

Ross P. Lanzafame, Esq.
Chair
National Board of Directors

Kathryn A. Forbes, CPA
Chair-Elect
National Board of Directors

Albert A. Rizzo, M.D.
Past-Chair

John F. Emanuel
Secretary/Treasurer

Marcia D. Williams, Ed.D.
Speaker
Nationwide Assembly

Audrene Lojovich
Speaker-Elect
Nationwide Assembly

Geri Reinardy, M.P.A.
Past-Speaker
Nationwide Assembly

NATIONAL HEADQUARTERS

1301 Pennsylvania Ave., NW
Suite 800
Washington, DC 20004-1725
Phone: (202) 785-3355
Fax: (202) 452-1805

14 Wall St.
Suite 8C
New York, NY 10005-2113
Phone: (212) 315-8700
Fax: (212) 608-3219

www.Lung.org

**American Lung Association
Comments on the
U.S. Environmental Protection Agency's
Proposed Revisions to National Emissions
Standard for Hazardous Air Pollutants for
Reciprocating Internal Combustion Engines
("RICE NESHAP")
Docket ID No. EPA-HQ-OAR-2008-0708**

August 9, 2012

Janice E. Nolen
Assistant Vice President, National Policy and Advocacy
202-785-3355

Lyndsay F. Moseley
Director of Advocacy, Healthy Air Campaign
202-785-3355

Summary

The American Lung Association appreciates this opportunity to comment on EPA's proposed amendments to the National Emission Standards for Hazardous Air Pollutants (NESHAP) for stationary reciprocating internal combustion engines (RICE). Specifically, we oppose the proposed amendment allowing greater operating hours for RICE or diesel backup generators (BUGs) participating in demand-response programs.

The American Lung Association opposes changes in the proposed rule that allow the use of BUGs for anything other than true emergencies. The EPA's proposed rule will encourage the use of backup generators, including uncontrolled generators, as routine suppliers of power to the electric grid, supplanting the use of cleaner sources of energy and creating a loophole for backup generators that will displace the deployment of cleaner, low-, and non-emitting resources.

The expanded non-emergency use of such generators will contribute emissions that will likely increase ozone and particulate matter levels and make it harder for communities to meet national air quality standards. Furthermore, such use of these generators threatens communities already disproportionately burdened by air pollution.

Background

In 2010, EPA adopted its first national emission standards for several classes of generators, including some primarily classified as emergency backup engines, or BUGs. Hospitals, office buildings, industrial and natural gas and petroleum extraction have historically used these backup generators to ensure reliable electricity in case of a temporary loss of power from the grid. Scheduled to take effect in 2013, EPA's emission standards would cover many previously unregulated BUGs at a variety of sources.

Unfortunately, EPA is proposing to revise their 2010 final rule to open the door to exempt these BUGs from having to clean up their emissions. Some owners of the BUGs have begun participating in the emerging demand response market in exchange for financial compensation. In this demand response market, the owners of BUGs can operate these units during non-emergency periods to provide power to the grid, displacing true demand reductions. Corporations that have historically sought to ensure backup generation in the event of a loss of grid electricity have recently begun to see these BUGs as revenue generators by allowing them to in effect sell power to the grid.

The EPA's proposed rule would encourage that transition of the BUGs from functioning as on-site emergency power supplier to peak power supplier to the grid. As EPA notes in the proposal, most BUGs operate approximately 1 to 2 hours per month. EPA cites the analysis by the California Air Resources Board that found the average yearly use in that state is 31 hours over a three-year period,

with most of those hours being maintenance and testing.¹ Under the final rule, these emergency BUGs could operate up to 15 hours per year in the demand response market, functioning as electricity grid sources without having to meet emissions standards.

However, the EPA proposes to increase the exemption from emission standards for these BUGs from 15 to 100 hours per year when used in demand response and when used more broadly, such as in “peak shaving.” Although designated in the rule as “emergency” demand response, the rule’s language highlights that these other uses are not emergencies. The proposed rule maintains the final rule’s provision that in a true emergency, when no other sources of power are available, there is no time limit on the use of these uncontrolled units.

Diesel Backup Generator Participation in Demand Response Programs

As we noted in our previous comments on the proposed consent decree, many stakeholders support the use of “demand response” to meet the needs for electricity. Used properly, demand response reduces energy use and may increase market efficiency and system reliability while reducing costs. In these programs, customers can reduce load either by actually reducing on-site power consumption (e.g., temporarily shutting down manufacturing production lines or reducing air conditioner use during times of peak electricity demand) or by enlisting other power sources to provide electricity that would otherwise be purchased from the grid.

Both reducing consumption and adding sources can solve the problem of having enough electricity, but each strategy has different health and environmental impacts. All too often, units used to generate additional power in demand response strategies are dirty, diesel BUGs, instead of cleaner alternatives such as solar panels. Using BUGs avoids having to reduce total consumption, so electrical demand is often met by using BUGs, not conservation. Because BUGs are often old, dirty diesel engines, the use of BUGs can increase emissions even as other grid sources are cleaning up.

Furthermore, policies affecting demand response strategy have shifted to increase the incentives to use BUGs. Participants in competitive markets receive incentive payments to make their BUGs available to reduce load. Some demand response providers encourage customers to operate existing BUG units as demand response generation resources rather than in the limited emergency situations for which they were originally intended. As a result, these resources are competing directly with other types of demand response resources as well as cleaner supply side generation resources for the opportunity to earn revenues (NESCAUM, 2012). This is not an “emergency” use of those engines, but an economic use.

¹ However, it should be noted that BUGs are precluded from participating in demand response programs in California; thus, this estimate is likely not relevant to other regions where this is allowed.

Unfortunately, little is known about the extent of the use of backup generators in demand response programs. This lack of information makes it difficult to evaluate the true air quality and health implications of the proposed rule. For example, a recent report by the Northeast States for Coordinated Air Use Management (NESCAUM) identifies the following problem:

What is sorely lacking is an inventory of the resources that are enrolled in or operate under demand response programs, including characteristics such as generator size, installation year, fuel type, emissions rates or controls, and run time. Without this information, air quality planners cannot reasonably assess the air quality impacts of these resources' participation in demand response programs. Older diesel generators, installed prior to national engine emission standards, could have emission rates of NO_x as high as 40 pounds per megawatt-hour (lb/MWh), greater than ten times the NO_x emission rates of well-controlled coal-fired power plants (NESCAUM, 2012).

Health Effects of Diesel Exhaust

Although EPA has made great strides in reducing sulfur content of diesel fuel and requiring new engines to meet more protective emissions standards, serious health risks remain, especially for older engines and engines that do not use ultralow sulfur fuels. Discussed below are some of the risks to public health.

Diesel Emissions

The International Agency for Research on Cancer concluded in June 2012 that diesel exhaust causes lung cancer and was associated with an increased risk of bladder cancer. (IARC Press Release 2012). Acute exposure to diesel exhaust has been associated with serious cardiovascular harm including increased risk of cardiovascular disease, ischemic heart disease and heart attack, as well as premature death. (Krivoshto et al., 2008). Acute or chronic exposure to diesel exhaust is associated with worsened asthma and chronic obstructive pulmonary disease, as well as increased risk for development of asthma. (Proitetti et al., 2003; Takizawa 2004; Holgate et al., 2003) Evidence exists that the damage to the lungs from diesel exhaust increases the risk of developing bacterial and viral bronchitis and pneumonia (Seigel et al., 2004; Krivoshto et al., 2008). Other studies have found links to hypertension, neurotoxicity, and perinatal health and infertility (Krivoshto et al, 2008).

Carbon Monoxide

Carbon monoxide causes a range of harmful effects, including, and particularly, those from cardiovascular effects. Carbon monoxide causes premature death and cardiovascular harm, particularly observed in increased hospital admissions and emergency department visits for ischemic heart disease, myocardial infarction, congestive heart failure and cardiovascular diseases as a whole. Multiple, large epidemiological studies have found strong associations with exposure to CO and serious, documented respiratory health endpoints, including hospital admissions and emergency room visits. (EPA, 2010).

Fine Particulate Matter (PM_{2.5})

Reductions in diesel emissions through requiring the cleanup of BUGs and RICE would provide a crucial collateral benefit: reduction in fine particulate matter. PM_{2.5} is made up of microscopic particles, including aerosols, which can bypass the body's natural defenses and lodge deep within the lungs (EPA, 2004, 2009). 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 S et al., 2002). 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, 2009).

Nitrogen Oxides (NO_x)

Nitrogen oxides (NO_x) are a class of gaseous air pollutants composed of nitrogen and oxygen emitted in diesel exhaust. The pollutant 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 NO_x can increase the risk of hospitalization by up to 20 percent (EPA, 2008). Nitrogen oxides are also precursors to nitrates (NO₃) which also are recognized as aerosolized fine particulate matter (PM_{2.5}) and discussed below. (EPA 2009)

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 own assessment of the health effects of diesel exhaust, many hydrocarbon components of diesel exhaust include organic hazardous air pollutants that most harm human health: formaldehyde, benzene, 1-3 butadiene, toluene, acrolein, dioxin, and PAHs. (EPA, 2002).

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 (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, 1999, 2000, 2007).

Volatile Organic Compounds as Precursors to Ozone (O₃)

As noted above, many volatile organic compounds (VOCs) are hazardous air pollutants. However, VOCs are also precursors to the secondary formation of ozone when they react with nitrogen oxides (NO_x) in the presence of sunlight. By reducing emissions of VOCs, the stronger final 2010 standard would 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, 1999; 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).

Populations at risk

As we mentioned above, many population groups face higher risk from air pollution, particularly children, seniors, people who suffer from respiratory or cardiovascular diseases or diabetics. In addition, people who have low incomes or who work or exercise outdoors face higher risk.

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 (OEHHA 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 as ample warning against encouraging the increased use of uncontrolled BUGs.

Communities of color and poorer people also appear to face higher risk, underscoring the need to properly assess harm to them. 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 2002 analysis of data from the inventory of nearly 4,000 BUGs in California found that most were clustered in urban areas, and within urban areas, they were located where populations were the densest. The populations living within the zone of highest risk for highest risk of exposure to carcinogens were “more likely to be low income, elderly and of a racial minority” (Ryan et al, 2002).
- 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, home to one of the world's largest petrochemical complexes (one of the primary industrial sectors using BUGs), 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).

Emissions Impacts

Given the wide range of health threats associated with exposure to emissions from uncontrolled diesel BUGs, the Lung Association is concerned that EPA has not adequately analyzed the public health impacts of relaxing hazardous air pollutant standards for BUGs participating in demand response programs. Limited data exist on the number, type, and emissions of uncontrolled RICE engines in operation, including those used in DR programs. As we expressed in our comments on the proposed settlement agreement, we continue to urge EPA to conduct a survey of all RICE units and to collect basic data, including emissions and emission controls, if any, sufficient to properly estimate the impacts of this proposal.

The Lung Association is also concerned that EPA has not considered the amount of additional uncontrolled generation that would be built or drawn into these programs in the future if this proposed rule were to be finalized. Of particular concern, EPA's "Summary of Reductions for Existing Stationary RICE" has not been updated to reflect a maximum runtime of 100 hours per year as provided for in the proposed revision. The proposal appears to be based on the assumption that only a small component of these engines will be used in demand response or that they will be rarely deployed. However, by creating a special exemption from both hazardous air pollutant and new source standards, EPA is providing a competitive advantage for diesel BUGs and should thus anticipate more units being deployed, particularly in competitive markets. As such, EPA should also conduct a comprehensive study of the environmental implications of allowing increased operation of existing resources as well as the health implications associated with the potential increased deployment of additional existing or new generators. Access to this information is critical in a comprehensive determination regarding the health and air emissions implications of the rule.

It should also be noted that many of these uncontrolled diesel BUGs, for which this exemption is being proposed, can be fitted with controls to comply with existing environmental standards or replaced with

modern and controlled units. Increased use of these uncontrolled engines may also hinder areas from maintaining or achieving national ambient air quality standards.

The Lung Association urges EPA to take the following steps before finalizing this revision to the rule:

- Conduct a survey of all RICE units, in cooperation with the relevant Independent System Operation/Regional Transmission Organizations, and collect basic data, including emissions and emission controls, if any, sufficient to properly estimate the impacts of this proposal; and
- Conduct a comprehensive study of the public health and environmental implications of allowing increased operation of existing backup generators and engines as well as the public health and environmental implications associated with the potential increased deployment of additional resources.

The EPA needs this information to adequately assess the air quality impacts associated with any such exemption for such generators operating in demand response programs.

Conclusion

While the Lung Association recognizes the need for emergency back-up generation, we cannot support regulations that increase the nation's reliance on high-emitting, uncontrolled generators to power the larger grid. To be clear: We want hospitals to be able to run generators when the power goes out and we want industrial facilities to run water pumps when there is a fire. However, the continued use of high emitting diesel generators is not required to accomplish those goals, nor should those generators be operated when no real emergency exists. In particular, hospitals and other emergency sources can and should take advantage of technology to retrofit their existing generators to reduce the well-documented acute impacts of diesel exhaust and particulate matter.

Furthermore, EPA must require the use of ultralow sulfur diesel in these generators. EPA should work with states to identify an appropriate time frame for phasing out the oldest and dirtiest diesel BUGs, an especially important step as the states look to meet the new particulate matter and NOx national air quality standards.

The Lung Association encourages the EPA to structure a rule that allows for emergency power supply without encouraging the increased use of uncontrolled BUGs as part of the nation's electricity mix. To increase reliability and protect public health, the appropriate response is to provide incentives to promote energy efficiency and decreased consumption during peak periods, rather than favor highly-polluting generation.

In conclusion, Lung Association opposes changes in the proposed rule that allow use of uncontrolled backup generators for any uses other than true emergencies.

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: 9 August 2012].

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: 9 August 2012].

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: 9 August 2012].

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.

Office of Environmental Health Hazard Assessment (OEHHA). In Utero and Early Life Susceptibility to Carcinogens: The Derivation of Age-at-Exposure Sensitivity Measures. California Environmental Protection Agency (CalEPA). May 2009. Web link: http://oehha.ca.gov/air/hot_spots/2009/AppendixEarly.pdf. [Accessed 9 August 2012].

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, 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.

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

Franklin M, Zeka A, Schwartz J. Association Between PM_{2.5} 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.

Holgate ST, Sandstrom T, Frew AJ, et al. Health effects of acute exposure to air pollution. Part I: healthy and asthmatic subjects exposed to diesel exhaust. Research Report/Health Effects Institute 2003; 112:1-30.

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.

International Agency for Research on Cancer (IARC). Press Release: IARC: Diesel Engine Exhaust Carcinogenic. World Health Organization (WHO). June 12, 2012. Web link: http://press.iarc.fr/pr213_E.pdf [Accessed 9 August 2012].

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

Krivoshto IN, Richards JR, Albertson TE, Derlet RW. The Toxicity of Diesel Exhaust: Implications for Primary Care (Clinical Review). *Journal of the American Board of Family Medicine (JABFM)*. January-February 2008 21(1):55-62.

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.

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

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

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.

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.

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.

McGwin G Jr., Lienert J, Kennedy JI Jr.. Formaldehyde Exposure in Children: A Systematic Review. *Environmental Health Perspectives*. March 2010;118(3):113-117.

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 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.

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: 9 August 2012].

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.

Northeast States for Coordinated Air Use Management (NESCAUM). *Air Quality, Electricity, and Back-up Stationary Diesel Engines in the Northeast*. August 1, 2012. Web link:

http://www.nescaum.org/documents/nescaum-aq-electricity-stat-diesel-engines-in-northeast_20120801.pdf [Accessed 9 August 2012].

Peters JM, Avol E, Gauderman WJ, Linn WS, Navidi W, London SJ, Margolis H, Rappaport E, Vora H, Gong H, Thomas DC. A study of twelve southern California communities with differing levels and types of air pollution II. Effects on pulmonary function. *American Journal of Respiratory and Critical Care Medicine* 1999;159:768-775.

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.

Proietti L, Spicuzza L, Polosa R. Urban air pollution at the crossroads of the allergic pandemic. *Annali Italiani di Medicina Interna* 2003;18:64-72.

Ryan NE, Larsen KM, Black PC. Smaller, Closer, Dirtier: Diesel Backup Generators in California. Environmental Defense. 2002. Web link: <http://www.edf.org/content/smaller-closer-dirtier> [Accessed 9 August 2012].

Siegel PD, Saxena RK, Saxena QB, et al. Effect of diesel exhaust particulate (DEP) on immune responses: contributions of particulate versus organic soluble components. *Journal of Toxicology and Environmental Health A*. 2004;67:221-31.

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.

Takizawa H. Diesel exhaust particles and their effect on induced cytokine expression in human bronchial epithelial cells. *Current Opinion in Allergy and Clinical Immunology*. 2004;4:355-9.

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. [Accessed 9 August 2012].

U.S. Environmental Protection Agency (EPA). 2006. 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), 2002. Health Assessment Document for Diesel Engine Exhaust. EPA 600/8-90/057F. Web link: <http://www.epa.gov/ttn/atw/dieselfinal.pdf>. [Accessed 9 August 2012].

U.S. Environmental Protection Agency (EPA), 2009. Integrated Science Assessment for Particulate Matter, EPA 600/R-08/139F. Web link: <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546>. [Accessed 9 August 2012].

U.S. Environmental Protection Agency (EPA). 2010. Integrated Science Assessment for Carbon Monoxide (Final Report). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-09/019F, 2010. Web link: <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=218686> [Accessed 9 August 2012].

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.