American Academy of Pediatrics ▪ American Heart Association
American Lung Association ▪ Asthma and Allergy Foundation of America
American Public Health Association ▪ American Thoracic Society
National Association of County and City Health Officials
Physicians for Social Responsibility

August 4, 2011

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

RE: Docket ID Nos. EPA-HQ-OAR-2009-0234 and EPA-HQ-OAR-2011-0044

Dear Administrator Jackson:

On behalf of our nation's medical and public health groups, we urge the U.S. Environmental Protection Agency to adopt the strongest possible standards to reduce mercury and air toxics from coal- and oil-fired power plants by November 16, 2011. As health and medical professionals who treat patients impacted by lung, cardiovascular, and neurological impairments, we are keenly aware of the harmful health effects of air pollution. Research has shown that these toxics 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. Our organizations call on the EPA to close the two-decade old loophole that has allowed power plants to avoid having to clean up, unlike all other industries. The cleanup of toxic air pollution from power plants is necessary for the protection of public health, appropriate for the EPA to undertake, and long overdue.

Hazardous Air Pollutants from Power Plants Cause Wide-ranging Health Harm

During the process of burning coal and oil, 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 hazardous air pollutants that harm human health include: corrosive substances (acid gases, such as hydrogen chloride

and hydrogen fluoride); carcinogens (formaldehyde, benzene, toluene, and other compounds); organic carbon-based toxics (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) (EPA 2007; ATSDR 2011a).

Coal-fired power plants produce over 386,000 tons of 84 separate hazardous air pollutants from over 440 plants in 46 states, according to the most current data in the National Emissions Inventory (EPA, 2007). These plants produce 40 percent of all hazardous air pollutants released from industrial sources into the atmosphere, more than any other industrial pollution source. In addition, the combustion of coal to generate electricity also produces 76 percent of the total volume of acid gases, 60 percent of arsenic, and 46 percent of mercury released into the atmosphere (EPA 2007).

Some hazardous pollutants, such as acid gases, mercury, and sulfur dioxide, have immediate impacts on people, neighborhoods, and towns located near power plants. However, other pollutants, such as dioxins and metals, can travel much farther from the pollution source. When they adhere to fine particles, these pollutants can remain in the air for more than a week and be carried away by winds to distant locations. This makes toxic air pollution dangerous to public health and human health near and far from coal-fired power plants (EPA 2009).

We agree with the strong evidence the EPA provides to support their decision that the proposed rule is both appropriate and necessary to protect public health as required under Section 112 of the Clean Air Act. Exposure to likely harm from mercury and methylmercury continues, as does strong evidence of exposure to multiple, recognized carcinogens and other toxics that cause or increase risk of cardiovascular, respiratory, and other acute and chronic systemic damage.

The discussion below summarizes the evidence that these toxics pose serious threats to health and must be reduced.

Acid Gases (Examples: 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 (EPA, 2007). 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 to further travel to the alveolar regions of the lung (EPA, 1998). Chlorine is another toxic acid gas produced in coal combustion (EPA 2011).

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 were associated with asthma and related symptoms in children (Raizenne, 1996; Dockery 1996). Another

recent study also found that acid gases and particle pollution were associated with reduced lung function (Gauderman et al., 2004).

Hydrogen chloride (HCl)

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 Airway Dysfunction Syndrome (RADS). (ATSDR, 2010a). Both hydrogen chloride and hydrogen fluoride can irritate the eyes, nasal passages, and lungs (EPA 2000a, 2000b).

Hydrogen fluoride (HF)

Colorless hydrogen fluoride gas poses serious health risks when inhaled, thanks to what the Agency for Toxic Substances and Disease Registry describes as the fluoride ion's ~~"a~~ggressive, destructive penetration of tissues." (ATSDR, 2003b). Hydrogen fluoride irritates the nose, throat, and eyes, inflames the mucous membrane, causes coughing, and narrows 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. (ATSDR, 2003b).

Chlorine (Cl)

Chlorine is a highly reactive gas, usually broken down within minutes in the outside environment. At high concentrations, however, chlorine can damage the body, with the severity of symptoms varying with the duration of exposure to the gas and exposure concentration. Inhalation of small 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 much 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). Exposure to high concentrations of chlorine have also been linked to long-term neurological effects, such as memory loss, slow reaction time, impaired balance, hearing loss, and visual alterations (ATSDR, 2010c).

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. (Oh et al., 2001). As particles, they may remain airborne for more than ten days, spreading widely from their source, and depositing in water and soil (Atkinson, 1991). Dioxins have 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 (Hoffman et al., 2006). Researchers have found dioxins in the breast milk of nursing mothers (Lorber and Phillips, 2002).

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 carcinogen. (NTP 2011). (WHO, 2010, 2011; ATSDR, 1994, 1998a, 2000b). 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 (Casals-Casas and Desvergne, 2011). Last year, the World Health Organization concluded that the developing fetus and the newborn child were the most vulnerable to dioxin and furan exposure because of the rapid growth of their organ systems (WHO, 2010).

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 (ATSDR, 1990). Radioisotopes are known carcinogens, especially as relates to the lungs, bones, and lymphatic system. They can also cause kidney disease, pneumonia, anemia, and brain abscess (ATSDR, 1990, 2011, 2011b; WHO, 2011).

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, including motor vehicle exhaust, and from tobacco smoke and food. Fifteen PAH compounds are considered reasonably anticipated to be human carcinogens (HHS, 2011). In the air, PAHs adhere to particulate matter and deposit in the lungs (ATSDR, 1995 and 2011; Vineis P and Husgfvel-Purisainen K, 2005). New research warns that prenatal exposure to PAHs may impact the mental development of children to age 5 (Perara FP et al., 2009) (ATSDR, 1995; 2005a; WHO, 2011).

Volatile Organic Compounds (Examples: acetaldehyde, benzene, formaldehyde, toluene, xylene)

Volatile organic compounds take in a host of chemicals that include carcinogens and other toxics and also contribute to the formation of ozone. According to the 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 carcinogens, while acetaldehyde is considered a probable carcinogen (HHS, 2011). Noncancer 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, impair memory, and slow response to visual stimuli (ATSDR, 1999a, 2000a, 2007a, 2007b, 2010b, 2011; WHO 2011).

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 (EPA, 2011) Microorganisms convert mercury into methylmercury, a highly toxic form of mercury that bioaccumulates in fish and shellfish (ATSDR, 1999b; Grandjean, 2010). 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 (EPA, 1997).

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 (ATSDR 1999b). Mercury exposure can lead to developmental birth defects and interfere with neurological development (Bose-O'Reilly et al., 2010). Pregnant women who consume fish and shellfish can transmit that methylmercury to their developing fetuses, and infants can ingest methylmercury in breast milk. Children can also become exposed by eating contaminated fish (ATSDR 1999). 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 (Trasande et al., 2005; Axelrad et al., 2007). Mercury can damage the kidneys, liver, brain, and nervous system as a potent neurotoxin, even in adults (ATSDR, 1999b, 2011a; WHO, 2011). A recent study has also found that methylmercury exposure may lessen the cardiovascular benefits of regular fish consumption (Domingo, 2007).

Non-Mercury Metals

Non-mercury metals and metal-like substances (e.g. arsenic and selenium) comprise a significant part of fine particulate matter ($PM_{2.5}$) emitted from coal-fired power plants. These primary particles come in addition to the secondary particles formed as a result of chemical reactions in sulfur dioxide and nitrogen oxide emissions. Those secondary particles, notably sulfates and nitrates, pose similar life-threatening risks.

Inhaled particles deposit along the respiratory tract or penetrate deeply into the gas-exchange region of the lung. The EPA has already concluded that exposure to fine particulate matter ($PM_{2.5}$) causes cardiovascular effects and premature mortality and is likely to cause respiratory harm. They concluded that 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).

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) (EPA 2009). Evidence has also grown to warn that long-term exposure to $PM_{2.5}$ can increase the risk of low birth weight and infant mortality, as well as cancer, especially lung cancer (EPA, 2009).

The level of toxicity of fine particles varies and is likely impacted by the presence of metals or other pollutants (Bell et al., 2007). Metals interact with particles to create ~~"r~~reactive oxygen species" which limit the body's ability to repair damage to its cells and contribute to tissue inflammation (Carter et al., 1997; Gurgueira et al., 2002; Wilson et al., 2002). Research has shown that sulfate, selenium, iron, nitrate, and organic carbon affect immune cell response and heart variability (Huang et al., 2003; Chuang et al., 2007). 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 (Zanobetti et al., 2009). 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 high levels of arsenic, organic carbon, and sulfate in PM—potential indicators of coal combustion—were associated with increased hospital admissions for diabetics (Zanobetti et al., 2009). A large study of 25 U.S. communities found more deaths when the fraction of aluminum, sulfate, and nickel in PM was highest (Franklin et al., 2008). This study found additional evidence warning that the combination of metals in particles, a common occurrence, may increase their toxicity.

<u>Arsenic</u>

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 (HHS, 2011). 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 and conjunctivitis, and damage nasal tissue (ATSDR, 1998b). Similar to effects of inhaling arsenic, arsenic in drinking water has also been linked to skin, bladder, lung, and liver cancer (HHS, 2011). Over a long period, the metal-like substance can result in anemia, lesions, liver, kidney, and nerve damage, and affect the digestive system (ATSDR, 2007c).

<u>Beryllium</u>

Beryllium is a known carcinogenic metal (HHS, 2011). Inhaled beryllium has been found to increase the risk of lung cancer (Steenland and Ward 1991, Ward et al., 1992). Breathing large amounts of beryllium compounds can damage the lungs and cause the lungs to resemble pneumonia with reddening and swelling. Long-term exposure over many years may cause chronic beryllium disease, when a chronic inflammatory reaction, called a granuloma, within people who are allergic to beryllium occurs. People with chronic beryllium disease may experience weakness, fatigue, difficulty breathing, anorexia, weight loss, and blueness of the hands and feet. The disease can lead to heart enlargement, heart disease, and even death (ATSDR, 2002).

Cadmium

Cadmium is another known carcinogenic metal (HHS, 2011). Exposure to airborne cadmium causes lung cancer, as the International Agency for Research on Cancer reaffirmed in 2009 (Straif, et al., 2009). Prolonged inhalation of cadmium can also lead to gradual accumulation of the metal in the kidneys, resulting in kidney disease (ATSDR, 2008a).

Chromium

Chromium occurs in three main forms, one of which, chromium (IV) is a known carcinogen that can increase the risk of lung cancer (ATSDR, 2008; HHS, 2011). The metal primarily affects the respiratory system, though chromium (VI) can 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 some workers, inhaled chromium (VI) caused them to develop asthma and have asthma attacks (ATSDR, 2008.).

Lead

The health effects of lead exposure mainly focus on the nervous system and damaging its functions. Lead may cause weaknesses in the joints, anemia, and increases in blood pressure (ATSDR, 2007d). Although lead is harmful to both adults and children, children are most susceptible to the effects of lead exposure. Lead exposure can cause developmental disorders whose effects can persist beyond childhood (ATSDR, 2007e). 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 (HHS, 2011).

Manganese

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 (ATSDR, 2008c). High exposure levels may result in feelings of lethargy and weakness, psychological impacts, and tremors.

Nickel
Compounds containing nickel have been determined to be carcinogenic (HHS, 2011). A known health effect of nickel exposure is the increased risk of lung and nasal cancers from nickel dust (ATSDR, 2005b).

Selenium
Selenium exposure can harm the respiratory system by irritating mucous membranes and causing pneumonia, bronchitis, and pulmonary edema (ATSDR, 2003a). One selenium compound, selenium sulfide, is also considered to be a probable human carcinogen (HHS, 2011).

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. Although particulate matter is not listed as a hazardous air pollutant under Section 112, the pollutants in the sulfur dioxide and nitrogen oxide emissions from power plants will reduce these secondary particles because of the changes expected to meet limits for other listed air toxics. Measures that will reduce acid gases will reduce sulfur dioxide and consequently, reduce the burden of sulfate particles across the nation. The 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 in the summer months. Nitrogen oxides form nitrates, which are the third largest source of $PM_{2.5}$ (EPA RIA, 2011).

As discussed earlier, the evidence shows that $PM_{2.5}$ causes cardiovascular harm and premature mortality and is likely to cause respiratory harm. $PM_{2.5}$ was found to cause premature death from cardiovascular effects and is likely to cause respiratory effects as well. 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).

Millions of people face higher risk

Many face greater risk because of their age, health conditions, or rate of exposure to the pollutants. They include: infants, children and teenagers; older adults; pregnant women; people with asthma and other lung diseases; people with cardiovascular diseases; diabetics; people with low incomes; and people who work or exercise outdoors (EPA 1997, 2009). 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 eighty percent of their lungs between birth and adolescence. The early postnatal

period is when these delicate, growing tissues are at greatest risk. Children also breathe more rapidly, and tend to spend more time outdoors than adults, which exposes them to more pollutants. (American Academy of Pediatrics, 2004).

Even before birth, children face increased risk. As noted earlier, fetuses, infants, and children face impaired neurological development and cognitive abilities, memory, and language skills because of the toxic effects of methylmercury exposure. Dioxins and furans threaten the 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, limited information on toxicity and use of models that do not consider the potential for increased risk for children. (American Academy of Pediatrics, 2004).

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

- Asthma - 24.6 million people, including 7.0 million under age 18 (American Lung Association, 2011)
- Cardiovascular diseases – 82.6 million people (Roger et al., 2011)
- Diabetes – 25.8 million people (CDC, 2011)
- Chronic Obstructive Pulmonary Disease(COPD)—12.1 million adults age 18 and older (American Lung Association, 2010)

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

People who have low incomes or are members of racial and ethnic minorities bear a disproportionate burden of the health effects of air pollution. Because they are more likely to live closer to industrial facilities and high traffic areas, low-income and minority populations are at much higher risk of exposure to the most harmful pollutants (Levy et al., 2002; O'Neill et al., 2003). One study found that 68 percent of African Americans lived within 30 miles of a coal-fired power plant (Georgia Coalition for the Peoples' Agenda et al., 2002). Another study of five power plants in the Washington, DC area found that African Americans and those with less than a high school education were among the groups hardest hit by pollution from the power 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 with less than a high school education (Levy et al., 2002).

Since low income people may be likely to fish in public waterways to save money on food costs, they may be disproportionately exposed to methylmercury. As an example, attached to this letter are maps of the Southeastern states, showing the proximity between concentrations of low income people, including

both white and African-Americans, and the location of coal-fired power plants. The close proximity of these plants to waterways near their homes makes it more likely that fish from those waterways would be in their diet (see Figures 1 and 2 in the attachments).

EPA has provided some evidence of the benefits associated with reducing these toxic emissions. They estimate profound health benefits each year by 2016, including these:

- 6,800 to 17,000 lives saved;
- 11,000 nonfatal heart attacks avoided;
- 12,200 hospital and energy room visits averted;
- 120,000 asthma attacks prevented;
- 11,000 cases of acute bronchitis and 4,500 cases of chronic bronchitis prevented;
- 850,000 days when people won't miss work because of illness; and
- 5.1 million days when people won't miss out on their normal activities because of health problems (EPA, 2011).

In fact, these estimates undercount the total benefits. Studies that would enable researchers to quantify many health endpoints affected by these toxics were not available for modeling. For example, critical, real benefits such as reductions in the number of infants born with low birthweight or impaired cognitive development were not included in the projections. However, the benefits to public health that can be estimated alone provide powerful support to require facilities to meet the strongest possible limits on these toxic emissions. The evidence shows that the strongest possible limits on the emissions of these toxics are both appropriate and necessary.

Toxic Air Pollution Control Technologies Exist

A range of widely available, technical and economically feasible practices, technologies, and compliance strategies are available to power plants to meet the emissions limits and clean up hazardous air pollutants. These technologies already exist and are currently in use at facilities around the country. Power plants will have the flexibility to select the most cost-effective, facility-specific strategies to reduce pollutants in their emissions. EPA estimates that the benefits of reducing these toxic emissions will far outweigh the costs by anywhere from $5 to $13 in benefits for every $1 in costs depending on the assumptions (EPA, 2011).

Conclusion

The adoption of the safeguards against toxic air pollution from power plants, as required under the Clean Air Act, will protect our patients and communities from life-threatening pollution and prevent tens of

thousands of cases of illness and even premature death. Curbing the different types of toxic pollution will yield tremendous benefits and significantly reduce adverse health effects.

The nation needs the EPA to adopt a strong Mercury and Air Toxics standard to effectively protect the health of our patients and our communities. With these new limits on toxic pollutants, the electric power industry will be held accountable to the same standard as other industries and will no longer be allowed to emit pollutants that are hazardous to human health. For over 20 years, electric utilities have avoided requirements to clean up toxic pollutants as set in the Clean Air Act Amendments of 1990. With the first ever federal limits on air toxics from power plants, the EPA has a historic and momentous opportunity to clean the air of notoriously harmful pollutants that endanger human health. Our organizations call on the EPA to adopt a strong, final Mercury and Air Toxics Rule by November 16, 2011and give our patients and communities the clean air they deserve.

Sincerely,

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Attachments Page

Abstracts of Studies Cited in the Comments

arranged alphabetically by first author's last name

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Atmospheric lifetimes of dibenzo-p-dioxins and dibenzofurans.

Atkinson R.

Statewide Air Pollution Research Center, University of California, Riverside 92521.

Abstract

The experimental and theoretical data available concerning the gas- and particle-phase reactions of polychlorodibenzo-p-dioxins (PCDDs) and polychlorodibenzofurans (PCDFs) are discussed. These data lead to the expectation that the dominant tropospheric loss processes of gas-phase PCDDs and PCDFs will be photolysis and reaction with the OH radical, with the OH radical reaction being the most important for the less chlorinated species. The estimated tropospheric lifetimes of gas-phase PCDFs increase significantly more rapidly with the degree of chlorination than is the case for PCDDs. For particle-associated PCDDs and PCDFs, the dominant tropospheric removal processes are expected to be photolysis and wet and dry deposition, with wet and dry deposition of the host particles being the most important. The estimated lifetimes in the lower troposphere range from less than 1 day for dibenzo-p-dioxin, the mono-, di- and trichlorodibenzo-p-dioxins, dibenzofuran and the monochlorodibenzofurans present in the gas phase, to greater than or equal to 10 days for particle-associated PCDDs and PCDFs, with a general increase in the tropospheric lifetime with the degree of chlorination. While long-range transport of PCDDs is expected to occur for those PCDDs which are totally or mainly particle associated, gas- and particle-phase PCDFs containing four or more chlorine atoms are also expected to have sufficiently long tropospheric lifetimes to undergo long-range transport.

Toxicol Appl Pharmacol. 1997 Oct;146(2):180-8.

Cytokine production by human airway epithelial cells after exposure to an air pollution particle is metal-dependent.

Carter JD, Ghio AJ, Samet JM, Devlin RB.

National Health and Environmental Effects Research Laboratory, Environmental Protection Agency, Research Triangle Park, North Carolina 27711, USA.

Abstract

Despite the many epidemiological studies supporting the contention that ambient air pollution particles can adversely affect human health, there is no clear agreement as to a biologically plausible mechanism which can explain the acute mortality and morbidity associated with exposure to particles less than 10 microm in size. We tested the hypothesis that metals present in an air pollution particle can induce the synthesis and expression of the inflammatory cytokines IL-8, IL-6, and TNFalpha. A residual oil fly ash (ROFA) containing the transition metals vanadium, nickel, and iron was used as a model emission source air pollution particle. Normal human bronchial epithelial (NHBE) cells were exposed for either 2 or 24 hr to 0, 5, 50, or 200 microg/ml ROFA. Concentrations of

IL-8, IL-6, and TNF-alpha proteins were measured with commercially available ELISA kits. mRNA for these same cytokines was quantified by RT-PCR. NHBE cells exposed to ROFA produced significant amounts of IL-8, IL-6, and TNF, as well as mRNAs coding for these cytokines. Cytokine production was inhibited by the inclusion of either the metal chelator deferoxamine (1.0 mM) or the free radical scavenger dimethylthiourea (1.0 mM). In addition, vanadium containing compounds, but not iron or nickel sulfates, mimicked the effects of intact ROFA. These results demonstrate that metals present in ROFA may be responsible for production and release of inflammatory mediators by the respiratory tract epithelium and suggest that these mediators may contribute to the toxic effects of particulate air pollutants reported in epidemiology studies.

Annu Rev Physiol. 2011 Mar 17;73:135-62.
Endocrine disruptors: from endocrine to metabolic disruption.
Casals-Casas C, Desvergne B.

Center for Integrative Genomics, Faculty of Biology and Medicine, University of Lausanne, Switzerland.

Abstract
Synthetic chemicals currently used in a variety of industrial and agricultural applications are leading to widespread contamination of the environment. Even though the intended uses of pesticides, plasticizers, antimicrobials, and flame retardants are beneficial, effects on human health are a global concern. These so-called endocrine-disrupting chemicals (EDCs) can disrupt hormonal balance and result in developmental and reproductive abnormalities. New in vitro, in vivo, and epidemiological studies link human EDC exposure with obesity, metabolic syndrome, and type 2 diabetes. Here we review the main chemical compounds that may contribute to metabolic disruption. We then present their demonstrated or suggested mechanisms of action with respect to nuclear receptor signaling. Finally, we discuss the difficulties of fairly assessing the risks linked to EDC exposure, including developmental exposure, problems of high- and low-dose exposure, and the complexity of current chemical environments.

J Occup Environ Med. 2007 Jun;49(6):610-7.
Associations between particulate sulfate and organic carbon exposures and heart rate variability in patients with or at risk for cardiovascular diseases.
Chuang KJ, Chan CC, Su TC, Lin LY, Lee CT.

Institute of Occupational Medicine and Industrial Hygiene, College of Public Health, National Taiwan University, Taipei, Taiwan.

Abstract
OBJECTIVE:
It is still unknown whether specific components in fine particles are associated with heart rate variability (HRV) reduction.

METHODS:
We recruited 46 patients with or at risk for cardiovascular diseases to measure 24-hour HRV by ambulatory electrocardiographic monitoring. Fixed-site air-monitoring stations were used to represent participants' exposures to particles with aerodynamic diameters less than 10 microm (PM 10) and 2.5 microm (PM2.5), and particulate components of sulfate, nitrate, organic carbon (OC) and elemental carbon, and gaseous pollutants.
RESULTS:
We found that HRV reduction was associated with sulfate, OC, and PM2.5 but not with the other five pollutants in single-pollutant models. Sulfate was found to remain in significant association with HRV reduction adjusting for OC and PM2.5 in three-pollutant models.
CONCLUSIONS:
Exposures to sulfate and OC in PM2.5 were associated with HRV reduction in patients with or at risk for cardiovascular diseases.

Environ Int. 2007 Oct;33(7):993-8. Epub 2007 May 30.
Omega-3 fatty acids and the benefits of fish consumption: is all that glitters gold?
Domingo JL.

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Abstract
In recent years, a number of studies have clearly remarked the nutritional benefits of fish consumption: proteins, vitamins, minerals, and especially omega-3 polyunsaturated fatty acids (PUFAs), which may protect against several adverse health effects, including coronary heart disease mortality and stroke. However, some concerns about potential health risks derived from the environmental contaminants found in fish have been also raised. Therefore, balancing adequately the risks and benefits of fish consumption is currently a nutritional/environmental health key issue. In this paper, the most recent available scientific information concerning this issue is reviewed. It is concluded that although it seems evident that fish must be an important part of a balanced diet, to choose the most suitable species in terms of levels of PUFAs and pollutants, the frequency of consumption, and the meal size are essential aspects to balance benefits and risks of a regular consumption.

Epidemiology. 2008 Sep;19(5):680-9.
The role of particle composition on the association between PM2.5 and mortality.
Franklin M, Koutrakis P, Schwartz P.

Source
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Abstract

BACKGROUND:

Although the association between exposure to particulate matter (PM) mass and mortality is well established, there remains uncertainty about which chemical components of PM are most harmful to human health.

METHODS:

A hierarchical approach was used to determine how the association between daily PM2.5 mass and mortality was modified by PM2.5 composition in 25 US communities. First, the association between daily PM2.5 and mortality was determined for each community and season using Poisson regression. Second, we used meta-regression to examine how the pooled association was modified by community and season-specific particle composition.

RESULTS:

There was a 0.74% (95% confidence interval = 0.41%-1.07%) increase in nonaccidental deaths associated with a 10 microg/m3 increase in 2-day averaged PM2.5 mass concentration. This association was smaller in the west (0.51% [0.10%-0.92%]) than in the east (0.92% [0.23%-1.36%]), and was highest in spring (1.88% [0.23%-1.36%]). It was increased when PM2.5 mass contained a higher proportion of aluminum (interquartile range = 0.58%), arsenic (0.55%), sulfate (0.51%), silicon (0.41%), and nickel (0.37%). The combination of aluminum, sulfate, and nickel also modified the effect. These species proportions explained residual variability between the community-specific PM2.5 mass effect estimates.

CONCLUSIONS:

This study shows that certain chemical species modify the association between PM2.5 and mortality and illustrates that mass alone is not a sufficient metric when evaluating health effects of PM exposure.

N Engl J Med. 2004 Sep 9;351(11):1057-67.

The effect of air pollution on lung development from 10 to 18 years of age.

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.

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Erratum in

- N Engl J Med. 2005 Mar 24;352(12):1276.

Abstract

BACKGROUND:

Whether exposure to air pollution adversely affects the growth of lung function during the period of rapid lung development that occurs between the ages of 10 and 18 years is unknown.

METHODS:

In this prospective study, we recruited 1759 children (average age, 10 years) from schools in 12 southern California communities and measured lung function annually for eight years. The rate of attrition was approximately 10 percent per year. The communities represented a wide range of ambient exposures to ozone, acid vapor, nitrogen dioxide, and particulate matter. Linear regression was used to examine the relationship of air pollution to the forced expiratory volume in one second (FEV(1)) and other spirometric measures.

RESULTS:
Over the eight-year period, deficits in the growth of FEV(1) were associated with exposure to nitrogen dioxide (P=0.005), acid vapor (P=0.004), particulate matter with an aerodynamic diameter of less than 2.5 microm (PM(2.5)) (P=0.04), and elemental carbon (P=0.007), even after adjustment for several potential confounders and effect modifiers. Associations were also observed for other spirometric measures. Exposure to pollutants was associated with clinically and statistically significant deficits in the FEV(1) attained at the age of 18 years. For example, the estimated proportion of 18-year-old subjects with a low FEV(1) (defined as a ratio of observed to expected FEV(1) of less than 80 percent) was 4.9 times as great at the highest level of exposure to PM(2.5) as at the lowest level of exposure (7.9 percent vs. 1.6 percent, P=0.002).
CONCLUSIONS:
The results of this study indicate that current levels of air pollution have chronic, adverse effects on lung development in children from the age of 10 to 18 years, leading to clinically significant deficits in attained FEV(1) as children reach adulthood.

Comment in
- N Engl J Med. 2004 Dec 16;351(25):2652-3; author reply 2652-3.
- N Engl J Med. 2004 Sep 9;351(11):1132-4.
- N Engl J Med. 2004 Dec 16;351(25):2652-3; author reply 2652-3.
- N Engl J Med. 2004 Dec 16;351(25):2652-3; author reply 2652-3.

Environ Sci Technol. 2006 Sep 1;40(17):5340-6.
Statistically designed survey of polychlorinated dibenzo-p-dioxins, polychlorinated dibenzofurans, and co-planar polychlorinated biphenyls in U. S. meat and poultry, 2002-2003: results, trends, and implications.
Hoffman MK, Huwe J, Deyrup CL, Lorentzsen M, Zaylskie R, Clinch NR, Saunders P, Sutton WR.

Office of Public Health Science, Food Safety and Inspection Service, U.S. Department of Agriculture, Washington, D.C. 20056, USA.

Abstract
To obtain information on dioxin levels in the human diet, the Food Safety and Inspection Service of the United States Department of Agriculture recently determined levels of dioxin-like compounds (dioxins/dibenzofurans/PCBs) in four major slaughter classes (steers and heifers, market hogs, young chickens, and young turkeys) that comprise over 90% of the meat and poultry production in the United States. The data were analyzed and compared to data from smaller surveys carried out from 1994 to 1996. These surveys were conducted by different laboratories nearly 10 years apart, so a direct comparison of the data was not straightforward. Three approaches were taken: (1) comparison with nondetects set to zero, (2) comparison with nondetects set to half the limit of detection, and (3) comparison applying the earlier surveys' limits of detection to the newer data. The data analyses indicated that dioxin levels appear to have declined in three of the four slaughter classes, with young chickens, market hogs, and young turkeys declining 20-80%, while any declines in cattle dioxin levels, if real, are less than those observed in the other slaughter classes. Further study is needed to examine factors that might explain the differences in dioxin levels and distribution profiles in the four slaughter classes. A small number of market hog and steers/ heifers samples had dioxin toxic equivalency levels (TEQs)

greater than 2 pg/g lipid weight. Follow-up investigations for those samples indicated a common source for the market hog samples (a dioxin-contaminated mineral supplement), but no commonality was found for the steers/ heifers samples.

Comment in

- Environ Sci Technol. 2006 Sep 1;40(17):5168.

Inhal Toxicol. 2003 Apr 11;15(4):327-42.

The role of soluble components in ambient fine particles-induced changes in human lungs and blood.

Huang YC, Ghio AJ, Stonehuerner J, McGee J, Carter JD, Grambow SC, Devlin RB.

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Abstract

Normal individuals developed pulmonary neutrophilic inflammation and increased blood fibrinogen following inhalation of concentrated ambient particles (CAPS). In this study, we sought to determine how soluble components in CAPS contributed to these changes. We expanded and reanalyzed data from 37 young healthy volunteers from a previous study (Ghio et al., 2000) who were exposed to either filtered air or CAPS. Postexposure bronchoalveolar lavage (BAL) as well as pre- and postexposure venous blood samples was analyzed for cellular and acute inflammatory endpoints. Nine most abundant components in the water-soluble fraction of CAPS were correlated with these endpoints using principal component analysis. We found that a sulfate/Fe/Se factor was associated with increased BAL percentage of neutrophils and a Cu/Zn/V factor with increased blood fibrinogen. The concentrations of sulfate, Fe, and Se correlated highly with PM mass ($R > 0.75$) while the correlations between PM and Cu/Zn/V were modest ($R = 0.2$-0.6). These results from controlled human exposure linked specific PM components to pulmonary neutrolphil influx and blood fibrinogen increase, and indicated the soluble components of pollutant particles may differentially affect pulmonary and hematological systems in humans exposed to PM.

J Air Waste Manag Assoc. 2002 Jan;52(1):5-18.

Modeling the benefits of power plant emission controls in Massachusetts.

Levy JI, Spengler JD.

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Abstract

Older fossil-fueled power plants provide a significant portion of emissions of criteria air pollutants in the United States, in part because these facilities are not required to meet the same emission standards as new sources under the Clean Air Act. Pending regulations for older power plants need information about any potential public health benefits of emission reductions, which can be estimated by combining emissions information, dispersion modeling, and epidemiologic evidence. In this article, we develop an analytical modeling framework that can

evaluate health benefits of emission controls, and we apply our model to two power plants in Massachusetts. Using the CALPUFF atmospheric dispersion model, we estimate that use of Best Available Control Technology (BACT) for NOx and SO2 would lead to maximum annual average secondary particulate matter (PM) concentration reductions of 0.2 microg/m3. When we combine concentration reductions with current health evidence, our central estimate is that the secondary PM reductions from these two power plants would avert 70 deaths per year in a population of 33 million individuals. Although benefit estimates could differ substantially with different interpretations of the health literature, parametric perturbations within CALPUFF and other simple model changes have relatively small impacts from an aggregate risk perspective. While further analysis would be required to reduce uncertainties and expand on our analytical model, our framework can help decision-makers evaluate the magnitude and distribution of benefits under different control scenarios.

Atmospheric Environment Volume 35, Issue 24, August 2001, Pages 4125-4134

Gas/particle partitioning of polychlorinated dibenzo-*p*-dioxins and dibenzofurans in atmosphere; evaluation of predicting models

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Abstract

The gas/particle partitioning of polychlorinated dibenzo-*p*-dioxins and furans (PCDD/Fs) was measured at three sites for a year in order to monitor the variation of PCDD/Fs levels and describe their partitioning. The air concentrations of PCDD/Fs ranged from 71 to 1161 fg I-TEQ/m^3 and large changes in these levels did not correlate with seasonal changes during this study. Different homolog patterns were observed in the gas/particle phase. High chlorinated dioxin/furans dominated the particle phase while low chlorinated dioxin/furans dominated the gas phase. The high correlation coefficient between log [$(PCDD/Fs_{vap})(TSP)/(PCDD/Fs_{pat})$] and $1/T$ was observed in lower chlorinated dioxin/furans unlike in OCDD/F. The slope of homolog ranged from −0.410 to −1.025 and that of 2,3,7,8-substituted isomers ranged from −0.379 to −0.772 in plots of the log partition coefficient (K_p) versus the log subcooled vapor pressure ($P_L°$).

The octanol/air partition coefficient (K_{oa})-based model of PCDD/Fs is more compatible with experimental data than those of the Junge–Pankow model that tends to overestimate results, even though both models include some level of uncertainty. However, both models can underestimate the particle phase of PCDD/Fs, especially when the ambient air temperature is extremely low in winter.

Circulation, Volume 123, Issue 4, Pages e18-e209, 2011

Heart disease and stroke statistics: 2011 update: a report from the American Heart Association.

Roger VL, Go AS, Lloyd-Jones DM, et al. 2011.

Summary

Each year, the American Heart Association (AHA), in conjunction with the Centers for Disease Control and Prevention, the National Institutes of Health, and other government agencies, brings together the most up-to-date statistics on heart disease, stroke, other vascular diseases, and their risk factors and presents them in its Heart Disease and Stroke Statistical Update. The Statistical Update is a valuable resource for researchers, clinicians, healthcare policy makers, media professionals, the lay public, and many others who seek the best national data available on disease morbidity and mortality and the risks, quality of care, medical procedures and operations, and costs associated with the management of these diseases in a single document. Indeed, since 1999, the Statistical Update has been cited more than 8700 times in the literature (including citations of all annual versions). In 2009 alone, the various Statistical Updates were cited 1600 times (data from ISI Web of Science). In recent years, the Statistical Update has undergone some major changes with the addition of new chapters and major updates across multiple areas. For this year's edition, the Statistics Committee, which produces the document for the AHA, updated all of the current chapters with the most recent nationally representative data and inclusion of relevant articles from the literature over the past year and added a new chapter detailing how family history and genetics play a role in cardiovascular disease (CVD) risk. Also, the 2011 Statistical Update is a major source for monitoring both cardiovascular health and disease in the population, with a focus on progress toward achievement of the AHA's 2020 Impact Goals. Below are a few highlights from this year's Update.

The Lancet Oncology, Volume 10, Issue 5, Pages 453 - 454, May 2009 doi:10.1016/S1470-2045(09)70134-2

A review of human carcinogens—Part C: metals, arsenic, dusts, and fibres

Kurt Straif a, Lamia Benbrahim-Tallaa a, Robert Baan a, Yann Grosse a, Béatrice Secretan a, Fatiha El Ghissassi a, Véronique Bouvard a, Neela Guha a, Crystal Freeman a, Laurent Galichet a, Vincent Cogliano a, on behalf of the WHO International Agency for Research on Cancer Monograph Working Group

In March, 2009, 27 scientists from eight countries met at the International Agency for Research on Cancer (IARC) to reassess the carcinogenicity of metals, arsenic, dusts, and fibres previously classified as ~~"~~carcinogenic to humans" (Group 1) and to identify additional tumour sites and mechanisms of carcinogenesis. These assessments will be published as part C of Volume 100 of the IARC Monographs.

J Natl Cancer Inst. 1991 Oct 2;83(19):1380-5.

Lung cancer incidence among patients with beryllium disease: a cohort mortality study.

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Abstract

We have conducted a cohort mortality study on 689 patients with beryllium disease who were included in a case registry. An earlier mortality study on 421 of these patients was limited to males and resulted in a determination of a nonsignificant twofold lung cancer excess based on only seven lung cancer deaths. We have extended this earlier study by including females and by adding 13 years of follow-up. Comparison of the 689 beryllium disease patients with the U.S. population resulted in a lung cancer standardized mortality ratio (SMR) of 2.00 (95% confidence interval = 1.33-2.89) based on 28 observed lung cancer deaths. Adjustment for smoking did not change these results. All causes of mortality were also significantly elevated (SMR = 2.19), largely because of the very high rate of deaths due to pneumoconioses (primarily beryllium disease) (SMR = 34.23; 158 deaths). No other causes of death were significantly elevated. The excess of lung cancer was consistent for both sexes and did not appear to increase with duration of exposure to beryllium or with time elapsed since first exposure to this element. The case registry included those with acute beryllium disease, which resembles a chemical pneumonitis, and those with chronic beryllium disease, which resembles other pneumoconioses. The lung cancer excess was more pronounced among those with acute disease (SMR = 2.32) than among those with chronic disease (SMR = 1.57).

Comment in

- J Natl **Cancer** Inst. 1993 Oct 20;85(20):1697-9.

Carcinogenesis. 2005 Nov;26(11):1846-55. Epub 2005 Aug 25.

Air pollution and cancer: biomarker studies in human populations.

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Abstract

Large cohort studies in the U.S. and in Europe suggest that air pollution may increase lung cancer risk. Biomarkers can be useful to understand the mechanisms and to characterize high-risk groups. Here we describe biomarkers of exposure, in particular DNA adducts as well as markers of early damage, including mutagenicity, other endpoints of genotoxicity and molecular biomarkers of cancer. Several studies found an association between external measures of exposure to air pollution and increased levels of DNA adducts, with an apparent levelling-off of the dose-response relationship. Also, numerous experimental studies in vitro and in vivo have provided unambiguous evidence for genotoxicity of air pollution. In addition, due to the organic extracts of particulate matter [especially various polycyclic aromatic hydrocarbon (PAH) compounds], particulate air pollution induces oxidative damage to DNA. The experimental work, combined with the data on frequent oxidative DNA damage in lymphocytes in people exposed to urban air pollution, suggests 8-oxo-dG as one of the important promutagenic

lesions. Lung cancer develops through a series of progressive pathological changes occurring in the respiratory epithelium. Molecular alterations such as loss of heterozygosity, gene mutations and aberrant gene promoter methylation have emerged as potentially promising molecular biomarkers of lung carcinogenesis. Data from such studies relevant for emissions rich in PAHs are also summarized, although the exposure circumstances are not directly relevant to outdoor air pollution, in order to shed light on potential mechanisms of air pollution-related carcinogenesis.

Am J Ind Med. 1992;22(6):885-904.
A mortality study of workers at seven beryllium processing plants.
Ward E, Okun A, Ruder A, Fingerhut M, Steenland K.

Industrywide Studies Branch, National Institute for Occupational Safety and Health, Cincinnati, OH 45226.

Abstract
The International Agency for Research on Cancer (IARC) has found that the evidence for the carcinogenicity of beryllium is sufficient based on animal data but "limited" based on human data. This analysis reports on a retrospective cohort mortality study among 9,225 male workers employed at seven beryllium processing facilities for at least 2 days between January 1, 1940, and December 31, 1969. Vital status was ascertained through December 31, 1988. The standardized mortality ratio (SMR) for lung cancer in the total cohort was 1.26 (95% confidence interval [CI] = 1.12-1.42); significant SMRs for lung cancer were observed for two of the oldest plants located in Lorain, Ohio (SMR = 1.69; 95% CI = 1.28-2.19) and Reading, Pennsylvania (SMR = 1.24; 95% CI = 1.03-1.48). For the overall cohort, significantly elevated SMRs were found for "all deaths" (SMR = 1.05; 95% CI = 1.01-1.08), "ischemic heart disease" (SMR = 1.08; 95% CI = 1.01-1.14), "pneumoconiosis and other respiratory diseases" (SMR = 1.48; 95% CI = 1.21-1.80), and "chronic and unspecified nephritis, renal failure, and other renal sclerosis" (SMR = 1.49; 95% CI = 1.00-2.12). Lung cancer SMRs did not increase with longer duration of employment, but did increase with longer latency (time since first exposure). Lung cancer was particularly elevated (SMR = 3.33; 95% CI = 1.66-5.95) among workers at the Lorain plant with a history of (primarily) acute beryllium disease, which is associated with very high beryllium exposure. The lung cancer excess was not restricted to plants operating in the 1940s, when beryllium exposures were known to be extraordinarily high. Elevated lung cancer SMRs were also observed for four of the five plants operating in the 1950s for workers hired during that decade. Neither smoking nor geographic location fully explains the increased lung cancer risk. Occupational exposure to beryllium compounds is the most plausible explanation for the increased risk of lung cancer observed in this study. Continued mortality follow-up of this cohort will provide a more definitive assessment of lung cancer risk at the newer plants and among cohort members hired in the 1950s or later at the older plants. Further clarification of the potential for specific beryllium compounds to induce lung cancer in humans, and the possible contribution of other exposures in specific processes at these plants, would require a nested case-control study. We are currently assessing whether available industrial hygiene data would support such an analysis.

Comment in

- Am J Ind Med. 2001 Sep;40(3):284-8.

Toxicol Appl Pharmacol. 2002 Nov 1;184(3):172-9.

Interactions between ultrafine particles and transition metals in vivo and in vitro.

Wilson MR, Lightbody JH, Donaldson K, Sales J, Stone V.

Source

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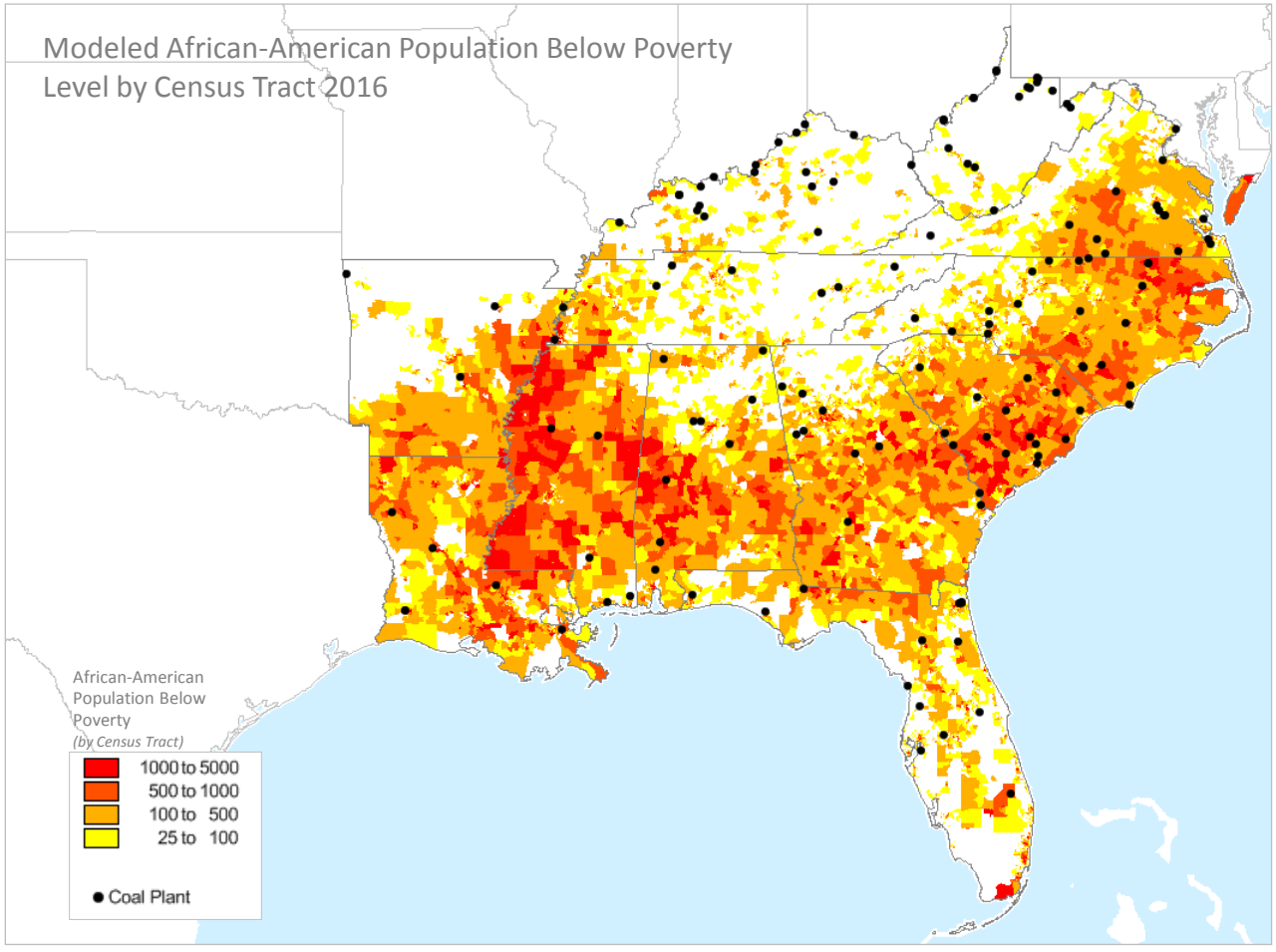
Abstract

Both the ultrafine particle and transition metal components of particulate air pollution (PM(10)) have been hypothesized to be important factors in determining toxicity and potential adverse health effects. In this study we aimed to investigate interactions between transition metal salts and a surrogate environmental particle-ultrafine carbon black (ufCB). In all experimental systems employed, the ufCB was found to be more reactive than its fine counterpart (CB). Incubation of ufCB with the reactive oxygen species (ROS)-sensitive probe dichlorofluorescin in the absence of cells generated significantly more ROS than CB. With addition of either cupric sulfate (CuSO(4)), ferrous sulfate (FeSO(4)), or ferric chloride (FeCl(3)), the ROS generation in the presence of ufCB was enhanced in a potentiative manner. In Mono Mac 6 macrophages, ufCB again produced more ROS than CB. However, addition of iron salts had no additive effect over and above that induced in the macrophages by ufCB. In the mouse macrophage cell line J774, ufCB decreased the cellular content of GSH and ATP. Addition of iron further decreased both GSH and ATP and a potentiative interaction between ufCB and FeSO(4) was observed, but only at the highest iron concentrations tested. A concentration-dependent increase in tumor necrosis factor-alpha production by J774 cells was also observed following exposure to ufCB, which was not further enhanced by the addition of iron. J774 cells were also found to sequester or chelate iron without inducing toxicity. In the rat lung ufCB induced a significant neutrophil influx and this inflammatory effect was potentiativelly enhanced by the addition of FeCl(3) (100 microM). These findings suggest that (1) ultrafine particles and metals interact by chemical potentiation in a cell-free environment to generate ROS, (2) potentiation between ultrafine particles and metal salts is not observed in the presence of macrophages as iron is sequestered or chelated by the cells, (3) in the lung, ultrafine particles and iron salts interact in a potentiative manner to generate inflammation.

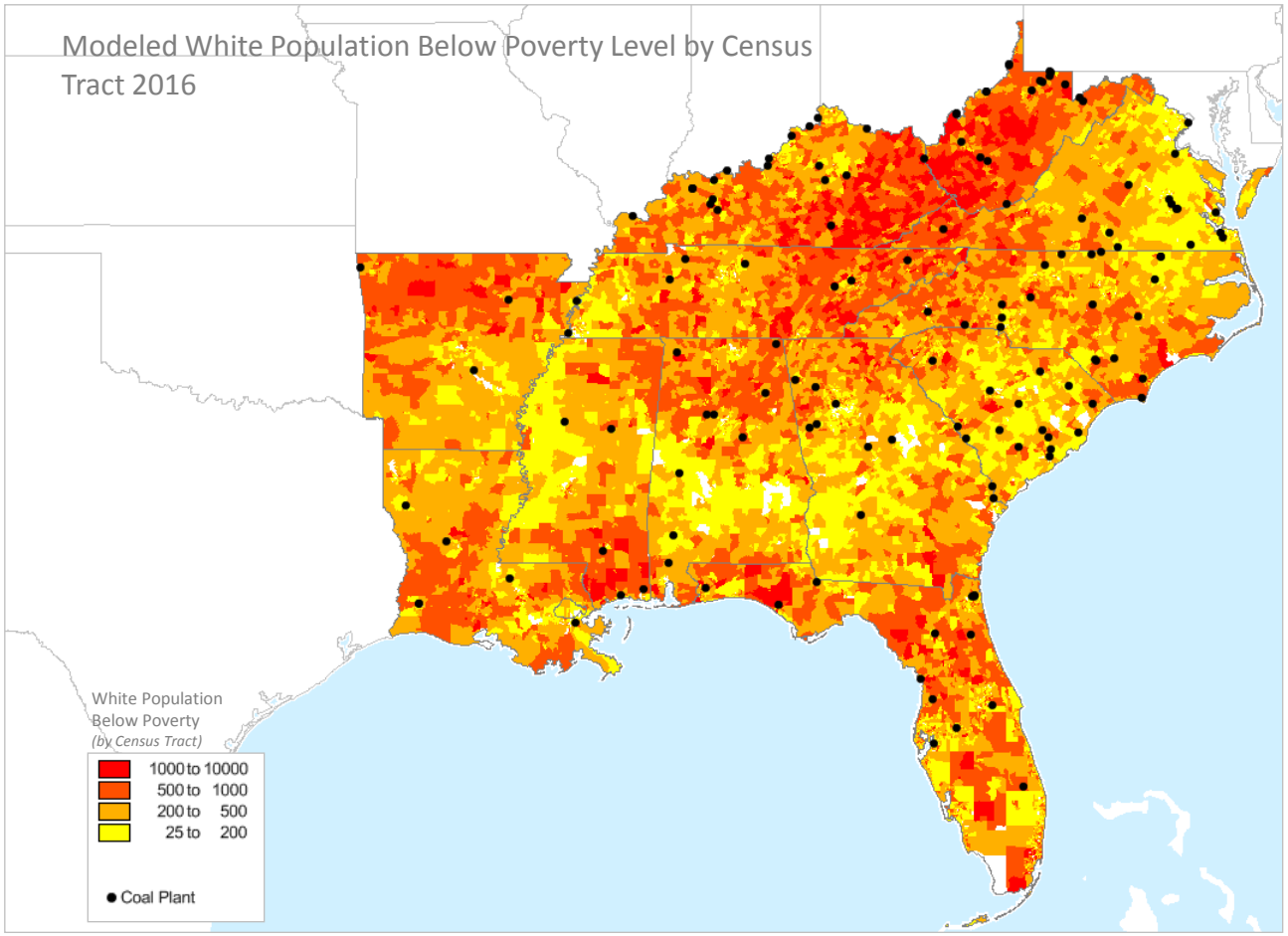
Figure 1: Coal-fired Power Plant Locations Relative to Modeled African-American Population below the Poverty Level by Census Tract in the Southeast for 2016



Sources: EPA, MJB&A Analysis, Ventyx

Sources: U. S. EPA; Ventyx *Velocity Suite; M.J. Bradley and Associates Analysis for the American Lung Association.*

Figure 2: Coal-fired Power Plant Locations Relative to Modeled White Population below the Poverty Level by Census Tract in the Southeast for 2016



Sources: EPA, MJB&A Analysis, Ventyx

Sources: U. S. EPA; Ventyx *Velocity Suite; M.J. Bradley and Associates Analysis for the American Lung Association.*