I. There Are Significant Adverse Public And Environmental Health Effects Associated With The Air Pollutants Regulated Under This Proposed Rule

A. Hazardous Air Pollutants from Power Plants Cause Wide-ranging Health Harm[1]

During the process of burning coal, power plants emit highly toxic chemicals that threaten human health through the air we breathe, the water we drink, and the food we eat. These pollutants include: corrosive substances (acid gases, such as hydrogen chloride and hydrogen fluoride); carcinogens (formaldehyde, benzene, toluene, and other compounds); organic carbon-based toxins (formaldehyde, dioxins, furans); metals (such as arsenic, nickel, and beryllium); neurotoxins (such as mercury and lead); polycyclic aromatic hydrocarbons (PAH); and radioactive materials (such as radium and uranium).[2,3]

According to the most current data in the National Emissions Inventory, coal-fired power plants produce over 386,000 tons of 84 separate hazardous air pollutants from over 440 plants in 46 states.[2] These plants produce 40 percent of all hazardous air pollutants released from domestic industrial sources, more than any other industrial source. In addition, the combustion of coal to generate electricity produces 76 percent of the total volume of acid gases, 60 percent of arsenic, and 46 percent of mercury released into the atmosphere.[2]

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[1] The studies and references cited in footnotes 2-74 are incorporated by reference, and provided (or abstracts are provided) with the comments submitted August 4: Letter to Administrator Lisa Jackson, U.S. EPA, RE: EPA-HQ-OAR-2009-0234 and EPA-HQ-OAR-2011-0044, by the American Academy of Pediatrics, American Heart Association, American Lung Association, *et al.*

[2] U.S. Environmental Protection Agency (EPA). 2007. National Emissions Inventory (NEI) 2002: Inventory Data: Point Sector Data – ALLNEI HAP Annual 01232008. Web Link: http://www.epa.gov/ttn/chief/net/2002inventory.html#inventorydata [Accessed 11 January 2011].

[3] Agency for Toxic Substances and Disease Registry (ATSDR). 2011. *Toxic Substances Portal: Toxicological Profiles*. Washington, DC, USA: ATSDR. Web Link: http://www.atsdr.cdc.gov/toxprofiles/index.asp [Accessed: 18 February 2011]. ("ATSDR, 2011a").

Research has shown that these toxins are especially dangerous because of the harm they can cause to the circulatory, respiratory, nervous, endocrine, and other essential life systems within humans. Toxic emissions can even cause developmental disorders and premature death. Pollutants such as acid gases, mercury, and sulfur dioxide, have immediate impacts on people, neighborhoods, and towns within the immediate vicinity of a power plant. However, other pollutants, such as dioxins and metals, can travel much farther from the pollution source. These pollutants, which adhere to fine particles, can remain in the air for more than a week and be carried by winds to distant locations. This makes toxic air pollution dangerous to public health and human health both near to and far from coal-fired power plants.[4]

We agree with the strong evidence that EPA provides in the proposed rule to support their decision that regulation of coal- and oil-fired power plants is both appropriate and necessary to protect public health. Not only is there clear evidence that the harm from mercury and methylmercury continues, but recognized carcinogens and other toxics that cause or increase risk of cardiovascular, respiratory, and other acute and chronic systemic damage are also emitted by power plants. The cleanup of toxic air pollution from power plants is necessary for the protection of public health, appropriate for EPA to undertake, and long overdue.

1. There is Evidence that These Toxics Pose Serious Threats to Health and Must Be Reduced

   a. Acid Gases (Including Hydrogen Chloride, Hydrogen Fluoride, Chlorine)

Strongly corrosive gases produced by coal-fired power plants include two of the largest volume toxic emissions by ton: hydrogen chloride and hydrogen fluoride.[2] Acid gas vapors can harm the respiratory tract by reacting with the moisture and tissues on the upper airways (due to their solubility in water) and bind to particles

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[4] U.S. Environmental Protection Agency (EPA), 2009. *Integrated Science Assessment for Particulate Matter*, EPA 600/R-08/139F. Available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546.

to further travel to the alveolar regions of the lung.[5] Chlorine is another toxic acid gas produced in coal combustion.[6]

Large-scale epidemiological studies have revealed a strong linkage between acid gases (bound to particles or as aerosols) and adverse respiratory effects. A study of 13,000 children in 24 cities found that the strong acid aerosols associated with episodes of bronchitis and reduced lung function and acid aerosols were associated with asthma and related symptoms in children.[7,8] Another recent study also found that acid gases and particle pollution were associated with reduced lung function.[9] Hydrogen chloride is a strong acid gas that reacts with moisture to form hydrochloric acid. Hydrogen chloride intensely irritates the mucous membranes of the respiratory system. At high concentrations, hydrogen chloride can cause swelling and spasms in the throat and suffocation. In addition, inhaled hydrogen chloride can lead to a chemical- or irritant-induced form of asthma called Reactive

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[5] U.S. Environmental Protection Agency (EPA). 1998. *Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units – Final Report to Congress, Volume 2. Appendices (EPA-453/R-98-004b).* Research Triangle Park, NC, USA: EPA, Office of Air Quality Planning and Standards, available at http://www.epa.gov/ttncaaa1/t3/reports/eurtc2.pdf.

[6] U.S. Environmental Protection Agency (EPA). 2011. 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 16 June 2011].

[7] Raizenne M, Neas LM, Damokosh AI, Dockery DW, Spengler JD, Koutrakis P, Ware JH, Speizer FE. 1996. Health Effects of Acid Aerosols on North American Children: Pulmonary Function. *Environmental Health Perspectives* 104(5):506-514.

[8] Dockery DW, Cunningham J, Damokosh AI, Neas LM, Spengler JD, Koutrakis P, Ware JH, Raizenne M, Speizer FE. 1996. Health Effects of Acid Aerosols on North American Children: Respiratory Symptoms. *Environmental Health Perspectives* 104(5):500-504.

[9] Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D. 2004. The effect of air pollution on lung function development in children aged 10 to 18 years. *New England Journal of Medicine.*351:1057-1067.

Airway Dysfunction Syndrome (RADS).[10] Both hydrogen chloride and hydrogen fluoride can irritate the eyes, nasal passages, and lungs.[11,12]

Colorless hydrogen fluoride gas poses serious health risks when inhaled, as a result of what the Agency for Toxic Substances and Disease Registry describes as the fluoride ion's "aggressive, destructive penetration of tissues."[13] Hydrogen fluoride irritates the nose, throat, and eyes, inflames the mucous membrane, causes coughing, and induces narrowing of the bronchial tubes. Acute exposures can cause the throat to swell and narrow, obstructing breathing. The reaction to inhaled hydrogen fluoride may not appear for several hours to days after exposure. As with many chemicals, hydrogen fluoride does have benefits under the right circumstances, which are not present in this case: long-term oral exposure to low-levels of fluoride prevents dental cavities and hardens the bones.[13]

At high levels of exposure, chlorine can lead to harmful health impacts. Inhalation of low concentrations can irritate the nose and throat and cause headache. Intermediate amounts of chlorine can cause immediate chest pain, nausea, vomiting, cough, and shortness of breath. Acute exposure to higher levels can lead to more severe health effects and be life threatening; inflammation of lung tissue, pulmonary edema, pneumonia, and even death may result (ATSDR, 2010c).[14]

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[10] Agency for Toxic Substances and Disease Registry (ATSDR). 2010. *Medical Management Guidelines for Hydrogen Chloride (HCl)*. Updated September 1, 2010. Accessed February 27, 2011. http://www.atsdr.cdc.gov/MHMI/mmg173.pdf.
[11] U.S. Environmental Protection Agency (EPA). 2000. *Air Toxics Web Site: Hydrochloric Acid (Hydrogen Chloride) Hazards Summary*. Web Link: http://www.epa.gov/ttn/atw/hlthef/hydrochl.html [Accessed February 18, 2011].
[12] U.S. Environmental Protection Agency (EPA). 2000. *Air Toxics Web Site: Hydrogen Fluoride Hazards Summary*. Web Link: http://www.epa.gov/ttn/atw/hlthef/hydrogen.html [Accessed February 18, 2011].
[13] Agency for Toxic Substances and Disease Registry (ATSDR). 2003. Toxicological profile for Hydrogen Fluoride. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp11-c3.pdf [Accessed: 14 June 2011]. ("ATSDR 2003")
[14] Agency for Toxic Substances and Disease Registry (ATSDR). 2010. Toxicological profile for Chlorine. Atlanta, GA: U.S. Department of Health and Human Services,

Exposure to high concentrations of chlorine has also been linked to long-term neurological effects, such as memory loss, slow reaction time, impaired balance, hearing loss, and visual alterations.[14]

b. Dioxins and Furans (Example: 2,3,7,8-Tetrachlorodibenzo-p-dioxin, Known as TCDD)

Dioxins and furans are a family of toxic chemicals that primarily arise from the burning of fossil fuels, such as coal, and exist in the atmosphere both as a gas and particles.[15] As particles, they can remain airborne for more than ten days, traveling far from their source, and depositing in water and soil.[16] Dioxins have even been found in the U.S. food supply; in 2002-2003, the U.S. Department of Agriculture found dioxin-like substances in meat and poultry.[17] Researchers have found dioxins in the breast milk of nursing mothers. [18]

Short-term exposures can cause liver damage and skin lesions, while long-term exposures can harm the immune system, the developing nervous system, the reproductive system, and disrupt hormone function. One form of dioxin—2,3,7,8-Tetrachlorodibenzo-p-dioxin, or TCDD—is recognized as a known human

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Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp172-c3.pdf [Accessed: 14 July 2011]. ("ATSDR, 2010b")

[15] Oh JE, Choi JS, Chang YS. 2001. Gas/particle partitioning of polychlorinated dibenzo-p-dioxins and dibenzofurans in atmosphere; evaluation of predicting models. *Atmospheric Environment* 35(24): 4125-4134.

[16] Atkinson, R. 1991. Atmospheric lifetimes of dibenzo-p-dioxins and dibenzofurans. *The Science of the Total Environment* 104(1-2): 17-33.

[17] Hoffman MK, Huwe J, Deyrup CL, Lorentzsen M, Zaylskie R, Clinch NR, Saunders P, Sutton WR, USDA – ARS. 2006. Statistically Designed Survey of Polychlorinated Dibenzo-p-dioxins, Polychlorinated Dibenzofurans, and Co-Planar Polychlorinated Biphenyls in US Meat and Poultry, 2002-2003: Results, Trends, and Implications. *Environmental Science & Technology*. 40(17):5340-5346.

[18] Lorber M, Phillips L. 2002. Infant exposure to dioxin-like compounds in breast milk. *Environmental Health Perspectives* 100:A325-A332.

carcinogen.[19,20,21,22,23,24] Researchers are currently exploring the potential for dioxins to act as endocrine disrupters, by mimicking natural hormones in the body and altering their normal function.[25] Last year, the World Health Organization concluded that developing fetuses and newborn children are most vulnerable to the harmful effects of dioxin and furan exposure.[20]

c. Radioisotopes (Examples: Radium, Uranium)

Radioisotopes, or certain forms of elements that are radioactive, emit ionizing radiation that can damage cells and contribute to cancer and other illnesses. Coal combustion is the leading source of radium released into the air, according to the ATSDR.[26] Radioisotopes are known carcinogens, especially as relates to the lungs,

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[19] National Toxicology Program. 2011. *Report on Carcinogens, Twelfth Edition.* Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program. 499 pp.
[20] World Health Organization (WHO). 2010. Fact Sheet: Dioxins and their effects on human health. Geneva, Switzerland: WHO. Web Link: http://www.who.int/mediacentre/factsheets/fs225/en/index.html [Accessed 3 February 2011]. ("WHO 2010")
[21] World Health Organization (WHO). 2011. *Concise International Chemical Assessment Documents*. Geneva, Switzerland: WHO: Web Link: http://www.who.int/ipcs/publications/cicad/en/. [Accessed 3 February 2011]. ("WHO 2011")
[22] Agency for Toxic Substances and Disease Registry (ATSDR). 1994. Toxicological profile for chlorodibenzofurans (CDFs). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp32-c2.pdf [Accessed 14 Jun 2011].
[23] Agency for Toxic Substances and Disease Registry (ATSDR). 1998. Toxicological profile for Chlorinated Dibenzo-p-dioxins (CDDs). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp104-c2.pdf [Accessed: 14 June 2011].
[24] Agency for Toxic Substances and Disease Registry (ATSDR). 2000. Toxicological profile for Polychlorinated Biphenyls (PCBs). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp17-c3.pdf [Accessed: 14 June 2011].
[25] Casals-Casas C, Desvergne B. 2011. Endocrine disruptors: from endocrine to metabolic disruption. *Annual Reviews of Physiology* 73:135-162.
[26] Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological profile for radium. Atlanta, GA: U.S. Department of Health and Human Services,

bones, and lymphatic system. They can also cause kidney disease, pneumonia, anemia, and brain abscess.[26,3, 27,21]

d. Polynuclear Aromatic Hydrocarbons (PAH) (Examples: Naphthalene, Benzo-a-anthracene, Benzo-a-pyrene, Benzo-b-fluoranthene, Chrysene, Dibenzo-a-anthracene)

Polynuclear-Aromatic Hydrocarbons or PAHs are a group of over 100 chemicals formed during incomplete combustion of coal and other fuels; they include probable human carcinogens. PAHs adhere to particulate matter and deposit in the lungs.[28,3,29] New research warns that prenatal exposure to PAHs may impact the mental development of children to age 5.[28, 30,21]

e. Volatile Organic Compounds (Examples: Acetaldehyde, Benzene, Formaldehyde, Toluene, Xylene)

Volatile organic compounds take in a host of chemicals that include carcinogens and other toxins and also contribute to the formation of ozone. According to EPA's Regulatory Impact Assessment, three organic hazardous air pollutants dominate the mass from coal-fired power plants: acetaldehyde, benzene, and formaldehyde. Benzene and formaldehyde are recognized as known human

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Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp144-c2.pdf [Accessed: 13 June 2011]. ("ATSDR, 1990")

[27] Agency for Toxic Substances and Disease Registry (ATSDR). 2011. Toxicological profile for Uranium *(Draft for Public Comment)*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. http://www.atsdr.cdc.gov/ToxProfiles/tp150-c3.pdf [Accessed: 13 June 2011].

[28] Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for Polycyclic Aromatic Hydrocarbons (PAHs). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp69-c2.pdf [Accessed: 14 June 2011].

[29] Vineis P and Husgfvel-Purisainen K. Air Pollution and cancer: biomarker studies in human populations. *Carcinogenesis* 2005; 26: 11: 1846-1855

[30] Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Toxicological profile for Naphthalene, 1-Methylnaphthalene, and 2-Methylnaphthalene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp67-c3.pdf [Accessed: 13 June 2011].

carcinogens, while acetaldehyde is considered a probable carcinogen.[31] Non-cancer effects associated with these organics include irritation of the skin, eyes, nose, and throat. 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 adverse effects to the nervous system, impaired memory, and slow response to visual stimuli.[32,33,34,35,36,3,21]

f. Mercury (Including Methylmercury)

Mercury is a primary metal emitted from coal-fired power plant combustion in three forms: as a vaporous gas of elemental mercury; oxidized, and bound with particles. Elemental mercury stays airborne, resulting in widespread distribution. Oxidized and particle-bound mercury deposit nearer to the sources. Once released to the atmosphere, mercury returns to the earth in rain or snowfall, and pollutes waterways and the wildlife in them.[6] Microorganisms convert mercury into

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[31] 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.
[32] Agency for Toxic Substances and Disease Registry (ATSDR). 1999. 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: 13 June 2011].
[33] Agency for Toxic Substances and Disease Registry (ATSDR). 2000a. 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: 13 June 2011].
[34] Agency for Toxic Substances and Disease Registry (ATSDR). 2007. 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: 13 June 2011].
[35] Agency for Toxic Substances and Disease Registry (ATSDR). 2007. 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: 13 June 2011].
[36] 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: 13 June 2011].

methylmercury, a highly toxic form of mercury that bioaccumulates in fish and shellfish.[37,38] Although a person can be exposed to mercury through breathing contaminated air or through skin contact, methylmercury is most easily absorbed by eating contaminated food, especially fish or shellfish. The long-term, low-level exposure to methylmercury that results from the regular consumption of contaminated fish is a primary health concern.[39]

Eating foods containing methylmercury can expose the brains of adults, children and developing fetus to harm. Critical periods are during pregnancy and in the early months after children are born.[37] Mercury exposure can lead to developmental birth defects and interfere with neurological development.[40] Pregnant women who consume fish and shellfish can transmit methylmercury to their developing fetus, and infants can ingest methylmercury in breast milk. Children can also become exposed by eating contaminated fish. [32] Each year, more than 300,000 children born in the US have levels of mercury in their blood high enough to impair performance on brain development test and permanently affect intelligence.[41,42] Additionally, mercury can damage the kidneys, liver, brain, and nervous system as a potent neurotoxin, even in adults.[37,3,21] A recent study has also

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[37] Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological profile for Mercury. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp46-c2.pdf [Accessed: 13 June 2011].
[38] Grandjean P. 2010. Adverse effects of methylmercury: environmental health research implications. *Environmental Health Perspectives* 118: 137-1145.
[39] U.S. Environmental Protection Agency (EPA). 1997. *Mercury Study Report to Congress*, Volumes I – VIII: (EPA-452/R-97-003 through EPA-452/R-97-010). Washington, DC, USA: EPA.
[40] Bose-O'Reilly S, McCarty KM, Steckling N, Lettmeier B. 2010. Mercury Exposure and Children's Health. *Current Problems in Pediatric and Adolescent Health Care* 40(8):186-215.
[41] Trasande L, Landrigan PJ, Schechter C. 2005. Public health and economic consequences of methylmercury toxicity to the developing brain. *Environmental Health Perspectives* 113(5): 590-596.
[42] Axelrad DA, Bellinger DC, Ryan LM, Woodruff TJ. 2007. Dose-response relationship of prenatal mercury exposure and IQ: An integrative analysis of epidemiologic data. *Environmental Health Perspectives*. 115(4): 609-615.

found that methylmercury exposure may lessen the cardiovascular benefits of regular fish consumption.[43]

g. Non-Mercury Metals (Examples: Arsenic, beryllium, cadmium, chromium, lead, nickel, selenium, manganese)

Non-mercury metals and metal-like substances (e.g. arsenic and selenium) comprise a significant part of fine particulate matter (PM2.5) directly emitted from coal-fired power plants. Fine particle pollution is aggravated by the secondary particles formed as a result of chemical reactions in sulfur dioxide and nitrogen oxides emissions. Those secondary particles, notably sulfates and nitrates, pose life-threatening risks similar to those from primary particle emissions.

Inhaled particles deposit along the respiratory tract or penetrate deeply into the gas-exchange region of the lung. EPA has already concluded that exposure to fine particulate matter (PM2.5) causes cardiovascular effects and premature mortality and is likely to cause respiratory harm. The Agency concluded that the evidence suggests that long-term exposure to PM2.5 causes reproductive and developmental effects as well as cancer, mutagenicity and genotoxicity. [4] The risks of cardiovascular harm include acute myocardial infarction, congestive heart failure, cardiac arrhythmias, and strokes. Risks of respiratory harm include coughing, wheezing, difficulty breathing, asthma exacerbations, and increased hospitalization for chronic obstructive pulmonary disease (COPD).[4] Evidence has also grown to warn that long-term exposure to PM 2.5 can increase the risk of low birth weight, infant mortality, and cancer, especially lung cancer.[4]

The level of toxicity of fine particles varies and is likely impacted by the presence of metals or other pollutants.[44] Metals interact with particles to create

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[43] Domingo JL. 2007. Omega-3 fatty acids and the benefits of fish consumption: is all that glitters gold? *Environment International*. 33(7):993-8.

"reactive oxygen species" which limit the body's ability to repair damage to its cells and contribute to tissue inflammation.[45,46,47] Research has shown that sulfate, selenium, iron, nitrate, and organic carbon affect immune cell response and heart variability.[48,49] Elevated presence of chromium, lead, and other metals in PM has been associated with greater effects on hospital admissions for cardiovascular disease, according to a study of Medicare recipients in 26 communities.[50] Zanobetti et al. found that admissions for heart attacks were higher where the PM was enriched in arsenic, chromium, manganese, nickel, and organic carbon. The same study found that showing up in high levels of arsenic, organic carbon, and sulfate in PM—potential indicators of coal combustion—were associated with increased hospital admissions for diabetics.[50] A large study of 25 U.S. communities found increased mortality rates when the fraction of aluminum, sulfate, and nickel in

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[44] Bell ML, Dominici F, Ebisu K, Zeger SL, Samet JM. 2007. Spatial and temporal variation in PM2.5 chemical composition in the United States for health effects studies. *Environmental Health Perspectives* 115(7):989-995.
[45] Carter JD, Ghio AJ, Samet JM, Devlin RB. 1997. Cytokine Production by Human Airway Epithelial Cells after Exposure to an Air Pollution Particle Is Metal-Dependent. *Toxicology and Applied Pharmacology*. 146(2):180-188.
[46] Gurgueira SA, Lawrence J, Coull B, Krishna Murthy GG, Gonzalez-Flecha B. 2002. Rapid increases in the steady-state concentration of reactive oxygen species in the lungs and heart after particulate air pollution inhalation. *Environmental Health Perspectives* 110(8): 749-765.
[47] Wilson MR, Lightbody JH, Donaldson K, Sles J, Stone V. 2002. Interactions between Ultrafine Particles and Transition Metals *in Vivo* and *in Vitro*. *Toxicology and Applied Pharmacology* 184(3): 172-179.
[48] Huang, Y.-CT, Ghio AJ, Stonehuerner J, McGee J, Carter JD, Grambow SC, and Devlin RB. 2003. The role of soluble components in ambient fine particles-induced changes in human lungs and blood. *Inhalation Toxicology*. 15:327-342.
[49] Chuang KJ, Chan CC, Su TC, Lin LY, Lee CT. 2007. Associations between particulate sulfate and organic carbon exposures and heart rate variability in patients with or at risk for cardiovascular diseases. *Journal of Occupational and Environmental Medicine* 49(6):610-617.
[50] Zanobetti A, Franklin M, Koutrakis P, Schwartz J. 2009. Fine particulate air pollution and its components in association with cause-specific emergency admissions. *Environmental Health* 8:58.

particulate matter was highest.[51] This study found additional evidence warning that the combination of metals in particles, a common occurrence, may increase their toxicity.

Arsenic exposure can occur through the dermal, oral, and inhalation routes. As a known carcinogen, inhalation of arsenic has been strongly associated with lung cancer.[31] Short-term inhalation can harm the gastrointestinal tract, cause nausea and diarrhea, and even adversely affect the nervous system. Long-term inhalation has been associated with irritation of the skin and mucous membranes. Exposure can lead to respiratory tract irritation, conjunctivitis, and damage nasal tissue.[52] Similar to effects of inhaling arsenic, arsenic in drinking water has also been linked to skin, bladder, lung, and liver cancer.[31] Long-term exposure through ingestion can result in anemia, lesions, liver, kidney, and nerve damage, and affect the digestive system.[53]

Beryllium is a known carcinogenic metal.[31] Inhaled beryllium has been found to increase the risk of lung cancer.[54,55] Breathing large amounts of beryllium compounds can damage the lungs and cause the lungs to resemble pneumonia with reddening and swelling. Long-term exposure may cause chronic beryllium disease. People with chronic beryllium disease may experience weakness, fatigue, difficulty

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[51] Franklin M, Koutrakis P, Schwartz J. 2008. The Role of Particle Composition on the Association Between PM2.5 and Mortality. *Epidemiology*. 19: 680-698.
[52] Agency for Toxic Substances and Disease Registry (ATSDR). 1998. Medical Management Guidelines for Arsenic. Atlanta, GA: U.S. Department of Health and Human Services. Web link: http://www.atsdr.cdc.gov/mhmi/mmg168.html#bookmark02. [Accessed June 16, 2011].
[53] Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological profile for Arsenic. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp2-c3.pdf [Accessed: 13 June 2011].
[54] Steenland K, Ward E. 1991. Lung cancer incidence among patients with beryllium disease: a cohort mortality study. *J Natl Cancer Inst* 83(19): 1380-1385.
[55] Ward E, Okun A, Ruder A, Fingerhut M, Steenland K. 1992. A mortality study of workers at seven beryllium processing plants. *Am J Ind Med* 22(6): 885-904.

breathing, anorexia, weight loss, and blueness of the hands and feet. The disease can lead to heart enlargement, heart disease, and even death.[56]

Cadmium is another known carcinogenic metal.[31] Exposure to airborne cadmium causes lung cancer, as the International Agency for Research on Cancer reaffirmed in 2009.[57] Prolonged inhalation of cadmium can also lead to gradual accumulation of the metal in the kidneys, resulting in kidney disease.[58]

Chromium occurs in three main forms, one of which, chromium (IV), is a known carcinogen that can increase the risk of lung cancer.[59,31] The metal primarily affects the respiratory system, although chromium (VI) can also impact the gastrointestinal, immunological, hematological, reproductive and developmental systems, particularly if ingested. Inhaling chromium (VI) can cause coughing and wheezing, shortness of breath, bronchitis, pneumonia, decreased lung function, and other respiratory conditions. In workers, inhaled chromium (VI) has been linked develop asthma development and asthma attacks.[59]

The most common health effects associated with lead exposure impact the nervous system and damage its functions. Lead may also cause joint weakness,

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[56] Agency for Toxic Substances and Disease Registry (ATSDR). 2002. Toxicological profile for Beryllium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp4-c3.pdf [Accessed: 13 June 2011].
[57] Straif K, Benbrahim-Tallaa L, Baan R, Grosse Y, Secretan B, El Ghissassi F, et al. 2009. A review of human carcinogens—Part C: metals, arsenic, dusts, and fibres. *Lancet Oncol* 10(5):453-454.
[58] Agency for Toxic Substances and Disease Registry (ATSDR). 2008. Toxicological profile for Cadmium *(Draft for Public Comment)*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp5-c3.pdf [Accessed: 13 June 2011].
[59] Agency for Toxic Substances and Disease Registry (ATSDR). 2008. Toxicological profile for Chromium *(Draft for Public Comment)*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp7-c3.pdf [Accessed: 13 June 2011].

anemia, and increased blood pressure.[60] Although lead is harmful to both adults and children, children are most susceptible to its effects. Lead can cause developmental disorders whose effects can persist beyond childhood.[61] Exposure can affect a child's physical and mental growth, resulting in slower mental development and lower levels of intelligence. Lead is also a probable carcinogen.[31]

Similar to lead, manganese mostly affects the nervous system. For example, adverse effects to hand-eye coordination, hand steadiness, and visual reaction time were observed in humans exposed to manganese.[62] High exposure levels may result in feelings of lethargy and weakness, psychological impacts, and tremors.

Compounds containing nickel have been determined to be carcinogenic.[31] A known health effect of nickel exposure is the increased risk of lung and nasal cancers from nickel dust.[63]

Selenium exposure can result in adversely affect the respiratory system by irritating mucous membranes and causing pneumonia, bronchitis, and pulmonary

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[60] Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological profile for Lead. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp13-c3.pdf [Accessed: 14 June 2011].
[61] Agency for Toxic Substances and Disease Registry (ATSDR). 2007e. Public Health Statement for Lead. CAS#: 7439-92-1. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/phs13.html [Accessed: 16 June 2011].
[62] Agency for Toxic Substances and Disease Registry (ATSDR). 2008c. Toxicological profile for Manganese *(Draft for Public Comment)*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp151-c3.pdf [Accessed: 13 June 2011].
[63] Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Toxicological profile for Nickel. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp15-c3.pdf [Accessed: 13 June 2011].

edema.[64] One selenium compound, selenium sulfide, is also considered to be a probable human carcinogen.[31]

h. Secondary Particles

Meeting the limits for toxic air emissions set under the Mercury and Air Toxics standard provides a crucial collateral benefit: reduction in secondary PM 2.5, especially sulfates and nitrates. Reductions to sulfur dioxide and nitrogen oxide emissions are anticipated to occur as a result of the actions undertaken to meet the requirements of this rule. The reductions will also decrease secondary particle formation. Measures that reduce acid gases will reduce sulfur dioxide and consequently, reduce the burden of sulfate particles across the nation. EPA projects that combined pollution control technologies to meet the limits on mercury will also reduce oxides of nitrogen. In addition, fuel switching and retirements are also expected to reduce nitrogen oxide emissions, and consequently nitrate particles. Sulfates formed from sulfur dioxide comprise the majority of fine particulate matter in much of the United States, especially during the summer months. Nitrogen oxides form nitrates, the third largest source of PM 2.5.[65] As discussed earlier, there is evidence showing that PM 2.5 causes cardiovascular harm and premature mortality, and is likely associated with respiratory disease. Additional studies suggest that long-term exposure to PM2.5 may lead to reproductive and developmental effects, as well as cancer, mutagenicity, and genotoxicity.[4]

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[64] Agency for Toxic Substances and Disease Registry (ATSDR). 2003. Toxicological profile for Selenium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Web link: http://www.atsdr.cdc.gov/ToxProfiles/tp92-c3.pdf [Accessed: 13 June 2011].

[65] U.S. Environmental Protection Agency (EPA). 2011. 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 16 June 2011].

2. Millions of People Face Higher Risk

Many individuals face greater risk because of their age, health conditions, or rate of exposure to the pollutants. Individuals at greater risk include: infants, children and teenagers; seniors; pregnant women; individuals with asthma and other lung diseases; people with cardiovascular diseases; diabetics; low-income communities; and those who work or exercise outdoors.[4,39] The discussion below highlights special concerns for several of these groups.

Children are more vulnerable to the adverse health effects of acid gases (and to all air pollution) than adults. Children grow 80 percent of their lungs between birth and adolescence. During the early postnatal period these delicate, growing tissues are at greatest risk. Children also breathe more rapidly and tend to spend more time outdoors than adults, leading to higher pollutant exposures.[66]

Even before birth children confront increased risk. As noted earlier, fetuses, infants, and children face impaired neurological development and cognitive abilities, memory, language skills because of the toxic effects of methylmercury exposure. Dioxins and furans threaten their developing systems, including the nervous system, and these toxics and others may increase the risk of cancer in children. Furthermore, estimates for children may understate the risks from toxics because of limited monitoring, incomplete information on toxicity and use of models that do not consider the potential for increased risk in children.[66]

People with chronic diseases, including cardiovascular diseases, respiratory diseases and diabetes, face higher risk regardless of age. Current estimates of this group include:

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[66] American Academy of Pediatrics, Committee on Environmental Health. 2004. Ambient Air Pollution: Health Hazards to Children. *Pediatrics*; 114: 1699-1707. Reaffirmed in 2010.

- Asthma - 24.6 million people, including 7.0 million under age 18.[67]
- Cardiovascular diseases – 82.6 million people.[68]
- Diabetes – 25.8 million people.[69]
- Chronic Obstructive Pulmonary Disease (COPD)—12.1 million adults age 18 and older.[70]

As adults age, physiological process naturally decline. This places even healthy individuals advanced in age at risk from airborne pollutants. In addition, many adults also have one or more chronic diseases, increasing their susceptibility to the effects of toxic air emissions.[4]

Individuals with low incomes or that are members of racial and ethnic minorities bear a disproportionate burden of the health effects of air pollution. Since they are more likely to live close to industrial facilities and high traffic areas, low-income and minority populations are at much higher risk of exposure to the most harmful pollutants.[71,72] One study found that 68 percent of African Americans lived within 30 miles of a coal-fired power plant.[73] Another study of five power

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[67] American Lung Association. 2011. Trends in Asthma Morbidity and Mortality. Weblink: http://www.lungusa.org/finding-cures/our-research/trend-reports/asthma-trend-report.pdf.
[68] 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
[69] 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.
[70] American Lung Association. 2010. Trends in COPD (Chronic Bronchitis and Emphysema): Morbidity and Mortality. Weblink: http://www.lungusa.org/finding-cures/our-research/trend-reports/copd-trend-report.pdf.
[71] Levy JI, Greco SL, Spengler JD. 2002. The importance of population susceptibility for air pollution risk assessment: a case study of power plants near Washington, DC. *Environmental Health Perspectives.* 110(12):1253-60.
[72] O'Neill MS, Jerrett M, Kawachi I, Levy JI, Cohen AJ, Gouveia N, et al. 2003. Health, Wealth, and Air Pollution: Advancing Theory and Methods. *Environmental Health Perspectives* 111:1861-1870.
[73] Georgia Coalition for the Peoples' Agenda, Black Leadership Forum, the Southern Organizing Committee for Economic and Social Justice and Clear the Air. 2002. *Air*

plants in the Washington, DC area found that African Americans and individuals without a high school education were among the groups hardest hit by pollution from the plants. Almost half of the risks for premature death due to power plant pollution-related exposures were borne by the 25 percent of the population without a high school education.[71]

B. The HAPs Emitted by EGUs are Associated with Adverse Environmental Effects

We strongly support EPA's assessment that Mercury and the other Hazardous Air Pollutants covered by this proposed regulation have adverse impacts on wildlife, including endangered species, and ecosystem health. The body of scientific evidence referenced in the proposed rule clearly supports the Agency's assessment, however it is important to note that there is more to the story. We offer a set of additional studies that demonstrate how much more serious, and widespread, the mercury contamination problem is in the U.S. Additionally, we provide brief mention of environmental impacts from some of the additional hazardous air pollutants covered by the proposed rule. New research presented here is intended to build on the scientific information accurately presented by EPA in this proposed rulemaking.

1. Mercury

Mercury is a highly toxic heavy metal that affects the function and development of the central nervous system in wildlife, resulting in a broad range of adverse impacts such as reproductive problems, behavioral abnormalities, and compromised immune systems.[74,75,76,77] Historically, scientists have focused on fish-

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of Injustice. Access at http://www.catf.us/resources/publications/files/Air_of_Injustice.pdf.

[74] Tan, S.W, J.C. Miller, and K.R. Mahaffey. 2009. "The endocrine effects of mercury in humans and wildlife." *Critical Reviews in Toxicology* 39(3):228-269.

[75] Hawley, D.M., K.K. Hallinger, D.A. Cristol. 2009. "Compromised immune competence in free-living tree swallows exposed to mercury." *Ecotoxicology* (2009) 18:499-503

eating wildlife as those most at risk of exposure to mercury. However, over the last few years has been a growing awareness among the scientific community that mercury is accumulating in far more habitats, and therefore far more species, than previously thought.[78,79,80] Scientists are also gaining a greater understanding about the impacts of high levels of mercury accumulation on the health of specific species. It has become clearer in recent years that significant exposure to mercury can actually lead to wildlife population declines.

In National Wildlife Federation's September, 2006 report *Poisoning Wildlife*, the full range of species at risk from mercury contamination was highlighted.[81] The report covered scientific data finding elevated levels of mercury in fish, aquatic birds, forest birds, mammals, reptiles, amphibians, and invertebrates, demonstrating that there is literally no corner of the food web untouched by mercury. Recent studies continue to show high levels of mercury accumulation in our environment. For example, a 2007 study that looked at mercury levels in four

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[76] Hallinger, K.K., D.A. Cristol. 2011. "The role of weather in mediating the effect of mercury exposure on reproductivie success in tree swallows." *Ecotoxicology*. Published online 5/8/11.
[77] Scheuhammer, A. M., M. W. Meyer, M.B. Sandheinrich, M.W. Murray. 2007. "Effects of Environmental Methylmercury on the Health of Wild Birds, Mammals, and Fish." *Ambio* Vol. 36, No. 1. p. 12
[78] Evers, David C. 2005. *Mercury Connections: The extent and effects of mercury pollution in northeastern North America*. BioDiversity Research Institute. Gorham, Maine. Online at : http://www.briloon.org/old_site/about/staff/documents/BRIMercury.pdf
[79] Wada, H., D. Yates, D. Evers, R. Taylor, W. Hopkins. 2010. "Tissue mercury concentrations and adrenocotical responses of female big brown bats near a contaminated river." *Ecotoxicology* 19: 1277-1284.
[80] Sleeman, J.M., D.A. Cristol, A.E. White, D.C. Evers, R.W. Gerhold. 2010. "Mercury Poisoning in a Free-Living Northern River Otter." *Journal of Wildlife Diseases* 46(3) 1035-1039.
[81] National Wildlife Federation. 2006. *Poisoning Wildlife: The Reality of Mercury Pollution*. Online at: www.nwf.org/mercury

turtle species from a Virginia river found the highest levels of mercury ever recorded in reptiles.[82]

New studies connecting high levels of mercury with adverse impacts on wildlife populations are perhaps of greatest concern. It is clear that mercury levels in the environment have increased to levels posing risks to ecological and human health.

In a recent study of the Common loon in Maine and New Hampshire, adverse impacts on behavior, physiology, reproductive success, and survival were associated with high mercury levels. Specifically, higher levels of mercury lead to reduced number of chicks fledged per year. The study attributed high mercury levels as the driving stressor for declines in local breeding populations of loons in these areas.[83] Similar impacts were seen in a study of Avocet & Black-necked Stilt, where higher mercury levels were documented in dead chicks than in randomly selected live chicks of similar age.[84]

A recent study of American Kestrals also found that mercury bioavailability could have population level impacts. The research found that high levels of mercury lead to marked decrease in egg production, incubation performance, number of eggs hatched, and number successful fledglings. In cases where mercury levels were highest, total fledgling failure was observed.[85]

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[82] Bergeron, Christine M. et al. 2007. "Influence of Feeding Ecology on Blood Mercury Concentrations in Four Species of Turtles." *Environmental Toxicology and Chemistry*, Vol. 26. No. 8. p. 1733-1741.
[83] Evers, David C. et al. 2008. "Adverse Effects from Environmental mercury loads on breeding common loons." *Ecotoxicology* 17:69-81.
[84] Ackerman, J.T., J.Y. Takekawa, C.A. Eagles-Smith, S.A. Iverson. 2008. "Mercury contamination and effects on survival of American avocet and black-necked stilt chicks in San Francisco Bay. *Ecotoxicology* (2008) 17:103-116.
[85] Albers, Peter H. et al. 2007. "Effects of Methylmercury on Reproduction in American Kestrals." *Environmental Toxicology and Chemistry* Vol 26, No. 9, p.1856-1866.

Mercury contamination has also recently been linked to population declines for songbirds and species that forage in wetlands.[86] New research has lead to greater understanding of mercury level thresholds where impacts on nesting success can be predicted. Specifically, species including the Traill's Flycatcher, Yellow-throated Vireo, Seaside Sparrow, Red-winged Blackbird, Eastern Wood Pewee, Yellow Palm Warbler, Indigo Bunting, and Nelson's Sparrow have all been found with mercury levels above this threshold, indicating they are at risk of reduced nesting success.[87] Additionally, species including Carolina Wren, House Wren, and Song Sparrow in mercury-contaminated habitats have been found to have a lower diversity of song note types and lower tonal frequencies than those in areas with lower mercury levels.[88] This kind of behavioral abnormality can adversely affect bird's ability to perform functions critical to survival and reproduction.

In addition to birds, a recent study found adverse effects of elevated mercury levels in Salamanders. Specifically, high levels of mercury were linked to reduced speed and responsiveness, and as well as inability to successfully execute tasks critical for survival such as hunting.[89]

2. Acid Gases

We strongly support the proposed regulation's reduction of acid gas emissions, given the devastating impact that acid rain has on our forests, lakes, and

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[86] Edmonds, Samuel T. et al. 2010. "Geographic and Seasonal Variation in Mercury Exposure of the Declining Rusty Blackbird." *The Condor* 112(4): 789-799.
[87] Evers et al. 2011 (DRAFT). Hidden Risk – Mercury in Terrestrial Ecosystems. BioDiversity Research Institute . Gorham, Maine. p.11. Draft report, due to be released August 2011. Online at: www.briloon.org
[88] Hallinger, K.K. 2010. "Birdsong Differs Between Mercury-Polluted and Reference Sites" *The Auk* 127(1):156-161.
[89] Burke, J.N., C.M. Bergeron, B.D. Todd, W.A. Hopkins. 2010. "Effects of mercury on behavior and performance of northern two-lined salamanders. *Environmental Pollution* 158(2010):3546-3551

streams.[90] Acidified ecosystems leach calcium and magnesium from soil, which reduces the availability of invertebrates, a critical prey source for many species, and adversely impacts birds breeding success.[91] Acidification has also been linked to fish[92] and bird[93] species declines, and a range of impacts on other wildlife.[94]

It is also important to note that acidified ecosystems also exacerbate mercury contamination problems. Studies have shown that highly acidic environments facilitate greater methylation of mercury, which leads to more significant uptake into the food web.[95,96,97]

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[90] Driscoll, Charles T et al. 2001. *Acid Rain Revisited*. Hubbard Brook Research Foundation. Science Links Publication. Vol. 1 No. 1. Online at: http://www.hubbardbrook.org/6-12_education/Glossary/AcidRain.pdf

[91] Likens, G.E., C.T. Driscoll, and D.C.Buso. 1996. "Long term effects of acid rain: response and recovery of a forest ecosystem." *Science* 272:2444-246.

[92] Dana R. Warren, Gene E. Likens, Donald C. Buso and Clifford E. Kraft. "Status and Distribution of Fish in an Acid-Impacted Watershed of the Northeastern United States (Hubbard Brook, NH)." *Northeastern Naturalist* Vol. 15, No. 3 (2008), p. 375-390.

[93] Ralph S. Hames, Kenneth V. Rosenberg, James D. Lowe, Sara E. Barker and André A. Dhondt. "Adverse Effects of Acid Rain on the Distribution of the Wood Thrush Hylocichla mustelina in North America." *Proceedings of the National Academy of Sciences of the United States of America*. Vol. 99, No. 17 (Aug. 20, 2002), p. 11235-11240.

[94] R. Kent Schreiber and James R. Newman. "Acid Precipitation Effects on Forest Habitats: Implications for Wildlife." *Conservation Biology* Vol. 2, No. 3 (Sep., 1988), pp. 249-259.

[95] Driscoll, Charles T., et al. 2007. "Mercury Contamination in Forest and Freshwater Ecosystems in the Northeastern United States." *Bioscience* Vol. 57 No. 1. p. 17.

[96] Gilmour, Cynthia. "The Effect of the Sulfur Cycle on Methylmercury Production." *2003 Everglades Consolidated Report*. Appendix 2B-2: Status Report on the Effect of Water Quantity and Quality on Methylmercury Production. A-2B-2-1. Online at: http://www.dep.state.fl.us/water/wqssp/everglades/consol_rpt.htm

[97] Cristol, Daniel A. et al. 2008. "The Movement of Aquatic Mercury Through Terrestrial Food Webs." *Science* Vol. 320. p. 335

3. Other Hazardous Air Pollutants

This proposed regulation seeks to reduce a large range of toxic emissions, many of which we know have devastating impacts on wildlife health. A recent publication from the Society of Environmental Toxicology and Chemistry (SETAC)[98] provides a comprehensive look at the impacts of toxic exposure to a variety of wildlife.[99] A selection of key findings from this research and others are summarized in this subsection.

Arsenic has long been recognized for its toxic properties. Studies have linked arsenic exposure, even at very low doses, with severely adverse impacts on cell growth.[100] At higher doses, arsenic exposure can lead to cancer and death.[101] Arsenic in soils has also been linked reduced root growth[102] and loss of vegetative

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[98] http://www.setac.org/node/1

[99] W. Nelson Beyer, James P. Meador, eds. *Environmental Contaminants in Biota: Interpreting Tissue Concentrations*. 2011. CRC Press, Boca Raton, FL. Online at: http://www.amazon.com/Environmental-Contaminants-Biota-Interpreting-Concentrations/dp/1420084054/ref=sr_1_1?ie=UTF8&qid=1312391692&sr=8-1

[100] Fritzie T. Celino, Sonoko Yamaguchi, Chiemi Miura, Takeshi Miura. "Arsenic inhibits *in vitro* spermatogenesis and induces germ cell apoptosis in Japanese eel (*Anguilla japonica*)." *Reproduction* August 1, 2009. 138 279-287. Online at: www.reproduction-online.org/content/138/2/279.abstract?related-urls=yes&legid=reprod;138/2/279

[101] Gomez-Caminero, P. Howe, M. Hughes, E. Kenyon, D.R. Lewis, M. Moore, J. Ng, and by A. Aitio and G. Becking. "Environmental Health Criteria 224: Arsenic and Arsenic Compounds." Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organization, and the World Health Organization, and produced within the framework of the Inter-Organization Programme for the Sound Management of Chemicals, 2001. Online at: http://whqlibdoc.who.int/ehc/WHO_EHC_224.pdf

[102] Kapustka, L. A., Lipton, J., Galbraith, H., Cacela, D. and Lejeune, K. 1995. "Metal and arsenic impacts to soils, vegetation communities and wildlife habitat in southwest montana uplands contaminated by smelter emissions: II. Laboratory phytotoxicity studies." *Environmental Toxicology and Chemistry* 14: 1905–1912. Online at: http://onlinelibrary.wiley.com/doi/10.1002/etc.5620141112/abstract

cover, which was also accompanied by a reduction in capacity to support indigenous wildlife populations.[103]

Dioxin is a highly toxic compound that is linked to cancer and adverse reproductive impacts in humans. Scientists have also found that high levels of dioxin in fish (rainbow trout and minnows) have severe effects, including reduced reproductive success, slower growth rates, abnormal physiology, lesions, and mortality.[104]

Studies have found that small mammals with high levels of cadmium had adverse health effects that affect survival and fitness, and also experienced kidney damage.[105]

High levels of lead in mammals and birds have been linked to neurotoxic effects, reproductive problems, nephrotoxic and hematological changes, loss of body weight, kidney and brain damage, and death.[106,107]

High levels of selenium have been found to have extremely toxic effects on wildlife. Specifically, studies of birds with high selenium have found reduced egg hatchability, deformities in embryos/chicks, and other reproductive problems.[108,109]

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[103] Galbraith, H.; LeJeune, K.; Lipton, J. "Metal and arsenic impacts to soils, vegetation communities and wildlife habitat in southwest Montana uplands contaminated by smelter emissions: I Field evaluation." Environmental Toxicology and Chemistry, 14, 1895-1903. 1995. Online at: http://onlinelibrary.wiley.com/doi/10.1002/etc.5620141111/abstract

[104] Beyer & Meador, 2011. Chapter 3. "Dioxins, PCBs, and PBDEs in Aquatic Organisms." Richard J. Wenning, Linda Martello, and Anne Prusak-Daniel. p. 103 – 168.

[105] Beyer & Meador, 2011. Chapter 19. "Cadmium in Small Mammals." John. A. Cooke. p. 627-644.

[106] Beyer & Meador, 2011. Chapter 17. "Lead in Mammals." Wei-Chun Ma. p. 595-608.

[107] Beyer & Meador, 2011. Chapter 16. "Lead in Birds." J. Christian Franson and Deborah J. Pain. p. 563-594.

[108] Beyer & Meador, 2011. Chapter 21, "Selenium in Birds". Harry M. Ohlendorf and Gary H. Heinz. 669-702

Similar studies of fish linked high levels of selenium with reproductive failure and mortality.[110]

4. Reducing Mercury Emissions Will Benefit Local Environments

Recent research has confirmed that local sources of mercury emissions contribute the most to local contamination problems. In fact, a 2007 study confirmed the presence of mercury "hot spots" downwind from coal-fired power plants.[111] It is clear reducing mercury emissions from these stacks will greatly benefit downwind waters.

The 2007 hotspot study builds on previous studies confirming that coal-fired power plants within the United States are the primary source of mercury to the Great Lakes and the Chesapeake Bay.[112,113] It is also consistent with a major mercury deposition study conducted by EPA and the University of Michigan that concluded that approximately 70 percent of mercury wet deposition resulted from local fossil fuel emissions in the region.[114]

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[109] Fred L. Paveglio, Christine M. Bunck and Gary H. Heinz. "Selenium and Boron in Aquatic Birds from Central California." *The Journal of Wildlife Management* Vol. 56, No. 1 (Jan., 1992), pp. 31-42 Online at: http://www.jstor.org/pss/3808788
[110] Beyer & Meador, 2011. Chapter 5. "Selenium Accumulation and Toxicity in Freshwater Fishes." David K. DeForest and William J. Adams. P. 193-230.
[111] Evers, David C. et al. 2007. Biological Mercury Hotspots in the Northeastern United States and Southeastern Canada, Bioscience. Vol. 57 No. 1. p. 29
[112] M. Cohen, et al., *Modeling the Atmospheric Transport and Deposition of Mercury to the Great Lakes*, Environmental Research 95, 2004 (247-265).
[113] Mark Cohen, NOAA, *Modeling the Fate and Transport of Atmospheric Mercury in the Chesapeake Bay Region* (May 17, 2004), slide 34. Online at: http://www.arl.noaa.gov/data/web/reports/cohen/20_Ches_Bay_talk.pdf (visited November 20, 2006).
[114] Keeler, G., *et al.* 2006. "Sources of Mercury Wet Deposition in Eastern Ohio, USA," *Environmental Science & Technology*.

5. Economic Benefits of Reducing Environmental Effects of Air Toxics

Numerous studies have indicated that substantial economic benefits would be achieved by reducing environmental exposure to mercury and other HAP. Some of the findings from these studies are listed below:

- The Environmental Protection Agency underestimated by $10.5 billion the annual net benefits of a proposal to set standards for mercury and other air toxics from power plants, according to a Navigant Consulting Inc. report released July 14, 2011.[115] In total, the annual net benefits of the rule would be $52.5 billion to $139.5 billion, said the report, which was sponsored by the Clean Air Council and other organizations.
- The Minnesota Pollution Control Agency and the Legislative Commission on Minnesota Resources estimate the economic benefits of reduced mercury pollution at $212 million. This is based on a 50 percent reduction in regional Midwest emissions, which is projected to reduce mercury deposition in Minnesota by approximately 12 percent.[116] This study clearly understates the total economic benefits of reduced mercury pollution, because it takes into account only Minnesota residents' willingness to pay, and not economic benefits due to factors such as improved public health.
- Researchers from the Harvard School of Public Health estimate the health-related economic impact of mercury pollution at over $5.5 billion. The study estimates the costs of lost productivity due to reduced IQs resulting from mercury pollution at $1.625 billion, the costs of excess mental retardation cases from mercury emissions at approximately $361 million, and the costs of

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[115] *Why EPA's Mercury and Air Toxics Rule is Good for the Economy and America's Workforce*, is available at http://www.cleanair.org/sites/default/files/MercuryRuleEconomicsReport.pdf.

[116] Daniel A. Hagen, et al, *Economic Benefits of Reducing Mercury Deposition in Minnesota* (June 1999), Minnesota Pollution Control Agency report, available at http://www.pca.state.mn.us/index.php/component/option,com_docman/task,doc_view/gid,313.

excess cardiovascular disease from mercury emissions at approximately $3.54 billion.[117]

- In Minnesota, a long-term decline in angling of just 25 percent due to the lake- and stream-fouling effects of mercury could cost the state's economy $706 million per year and jeopardize a portion of the estimated 25,955 jobs in Minnesota that depend on fishing.[118]
- In Wisconsin, a long-term decline in angling of just 25 percent due to the lake- and stream-fouling effects of mercury could cost the state's economy $516 million per year and jeopardize a portion of the estimated 21,459 jobs in Wisconsin that depend on fishing.[119]

Based on these and other similar studies, we urge you to recognize that the pollution emitted by EGUs results in a cost that is being unfairly borne by others. Reducing and in some cases eliminating pollution is therefore an appropriate and necessary shift of the cost back to where it belongs—on the polluting source and unit itself. In addition, the cost-benefit analysis provided by EPA is welcome but insufficient. It includes an important focus on human health but fails to include sufficient assessment and valuation of impacts on wildlife and ecosystems. Not only is it the duty of EPA to prepare such estimates for alternative sources as part of the Biological Assessment EPA must bring to its consultation with the wildlife agencies under the ESA, but the Clean Air Act also requires EPA in general to address the needs of wildlife and ecosystems, in setting secondary standards, for example. Costs and benefits of pollution reductions for wildlife are harder to assess since more research is needed on levels of pollutants in many wildlife species and in assessing

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[117] Paul R. Epstein, et al, *Full cost accounting for the life cycle of coal*, ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, 1219:73-98 (2011), available at http://onlinelibrary.wiley.com/doi/10.1111/j.1749-6632.2010.05890.x/full.
[118] Minnesota Environmental Partnership, Mercury Pollution Threatens Minnesota's Fishing Industry, Press Release, Aug. 17, 2004, available at http://www.mepartnership.org/mep_newsroom.asp?new_id=787.
[119] Clean Wisconsin, 25% Drop in Wisconsin Fishing Due to Mercury Would Cost $516M Annually, Press Release, Aug. 16, 2004, available at http://www.cleanwisconsin.com/index.php?module=cms&page=323.

the full range of the pollutants' impacts on ecosystems. But there has also been much progress in such research, and in recognition of the economic role that ecosystems play. We therefore urge that in this instance, and indeed in all instances where cost-benefit analyses of rules and regulations are involved, such analysis be broadened to include valuation of impacts on wildlife and ecosystems, however incomplete it may be.

6. Duty to Formally Consult with the Fish and Wildlife Service and the National Marine Fisheries Service (NMFS) Regarding Impacts on Endangered and Threatened Species, and the Level at Which Standards Must Be Set to Protect Them

Since endangered, threatened and other protected species such as the Indiana bat, Florida panther, bald eagle, and polar bear have been documented with high levels of mercury, and since the livelihood and reproductive success of these creatures can be affected by this and other hazardous pollutants, EPA is required to consult with the appropriate agencies (FWS and NMFS) to determine the levels at which emissions need to be set not only to ensure that the levels allowed by EPA will not be likely to jeopardize the continued existence and recovery in the wild of these species, as required under Section 7(a)(2) but also to meet EPA's Section 7(a)(1) duty to determine how EPA can use its authority to enhance the recovery of these listed species. In addition, the funding for the research that is needed in this area should come from operating and proposed fossil power plant operators.

C. EPA's new technical analyses reaffirm that it is appropriate and necessary to regulate U.S. Hazardous Air Pollutants ("HAP") from coal- and oil-fired electric utility steam generating units ("EGUs")

EPA's determination in 2000 that it was appropriate and necessary to regulate HAP from U.S. EGU required that the Agency set toxic air pollution standards for these facilities (see *infra*, chapter II). Nonetheless, EPA has conducted new and extensive technical analyses prior to publication of the 2011 proposed rule. These analyses, although not required, reaffirm the 2000 finding that regulation of toxic

air pollution from EGUs under section 112 is appropriate and necessary. Additionally, the new technical analyses incorporate present and future projections of HAP emissions, modern air pollution modeling tools, current control technologies, and updated pollutant emissions regulations. The results clearly demonstrate that not only was the 2000 decision justified at the time, but that 11 years later, the science shows that it is even more critical to regulate HAP emissions from U.S. EGUs.

1. HAP Emissions from U.S. EGUs Continue to Pose Health and Environment Hazards

In the 2011 proposal, three distinct quantitative and qualitative technical analyses were conducted by EPA that determined HAP emissions from U.S. EGUs contributed to the risk of adverse effects to public health or the environment. These were: (i) EPA's national-scale mercury risk assessment; (ii) EPA's case studies of cancer and non-cancer inhalation risks for non-Hg HAP; and (iii) EPA's qualitative assessment of potential environmental risks from deposition of HAP. The results from all three risk analyses conducted indicate that HAP from U.S. EGUs pose hazards to public health and the environment.

a. EPA's National-Scale Mercury ("Hg") Risk Assessment Demonstrates that U.S. EGU Hg Emissions Pose a Hazard to Public Health

First, EPA conducted a comprehensive technical analysis that quantified the potential risk to public health posed by U.S. EGU Hg emissions. The results of the assessment clearly indicate that Hg emitted by U.S. EGUs currently poses a hazard to public health and will continue to do so in the future. This assessment analyzes human exposure to methylmercury ("MeHg") through ingestion of fish from U.S. freshwater lakes and streams and does not quantify adverse effects from the ingestion of MeHg in seafood. Were EPA to take into account consumption of seafood, MeHg consumption in the U.S. is of even greater concern. A recent study focused on estuarine and marine fish consumption in the U.S. finds that seafood

significantly contributes to national exposure to MeHg.[120] This study considers differences in fish intake and geographic variability of seafood. The results indicate that seafood consumption under certain conditions can cause exposures to women of child-bearing age above EPA's reference dose (RfD) for MeHg.[121] Subpopulations with heavy intakes of regional seafood may also be at risk. For example, one study on recreational anglers finds that these subpopulations have high levels of exposure to MeHg, which the study largely attributed to recreationally caught seafood.[122] Results from a different study indicate that women of child-bearing age living in coastal areas are at considerably higher risk of having blood Hg concentrations exceeding acceptable health-based levels due to higher consumption of fish and shellfish.[123] Although a much smaller effect on seafood MeHg levels is anticipated from this rule compared to that in freshwater fish, emissions from U.S. EGUs nevertheless contribute to the global problem of marine MeHg bioaccumulation. Ingestion of seafood could further increase the impact of Hg emissions from EGUs on public health beyond that estimated from an analysis based exclusively on consumption of freshwater fish.

EPA's determination that Hg emissions pose a risk to public health is based on a MeHg reference dose (RfD) of 0.1 μg/kg per day. 76 Fed. Reg. at 25,000. The Rfd for MeHg is the daily amount of MeHg ingestion expected not to produce adverse health effects to the most sensitive subpopulations over a lifetime. The

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[120] Elsie M. Sunderland, *Mercury Exposure from Domestic and Imported Estuarine and Marine Fish in the U.S. Seafood Market*, 115 Envtl. Health Persp. 239 (2007), available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1817718/.
[121] *Id.* at 241.
[122] Rebecca A. Lincoln et al., *Fish Consumption and Mercury Exposure among Louisiana Recreational Anglers*, 119 Envtl. Health Persp. 248-249 (2011), available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3040613/.
[123] Kathryn R. Mahaffey et al., *Adult Women's Blood Mercury Concentrations Vary Regionally in the United States: Association with Patterns of Fish Consumption (NHANES 1999–2004)*, 117 Envtl. Health Persp. 51 (2009), available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2627864/.

utility industry has argued that this RfD is too conservative.[124] Rather than being too conservative, this RfD is based on sound science. Further, Section 112 of the Clean Air Act acknowledges the particular danger posed by the congressionally-listed HAPs and takes an intentionally conservative approach to protecting human health. For example, when Congress passed the Clean Air Amendments of 1990, it shifted the burden of proving a substance's health hazard from those who support regulation to those that oppose regulation. See Section 112(b)(3) (requiring those who wish to remove a substance from the congressionally-created list of HAPs to provide "adequate data . . . [showing] the substance may not reasonably be anticipated to cause any adverse effects to human health or adverse environmental effects"). Given the overall emphasis on protecting human health, if faced with two alternatives methods of determining RfD, it is reasonable for the Agency to choose the method that is more conservative with respect to human health. Thus, EPA has applied an appropriate RfD in its analysis based Faroe Islands study, a decison supported by the findings of the National Academy of Sciences' Study on Mercury. 76 Fed. Reg. at 25,000.

In the 2000 determination, some utility groups took issue with EPA's reliance on a Faroe Islands study to determine a proper RfD.[125] This industry argument was largely based on the fact that (1) Faroe Islanders were exposed to high levels of polychlorinated biphenyl in addition to mercury and (2) the diet of Faroe Islanders differed from that of Americans.[126] However, this argument fails to acknowledge that the findings of the Faroe Island study were consistent with a smaller study conducted in New Zealand (0.1 μg/kg-day) for which neither of these issues was present. 76 Fed. Reg. at 25,000. Further, analyses done since 2000 integrating data from both the Faroe Island and New Zealand studies with a third study conducted

[124] Comments of Utility Air Regulatory Group, Docket ID No. OAR-2002-0056, at 16-18 (June 29, 2004).
[125] *Id.*
[126] *Id.*

in the Seychelles demonstrate a significant relationship between prenatal MeHg exposure and neurobehavioral deficits.[127]

Since the 2000 Finding, additional studies from Poland and the United States support the conclusion of the New Zealand, Faroe Island, and Seychelles studies, and find that there is a negative correlation between maternal mercury levels and neurological development.[128] These more recent studies, also find a clear associations between maternal blood Hg levels and delayed child development using new cohorts based on urban populations.

Finally, the RfD applied by EPA in this assessment is based on neurobehavioral test results. Other potential health impacts of prenatal mercury exposure have been identified. For example, an association between cardiovascular effects and MeHg exposure has been reported. A recent study finds significant associations between mercury exposure and indicators of cardiovascular disease.[129] Additionally, the potential effects of co-pollutants, those derived from exposures to pollutant mixtures, related to MeHg are ignored in the Agency's assessment. One recent study finds that such an omission may lead to additional health effect associated with MeHg exposure being overlooked.[130] This study identifies health

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[127] Daniel A. Axelrad, *Dose-Response Relationship of Prenatal Mercury Exposure and IQ: An Integrative Analysis of Epidemiologic Data*, 115 Envtl. Health Persp. 609 (2007), available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1852694/.
[128] See Wieslaw Jedrychowski et al., *Effects of Prenatal Exposure to Mercury on Cognitive and Psychomotor Function in One-Year-Old Infants: Epidemiologic Cohort Study in Poland*, 16 Annals of Epidemiology 439 (2006), abstract available at http://www.mendeley.com/research/effects-prenatal-exposure-mercury-cognitive-psychomotor-function-oneyearold-infants-epidemiologic-cohort-study-poland/; S.A. Lederman et al., *Relation Between Cord Blood Mercury Levels and Early Child Development in a World Trade Center Cohort*, 116 Envtl. Health Persp. 1085 (2008), available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2516590/.
[129] Anna L. Choi et al., *Methylmercury Exposure and Adverse Cardiovascular Effects in Faroese Whaling Men*, 117 Envtl. Health Persp. 367 (2009), available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2661905/.
[130] Takasi Yorifuji et al., *Parental Exposure to lead and cognitive deficit in 7- and 14-Year-Old Children in Presence of Concomitant Exposure to Similar Molar Concentration of Methylmercury*, 33 Neurotoxicology & Teratology 205 (2011),

effects associated with prenatal lead exposure when simultaneously exposed to MeHg.

EPA's national-scale assessment unequivocally identifies public health risks associated with Hg emissions from U.S. EGUs. The assessment results indicate that:

- EGU-attributable MeHg poses a hazard to public health at 28 percent of watersheds analyzed when considering all sources of Hg deposition. 76 Fed. Reg. at 25,010.
- Hg deposition attributable to U.S. EGUs alone poses a health risk at 12 percent of the watersheds analyzed without considering any other Hg source. *Id.*
- The contribution of U.S. EGUs to total Hg deposition may be greater than 10 percent at hundreds of watersheds. *Id.* at 25,009.

Additionally, the quantitative and qualitative sensitivity analyses completed by EPA for the most significant uncertainties in the analysis indicate that these uncertainties should not alter the Agency's conclusions. *Id.* at 25,011. EPA further acknowledges that limitations in the availability of fish tissue MeHg data could be leading to a substantial underestimation of the number of watersheds where populations are at risk of exposure to EGU-attributable MeHg. Thus, the Agency reasonably determined that mercury emissions pose a public health hazard, and regulation of EGUs is appropriate. EPA has correctly requested peer-review of national-scale risk analysis. The Agency will consider the results of the peer review prior to the final rule publication and make these available along with the Agency's response.

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abstract available at
http://www.sciencedirect.com/science/article/pii/S0892036210001686.

b. EPA's Case Studies For Non-Hg HAP Demonstrate That Non-Hg HAP from U.S. EGUs Pose a Hazard to Public Health

Although the risk to public health and the environment posed by Hg is sufficient to require regulation of HAP emissions from EGUs (see chapter 2, *infra*), EPA's risk assessment for non-Hg HAP demonstrates additional potential public health impacts and clearly calls for regulation. EPA conducted 16 test cases quantifying the health risk from chronic inhalation of non-Hg HAP from EGUs. Over 40 percent of the case studies conducted by EPA to quantify health hazards associated with the inhalation of non-Hg HAP indicated a cancer risk greater than or equal to the one in one million threshold level threshold required to delist a source as a category regulated under section 112 of the Clean Air Act. 76 Fed. Reg. at 25,011; *see also* Section 112(c)(9)(B) (indicates that "no source in the category . . . emits [carcinogenic] air pollutants in quantities which may cause a lifetime risk of cancer greater than one in one million to the individual in the population who is most exposed to emissions of such pollutants from the source."). The case study examining cancer risk from an oil-fired EGU indicated that the greatest cancer risk estimate quantified in the assessment, ten in one million, is associated with this source. 76 Fed. Reg. at 25,012. This reaffirms the need to regulate HAP emitted by both coal- and oil-fired EGUs.

In addition to other metallic HAP, there are several acid gases emitted by EGUs that also support regulation which were not included in the test cases. Studies of these acid gases were not included due to uncertainties in their emission rates. Potential overlapping impacts from different EGUs and other pollutant sources were also not quantified. All of these additional factors likely compound the cancer risks estimated in the case studies. We agree with EPA's decision to only peer-review the speciation of chromium and nickel in the analysis of the health risks posed by non-Hg EGU emissions. The AERMOD modeling system methodology applied in EPA's non-Hg HAP chronic inhalation test cases is well established and has undergone numerous evaluations. For example, one recent

comparative study finds that AERMOD produces the most reliable exposure risk simulation results among four different common exposure assessment methods.[131]

c. EPA's Qualitative Assessment of Potential Environmental HAP Impacts Indicates that HAP Emissions from U.S. EGUs Pose a Hazard to the Environment

When it made the 2000 Finding, EPA included evidence of adverse effects of mercury on the environment, which has been further supported by newer studies. Several of these newer studies are included in a special issue published by the journal *Ecotoxicology* in 2008 devoted to the effects of MeHg on wildlife.[132] Although EPA has not quantified the potential impacts of HAP from U.S. EGUs on the environment, a qualitative assessment conducted by the Agency reviewed existing literature reporting effects of Hg on fish and wildlife and acid gas contribution to ecosystem acidification. The potential adverse environmental effects associated with HAP are well documented and have been previously described in section (B) of this chapter. This qualitative assessment reaffirms EPA's determination that it is appropriate and necessary to regulate HAP emissions from EGUs.

2. EPA's Analyses Demonstrate that EGUs Remain the Largest Source of Hg, Hydrogen Chloride ("HCl"), Hydrogen Fluoride ("HF"), and Selenium, ("Se") in the U.S., as Well as a Major Source of Hydrogen Cyanide ("HCN") and Other Metallic HAP

The technical analyses conducted by EPA demonstrate that EGUs remain the largest source of Hg in the United States, accounting for approximately half of U.S. anthropogenic Hg emissions. 76 Fed. Reg. at 25,002. Hg emissions from EGUs are

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[131] Bin Zou, *How should environmental exposure risk be assessed? A comparison of four methods for exposure assessment of air pollutions*, 166 Envtl. Monitoring & Assessment. 166 (2010), abstract available at http://www.springerlink.com/content/a68l242m75215377/.

[132] Special Issue on Effects of Methyl Mercury on Wildlife, 17 Ecotoxicology 67–171 (2008), available at http://www.springerlink.com/content/vtj02mn13516/?p=8f4430c82b2e48c3af1dbabf5799fdb6&pi=2

projected to remain relatively unchanged through 2016 and then be over six times larger than those from the second largest contributing source. *Id.* Additionally, the significant fractions of oxidized and particulate Hg in Hg emissions from EGUs make these emissions more relevant to local and regional deposition than natural emissions comprised of elemental Hg. In 2005, EGUs accounted for 82 percent of HCl emissions, 62 percent of HF emissions, 8 percent of HCN emissions, and 13 to 83 percent of several non-Hg metallic HAP emissions in the U.S. *Id.* at 25,005.

a. EPA's National-Scale Hg Risk Assessment Demonstrates that EGUs Can Significantly Contribute to Hg Deposition

EPA's national-scale Hg risk assessment demonstrates that U.S. EGUs can significantly contribute to watershed Hg deposition. High variability in the contribution of U.S. EGUs to total Hg deposition is evident from the analysis, *id.* at 25,008. EPA's modeling results indicate that the fraction of total Hg deposition attributable to EGUs is greater than 10 percent at hundreds of watersheds., *id.* at 25,009. As previously discussed, the Hg deposition attributable to U.S. EGUs alone may endanger sensitive populations near many of these watersheds. Thus, there are many areas of the nation for which EGUs are a significant contributor to potential Hg hazards to public health and the environment.

b. EPA's Local Hg Deposition Analysis Demonstrates that Areas Close to EGUs are at Greater Risk for Adverse Public Health and Environmental Effects

EPA has also demonstrated that deposition of Hg emitted by EGUs can be significantly larger at locations close to emission sources. The Agency's assessment of potential for deposition "hotspots" shows that Hg deposition near EGUs can be three times as large as the regional average. *Id.* at 25,013. This excess Hg deposition would substantially increase the health and environmental risks associated with emissions at these sites. Furthermore, the methodology applied by EPA to quantify near-source Hg deposition is conservative. In EPA's analysis, the average local deposition is estimated from the area within 50 km of the EGU source.

This method averages local Hg deposition across a large area (over 7500 km2). Maximum excess local Hg deposition may be significantly underestimated by averaging high deposition sites downwind of an EGU in the direction of prevailing winds with lower excess deposition at locations close to but frequently upwind of the facility. A more rigorous alternative approach would be to identify potential "hotspots" from the modeled Hg deposition at individual grid cells within the modeling domain. For this technical analysis, the Community Multiscale Air Quality model used by EPA estimates Hg deposition individually for 144 km2 extensions of land corresponding to the 12 km model resolution selected.[133] The use of individual 144 km¬2 grid cells to quantify local deposition could increase the excesses Hg deposition at these locations significantly and place them at even greater risk of adverse health and environmental effects of HAP from U.S. EGUs. Though this alternative methodology might indicate the likelihood of much higher concentrations, EPA's methodology nonetheless quite clearly demonstrates that excess Hg deposition occurs in the vicinity of EGUs and is especially significant around the largest Hg emitters.

3. EPA's Analysis of Control Technologies for EGU HAP Demonstrates that Significant Emissions Reductions Can be Achieved with Available Controls

In its analysis, EPA identified the current control technologies available for HAP from U.S. EGUs and estimated the controls' expected impact on national total and category-specific emissions. The Agency's analysis identifies multiple available control technologies that would enable EGUs to comply with regulations, including electrostatic precipitators, fabric filters, flue gas desulfurization, activated carbon injection, and dry sorbent injection. Furthermore, significant reductions to Hg emissions may already be occurring at EGUs that have installed controls intended for other pollutants, such as particulate matter or sulfur oxides. 76 Fed. Reg. at 25,017. Available control technologies for metal HAP and acid gases were also

[133] Regulatory Impact Analysis of the Proposed Toxics Rule, Docket ID No. OAR-2011-0044, at 3-2 (March, 2011).

identified by EPA. As for Hg, the analysis finds that effective control of non-Hg HAP may be achieved as a co-benefit of particulate matter and sulfur oxides controls.

EPA's emissions projections clearly show that vast reductions to emitted HAP would result from this rule. Compliance with the rule is estimated to result in 79 and 91 percent reductions to U.S. EGU Hg and hydrochloric acid respectively. The reductions are also equivalent to 36 and 49 percent of national anthropogenic Hg and hydrochloric acid emissions respectively. The reduction in metallic HAP is projected to equal approximately 430 tons per year. These great reductions to U.S. HAP expected to result from this rule incontestably demonstrate that the proposed rule will produce effective results.

4. EPA's New Technical Analysis Provides Compelling Evidence that Demonstrates it is Appropriate and Necessary to Regulate HAP from U.S. EGUs

We commend EPA on the extensive technical analyses undertaken by the Agency prior to publication of this proposed rule. The science behind EPA's new technical analyses is comprehensive and robust. The technical analyses clearly demonstrate that HAP form U.S. EGUs pose a risk to public health and the environment. The new analysis reaffirms that utilities are the largest domestic source of Hg and a major source of non-Hg HAP. Proven effective control technologies for EGU HAP are available. The analyses incorporate updated data, newly published information, and current modeling tools. The Agency's assessments clearly show that HAP from U.S. EGUs pose hazards to public health and the environment after considering the requirements of the Clean Air Act.