). Institute of Medicine. *Toward Environmental Justice: Research, Education and Health Policy Needs*. 1999. Web link: http://www.nap.edu/openbook.php?isbn=0309064074 [Accessed: 4 January 2010].

National Institute for Occupational Safety and Health (NIOSH). 1985. International Chemical Safety Cards: Methane. Atlanta, GA: Centers for Disease Control and Prevention (CDC). Web link: http://www.cdc.gov/niosh/ipcsneng/neng0291.html [Accessed: 17 October 2011].

National Research Council (NRC), Committee on Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution. *Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution.* National Academy Press, 2008.

Norris G, YoungPong SN, Koenig JQ, Larson TV, Sheppard L, Stout JW. An Association Between Fine Particles and Asthma Emergency Department Visits for Children in Seattle. *Environmental Health Perspectives* 1999;107:489-493.

Olson, Laura. "Counties' shale fees could be delayed: Legislation is still being negotiated." *Pittsburgh Post Gazette*, October 13, 2011. Web link: http://www.post-gazette.com/pg/11286/1181796-454.stm

O'Neill MS, Jerrett M, Kawachi I, Levy JI, Cohen AJ, Gouveia N, Wilkinson P, Fletcher T, Cifuentes L, Schwartz J et al. Health, Wealth, and Air Pollution: Advancing Theory and Methods. *Environmental Health Perspectives* 2003: 111: 1861-1870

Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution. *JAMA* 2002; 287:1132-1141.

Pope CA III, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ. Cardiovascular Mortality and Year-round Exposure to Particulate Air Pollution: epidemiological evidence of general pathophysiological pathways of disease. *Circulation*. 2004;109:71-77.

Roger VL, Go AS, Lloyd-Jones DM, et al. 2011. Heart Disease and Stroke Statistics: 2011 Update: A Report From the American Heart Association. *Circulation*. 123(4):e18–e209

Secretary of Energy Advisory Board (SEAB), U.S. Department of Energy. Shale Gas Production Subcommittee 90-Day Report. August 18, 2011. Web link: http://www.shalegas.energy.gov/resources/081811_90_day_report_final.pdf.

Slaughter JC, Lumley T, Sheppard L, Koenig JQ, Shapiro GG. Effects of Ambient Air Pollution on Symptom Severity and Medication Use in Children with Asthma. *Annals of Allergy, Asthma, & Immunology* 2003; 91:346-53.

Tolbert PE, Mulholland JA, MacIntosh DD, Xu F, Daniels D, Devine OJ, Carlin BP, Klein M, Dorley J, Butler AJ, Nordenberg DF, Frumkin H, Ryan PB, White MC. Air Quality and Pediatric Emergency Room Visits for Asthma in Atlanta, Georgia. *American Journal of Epidemiology* 2000;151:798-810.

Triche EW, Gent JF, Holford TR, Belanger K, Bracken MB, Beckett WS, Naeher L, McSharry JE, Leaderer BP. Low-level ozone exposure and respiratory symptoms in infants. *Environmental Health Perspectives* 2006;114:911–916.

Tsai SS, Goggins WB, Chiu HF, Yang CY. Evidence for an Association Between Air Pollution and Daily Stroke Admissions in Kaohsiung, Taiwan. *Stroke*. 2003;34:2612-2616.

U.S. Department of Health and Human Services (HHS). National Toxicology Program. 2011. *Report on Carcinogens, Twelfth Edition.* Research Triangle Park, NC: U.S. Department of Health and Human Services.

U.S. Environmental Protection Agency (EPA). Air Quality Criteria for Particulate Matter. 2004. Web link: http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_cd.html.

U.S. Environmental Protection Agency (EPA). Air Quality Criteria for Ozone and Related Photochemical Oxidants (2006 Final). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-05/004aF-cF, 2006.

U.S. Environmental Protection Agency (EPA). Air Data. Monitor Value Report Criteria Pollutants, Sublette County, Wyoming. Ozone 2008. Web link: http://iaspub.epa.gov/airsdata/adaqs.monvals?geotype=co&geocode=56035&geoinfo=co~56035~Sublette+Co%2C+Wyoming&pol=O3&year=2008&fld=monid&fld=siteid&fld=address&fld=city&fld=county&fld=stabbr&fld=regn&rpp=25. [Accessed 18 October 2011].

U.S. Environmental Protection Agency (EPA). 2008. Integrated Science Assessment for Oxides of Nitrogen -- Health Criteria. EPA/600/R-08/071.

U.S. Environmental Protection Agency (EPA). 2009a. Risk and Exposure Assessment to Support the Review of the SO_2 Primary National Ambient Air Quality Standards: Final Report. EPA-452/R-09-007, August 2009.

U.S. Environmental Protection Agency (EPA), 2009b. *Integrated Science Assessment for Particulate Matter*, EPA 600/R-08/139F. Web link: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546. [Accessed 17 October 2011].

U.S. Environmental Protection Agency (EPA). 2011a. Regulatory Impact Analysis for Proposed New Source Performance Standards and Amendments to the National Emissions Standards for Hazardous Air Pollutants for the Oil and Natural Gas
Industry. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Web link: http://www.epa.gov/ttn/ecas/regdata/RIAs/oilnaturalgasfinalria.pdf [Accessed 17 October 2011].

U.S. Environmental Protection Agency (EPA). 2011b. Addressing Air Emissions from the Oil and Natural Gas Industry Overview of EPA's Proposed New Source Performance Standards and National Emission Standards for Hazardous Air Pollutants. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Web link: http://www.epa.gov/airquality/oilandgas/pdfs/20110728presentation.pdf [Accessed 18 October 2011].

U.S. Environmental Protection Agency (EPA). 2011c. Regulatory Impact Analysis for proposed Air Toxics Rule, Office of Air Quality Planning and Standards, Research Triangle Park, NC. Web link: http://www.epa.gov/ttn/ecas/regdata/RIAs/ToxicsRuleRIA.pdf [Accessed 17 October 2011].

U.S. Environmental Protection Agency (EPA). 2011d. Memorandum from Gina McCarthy to Air Division Directors, Regions 1-10. September 22, 2011. Web link: http://www.epa.gov/ozonepollution/pdfs/OzoneMemo9-22-11.pdf

U.S. Environmental Protection Agency (EPA). 2011e. Plan to Study the Potential Impacts of Hydraulic Fracturing on Drinking Water Resources. November 2011. Web link: http://water.epa.gov/type/groundwater/uic/class2/hydraulicfracturing/upload/FINAL-STUDY-PLAN-HF_Web_2.pdf

U.S. Environmental Protection Agency (EPA). 2011f. 2011 U.S. Greenhouse Gas Inventory Report Executive Summary. 76 Fed. Reg. 52,792. Web link: http://epa.gov/climatechange/emissions/downloads11/US-GHG-Inventory-2011-Executive-Summary.pdf

World Health Organization (WHO). 2003. Climate change and human health - risks and responses. Web link: http://www.who.int/globalchange/publications/cchhbook/en/ [Accessed: 17 October 2011].

World Health Organization (WHO). 2005. The Effects of Air Pollution on Children's Health and Development: a review of the evidence. 2005. Web link: http://www.euro.who.int/document/E86575.pdf

Zeger SL, Dominici F, McDermott A, Samet J. Mortality in the Medicare Population and Chronic Exposure to Fine Particulate Air Pollution in Urban Centers (2000-2005). *Environmental Health Perspectives* 2008: 116:1614-1619.