
June 29, 2004

I. INTRODUCTION


According to EPA’s 1999 “National Emissions Inventory,” coal-fired utility units emit about forty-one percent of all anthropogenic mercury air emissions in the United States. EPA estimates that these facilities emit more mercury than any other U.S. source category.

In addition to mercury, EPA’s 1998 Utility Air Toxics Study found that utility units emit 66 other HAPs on the list of 188 HAPs included on the Clean Air Act section 112(b) list. EPA assessed the inhalation and non-inhalation pathways for human exposure to these toxic air pollutants, and identified 13 priority HAPs for which the potential health risks demanded more detailed assessment. Specifically, EPA declared arsenic, beryllium, cadmium, chromium, lead, manganese, mercury, nickel, hydrogen
chloride, hydrogen fluoride, acrolein, dioxins, and formaldehyde to be priorities. Of the non-mercury HAPs, EPA identified arsenic, dioxins, and radionuclides from coal-fired units and nickel from oil fired units as highest priority for assessment.

1. **Mercury Emitted by Utility Units Causes Adverse Health Effects.**

According to EPA, a significant percentage of the mercury emitted from coal-fired utility units is deposited onto land or water bodies, where the chemical form of some amount of the deposited mercury can and does change into methylmercury. Methylmercury is a highly toxic form of mercury that bioconcentrates, or accumulates in the aquatic food web. It is taken in by microscopic animals or plants, which are in turn eaten by larger aquatic animals, which are themselves eaten. Because the rate at which methylmercury is ingested by fish is much faster than the very slow rate at which it is eliminated, larger fish can accumulate significant amounts of methylmercury in their tissues. In this way, methylmercury “can accumulate up the food chain in aquatic systems and lead to high concentrations of MeHg in predatory fish, which, when consumed by humans, can result in an increased risk of adverse effects in highly exposed or sensitive populations.”

In 2000, the National Research Council of the National Academy of Sciences described the potential adverse human health effects of consuming methylmercury (either directly, or in the case of a developing fetus, through the mother’s blood supply) in amounts above EPA’s “reference dose” (0.1 micrograms per kilogram of body weight per day, a level designed to reflect the safe amount that can be consumed daily over a lifetime). These effects include neurological and developmental problems.

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2 *Id.*, Table ES-1.
such as poor attention span and delayed language development, impaired memory and vision, problems processing information, and impaired fine motor coordination. Because a developing fetus is the most sensitive to the adverse effects of exposure to methylmercury, which is distributed in the mother’s blood supply, and passes through the placenta, women of child-bearing age are a population of most concern. Additionally, because children’s brain development continues after birth until at least age 14, children are also a population of concern.

Eating contaminated fish is the primary way that humans and wildlife are exposed to methylmercury. Once consumed, methylmercury remains in the human body for an extended period of time, 140-160 days on average. In June 2003, an international group of 50 scientists wrote to the Joint Expert Committee of Food Additives (a joint committee of the Food and Agriculture Organization of the United Nations and the World Health Organization (WHO)) in support of a tighter WHO standard for the consumption of mercury-contaminated fish. The scientists’ letter, (which is incorporated here by reference and attached as Appendix 1) was submitted to provide the WHO with updated research findings (since 2000) on mercury toxicity. The letter addresses emerging data on cardiovascular effects, additional tests on the Faroe Islands children’s cohort, and evidence that mercury exposure is widespread in the general public. In addition to the research summarized in this letter, two recent papers point to irreversible brain damage in the Faroe Islands children who were exposed to mercury in utero. These results

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4 Letter from 50 signatories to Samuel W. Page, PhD., Acting WHO Secretary to JECFA, World Health Organization. Re: 61st JECFA meeting (June 10, 2003).
demonstrate that although mercury affects fetal development in the womb, the damage persists throughout adolescence, and may be permanent.\textsuperscript{5,6}

Additionally, in 2003, the United States Centers for Disease Control (CDC) tested the blood mercury levels of a representative sample of women of childbearing age. Based on these CDC data and new research indicating that fetal blood levels are 70 percent higher than maternal levels, in February 2004 EPA scientific staff estimated that as many as 630,000 children may be born each year with unhealthy levels of mercury in their blood (\textit{i.e.}, at or above 5.8 micrograms per liter of blood).\textsuperscript{7}

In 2002, over 40 states across the country issued fish consumption advisories. These warnings advise people – particularly women of child-bearing age and children -- to avoid or limit their fish consumption due to mercury contamination of some or all of the fish taken from bodies of water in the state. This number of advisories represents nearly a 60 percent increase from the 27 states with active advisories in 1993. Based on our analysis of active advisories in 2002, this translates into 2,148 mercury advisories in effect for at least:

- 12,111,733 acres of lakes (including statewide advisories), or almost 30% of all lake acres;
- 453,101 miles of river (including statewide advisories), or almost 13% of all river miles;
- 15,639 miles of coastal areas (not including statewide advisories);
- 2,333 miles of our Great Lake coasts and tributaries; and
- 166,534 acres of bayou.\textsuperscript{8}

\textsuperscript{5} Grandjean, P. \textit{et al.}, Cardiac autonomic activity in methylmercury neurotoxicity: 14-year follow-up of a Faroese birth cohort, 50 \textit{J. of Pediatrics} 1-169-176 (February 2004).
\textsuperscript{6} Murata, K., \textit{et al.}, Delayed brainstem auditory evoked potential latencies in 14-year-old children exposed to methylmercury. 50 \textit{J. of Pediatrics}, 1-177-183 (February 2004).
\textsuperscript{8} U.S. PIRG, Fishing For Trouble, at 3 (June 2003).
Nineteen states -- Connecticut, Florida, Illinois, Indiana, Kentucky, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, North Dakota, New Hampshire, New Jersey, Ohio, Pennsylvania, Rhode Island, Vermont, and Wisconsin -- have issued statewide advisories for all of their inland freshwater lakes and/or rivers for at least one species of fish. Illinois, Florida, and Rhode Island have added, and North Carolina has rescinded, statewide advisories for inland waterways in the last year.  Eleven states -- Alabama, Florida, Georgia, Louisiana, Maine, Massachusetts, Mississippi, North Carolina, Rhode Island, South Carolina, and Texas -- also have issued statewide advisories for their entire coastal areas for at least one species of saltwater fish, with Rhode Island being the most recent state to issue such an advisory.

States’ mercury advisories also cover greater geographic areas than ever before. Since 2001, the number of river miles under advisory for mercury has increased by 9 percent (up from 414,973 in 2001), and the number of lake acres under advisory for mercury contamination has increased by 19 percent (up from 10,179,247 in 2001).

We note that additional data being collected about mercury exposure continue to confirm significant adverse public health effects. In light of this, and as discussed in detail below, EPA’s attempt to back away from aggressive controls on utility mercury emissions is deeply troubling. The direction of the science since December 2000 is entirely supportive of a strong regulatory approach to all power plant HAP emissions, as we will discuss further below. We furthermore are appalled, in light of this evidence, by the docket materials revealing that edits were made to the preamble during the

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9 Id.
10 Id.
11 Letter from 50 signatories to Samuel W. Page, PhD., Acting WHO Secretary to JECFA, World Health Organization. Re: 61st JECFA meeting (June 10, 2003).
interagency review process that intentionally downplayed the serious, confirmed health effects of mercury exposure.\textsuperscript{12}

2. \textbf{Adverse Public Health Effects Of Exposure To The 66 Non-Mercury HAPs Emitted By Utility Units.}

According to the 2001 Toxics Release inventory (TRI), electric utilities reported releasing over 700 million pounds of toxic chemicals to the air, making this industry the number one industrial toxic polluter.\textsuperscript{13} The health effects of these pollutants vary. Some are known to cause cancer, others impair reproduction and the normal development of children, and still others damage the nervous and immune systems. Many are respiratory irritants that can worsen already existing respiratory conditions such as asthma. Some of these pollutants are of environmental concern because they damage ecosystems and can harm the plants and animals that rely on these ecosystems.

In the 1998 Utility Air Toxics Study, EPA assessed inhalation exposures within 50 kilometers of utility plants, and also estimated the additional inhalation risk and cancer risk due to transported utility air emissions of the pollutants of concern. Two of the 426 coal-fired plants were estimated to pose lifetime cancer risks of greater than one in one million ($1 \times 10^{-6}$) due to inhalation of the HAPs they emit, with arsenic and chromium contributing most to this risk.\textsuperscript{14} Up to 11 of the 137 oil-fired plants analyzed were estimated to pose inhalation cancer risks from nickel of greater than 1 in one million.\textsuperscript{15}

\textsuperscript{12} Docket Item OAR-2002-0056-0107. Facsimile copy of the comments on the draft regulatory proposal received during the interagency review.

\textsuperscript{13} Clear The Air, “Toxic Neighbors” (2003), Appendix A at 26, Table 3 (available online at http://www.cleartheair.org/reports/toxic_neighbors/tn_tables.pdf.

\textsuperscript{14} Utility Air Toxics study at ES-7, ES-8, & Figure ES-2. Under the Clean Air Act, risks in excess of one in one million (or $1 \times 10^{-6}$) are generally of regulatory significance. For instance, if the “lifetime risk of cancer to the individual in the population who is most exposed to emissions of such pollutants” exceeds this threshold, 112(c)(9) of the Act provides that the industry cannot be removed from the list of industries under 112(c) requiring MACT regulation.

\textsuperscript{15} \textit{Id.} at ES-14.
EPA concluded that while mercury was the “HAP of greatest potential concern,” dioxins and arsenic from coal-fired plants and nickel from oil-fired plants are also of potential concern from a public health risk perspective.\textsuperscript{16}

Many power plant toxics belong to a class of chemicals that are persistent, bioaccumulative, toxic (PBT) pollutants. PBT pollutants either do not break down at all in the environment (for example, all metals) or break down very slowly (e.g., over decades), like dioxin. Continual loading of power plant pollution to the environment is especially important for PBT chemicals. Electric utilities released over 300,000 pounds of PBT chemicals to the air in 2001.\textsuperscript{17} Mercury accounted for the vast majority of these emissions, but power plants released other extremely toxic chemicals such as lead and lead compounds, polychlorinated biphenyls (PCBs), and polycyclic aromatic compounds.\textsuperscript{18} Table I-1 below summarizes the health effects of several pollutants released from coal-fired power plants.

\textsuperscript{16} Id. at ES-27.
\textsuperscript{17} Clear The Air, Toxic Neighbors, Appendix A at 26, Table 3.
\textsuperscript{18} For a discussion of PCBs, mercury, and other endocrine disrupters, see Colburn \textit{et. al.}, Our Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival? New York: Dutton, 1996.
<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Acute Effects</th>
<th>Chronic Effects</th>
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<tbody>
<tr>
<td>Hydrogen chloride</td>
<td>Inhalation causes coughing, hoarseness, chest pain, inflammation of respiratory and GI tracts, bronchitis, gastritis, laryngeal and pulmonary edema, kidney and liver damage, and shock.</td>
<td>Cancer effects – not classifiable. “This substance/agent has not undergone a complete evaluation and determination under US EPA’s IRIS program for evidence of human carcinogenic potential.” Chronic occupational exposure has been associated with gastritis, chronic bronchitis, dermatitis, dental erosion and vision damage.</td>
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<tr>
<td>Hydrogen Fluoride</td>
<td>Inhalation causes severe irritation to skin, eyes, nose, throat, larynx, lungs and even GI tract resulting in difficulty breathing, cough, chills, cyanosis, respiratory damage, pulmonary edema, shock and cardiac failure, severe skin &amp; eye burns, nausea, vomiting and diarrhea, and irritability of the nervous system.</td>
<td>Cancer effects—limited evidence of increased lung cancer in occupational groups. Not classified at present. Chronic exposures through drinking water or air can cause bronchial hyperreactivity as well as skeletal fluorosis with increased bone density, calcification of ligaments, weight loss, malaise, anemia, low white count, mottling of teeth.</td>
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<tr>
<td>Arsenic</td>
<td>Acute exposure by inhalation or ingestion results in cough, headache, extreme weakness, burning lips, throat constriction, gastric pain, vomiting, diarrhea, bloody urine, anemia and low white cells, cardiovascular effects esp. arrhythmia, numbness and tingling, muscle cramps, skin eruptions, severe thirst, shock, convulsions, coma, and death.</td>
<td>Known human carcinogen with high potency. Inhalation causes lung cancer; ingestion causes lung, skin, bladder and liver cancer. Chronic exposure results in degeneration of liver and kidneys, toxicity to central and peripheral nervous system, fatigue, headache, dizziness, insomnia, numbness of extremities, irritation to upper respiratory tract &amp; eyes including conjunctivitis, laryngitis and bronchitis, indigestion, thirst, wasting, alterations in blood formation, skin lesions, peripheral vascular disease with loss of blood flow and gangrene of extremities, atherosclerosis and heart attacks (even in children), myocarditis, heart arrhythmia, and diabetes. Evidence of genotoxicity, fetotoxicity and developmental effects.</td>
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<tr>
<td>Beryllium</td>
<td>High inhalation exposure can result in a chemical pneumonitis, cyanosis, pulmonary edema, difficulty breathing, chest pain, bronchial spasm, heart failure, and also dermatitis and eye inflammation.</td>
<td>Cancer effects- Probable human carcinogen, lung. Chronic effects include sensitization and progression to chronic beryllium disease with granuloma-type lung lesions, difficulty breathing, cough, fatigue, weight loss, chest pain, enlarged spleen, liver and heart as well as heart failure. Granulomas can affect organs beyond lungs. Limited evidence of immune system and reproductive/developmental effects.</td>
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</table>

19 Acute exposures more frequently occur in occupational settings. The general population can receive acute high exposures as a result of accidental releases or by being exposed to high levels of a toxin in contaminated drinking water.


<table>
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<tr>
<th>Substance</th>
<th>Description</th>
<th>Notes</th>
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<tbody>
<tr>
<td><strong>Cadmium</strong></td>
<td>Cadmium is more lethal by inhalation than by ingestion. Acute exposure results in headache, vomiting, chest pain, cough, restlessness and irritability, metal fume fever, pneumonitis and pulmonary edema, shock, unconsciousness and convulsions. A single acute exposure to high levels of cadmium can result in long-lasting impairment of lung function and/or Parkinsonism.</td>
<td>Probable human carcinogen of medium potency for lung and possibly prostate cancer. Kidney damage is the most common chronic effect and this is often associated with high blood pressure. Other chronic effects include difficulty breathing, bronchitis, pulmonary fibrosis, emphysema, anemia, GI symptoms, loss of appetite and weight loss, bone changes and fragility and liver damage. There is some evidence of genotoxicity.</td>
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<tr>
<td><strong>Chromium VI</strong></td>
<td>High exposure to chromium VI results principally in severe irritation or corrosive effects to skin, lungs, GI tract, abdominal pain, vomiting, shock, liver damage, kidney damage and decreased urine output and death.</td>
<td>Chromium VI-by inhalation known human carcinogen of high potency. Chronic effects-inflammation of the respiratory tract, mouth &amp; eyes, bronchospasm, nasal perforation, sinusitis, dermatitis, hepatitis, effects on the kidneys and gastrointestinal tract.</td>
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<tr>
<td><strong>Dioxin</strong></td>
<td>Acute exposure results in headache, dizziness, blurred vision, acute irritation of eyes, skin and respiratory tract, difficulty breathing, diarrhea, vomiting, fever, abdominal pain, muscle and joint pain, impaired muscle coordination, nervousness, irritability, ulcers, numbness and tingling, also cardiac, lung, liver and pancreas abnormalities, skin inflammation and chloracne, personality changes, sleep disturbances, sexual dysfunction, weakness and wasting.</td>
<td>Known human carcinogen – multiple types of cancer, particularly soft tissue sarcomas. It is a cancer promoter. Other chronic effects are likely more serious than cancer effects. Dioxin is the most potent toxic substance known. It is also persistent and bioaccumulative and current human exposures from past releases are at or near levels at which immune suppression and endocrine disrupter effects are known to occur. Other effects-lung, cardiac and liver damage, increased susceptibility to infection, altered glucose and fat metabolism leading to diabetes and atherosclerosis, thyroid disorders, central and peripheral nervous system changes, skin effects including chloracne, male and female reproductive toxicity, decreased fertility, hormonal changes and adverse reproductive outcomes including birth defects and developmental problems.</td>
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<tr>
<td><strong>Lead</strong></td>
<td>High lead exposures by inhalation or ingestion can cause breakdown of red blood cells, liver injury and acute brain encephalopathy with lethargy, vomiting, irritability, dizziness, seizures, coma. Long term effects include epilepsy, retardation and blindness.</td>
<td>Cancer effects- Probable human carcinogen. Chronic effects include central and peripheral nerve damage, kidney damage and effects on blood formation with wt. loss, anemia, weakness, irritability, impaired mental performance including learning difficulties, nausea, abdominal pain, insomnia, anxiety, joint pain, hypertension and immune system impacts. Reproductive/developmental- infertility, decreased sperm motility, premature births and miscarriages.</td>
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<td><strong>Manganese</strong></td>
<td>Following inhalation- metal fume fever, pneumonitis, bronchitis, severe/fatal pneumonia, neurological &amp; psychiatric symptoms, Parkinson-like syndrome, “manganese madness”.</td>
<td>Cancer Effects—not classifiable as a carcinogen Nervous system effects- irreversible Parkinson-like syndrome, tremors, weakness, impaired balance &amp; gait, memory deficits, speech difficulty, irritability, mental disturbances, muscle rigidity &amp; stiffness, peripheral nerve impairment, joint pain, impotence. Also evidence of increased susceptibility to infection. Learning disabilities in children. Newborns particularly susceptible because they absorb more Mn, excrete less and Mn crosses the Blood-brain barrier more easily.</td>
</tr>
</tbody>
</table>
| **Mercury** | Elemental Mercury – High inhalation exposure results in corrosive bronchitis and pneumonitis, central and peripheral nervous system effects, kidney damage, pneumonia, cardiac arrhythmia, shock, GI disturbances and increased blood clotting with infarctions in brain and kidney.  
Methylmercury- Acute adult exposure results in tremors, numbness and tingling, difficulty walking, visual and hearing impairment. | Birth defects found in animals.  
Elemental Mercury- Cancer effects not classifiable  
Chronic effects include weakness, tremor, enlarged thyroid, rapid pulse, blood changes, kidney damage, neurological impairments and personality changes. Several immune system effects can occur including sensitization, reduced immunity to infections, and autoimmune disease involving the kidneys.  
Methylmercury – possible human carcinogen. Chronic exposure results in neurotoxicity in adults—numbness & tingling, clumsy, stumbling gait, weakness & fatigue, vision & hearing loss, spasticity & tremor. Methylmercury is toxic to the fetus and causes neurological developmental effects - vision and hearing difficulties, delays in the development of motor skills and language acquisition, and later, lowered IQ points, problems with memory and attention deficits. |
| **Nickel** | High inhalation exposure can result in respiratory distress syndrome, pulmonary edema, metal fume fever, cough, shortness of breath, nasal irritation, sore throat and hoarseness, asthma attack, dermatitis. Sensitization to future exposure can also occur. | Cancer effects- Nickel compounds- known human carcinogens. Elemental nickel- possible human carcinogen (IARC) Lung & nasal cancer & others have been reported-renal, stomach & prostate. Nickel subsulfide is a known human carcinogen (nasal and lung). Chronic effects include lung inflammation & fibrosis, sinusitis, eye irritation, pneumoconiosis, asthma and contact dermatitis. In animals birth defects and increased fetal mortality have been reported. |
| **Selenium** | Following inhalation, irritation of lungs and mucous membranes, nosebleeds, difficulty breathing, cyanosis, shock, arrhythmia, cardiac arrest, pulmonary edema, liver congestion, nausea, vomiting, GI disturbances, disorientation, impaired vision, dizziness & coma. | Cancer effects- Selenium sulfide is probable carcinogen. Other forms of Se are not classifiable as to carcinogenicity. Liver degeneration, GI problems, hair and nail loss, dermatitis, CNS effects- depression, emotional instability, nervousness, labored breathing, myocarditis, erosion of long bones, Selenium sensitization, and possibly birth defects. |

In the Regulatory Finding, EPA reiterated that chromium, nickel, and cadmium are of potential concern for carcinogenic effects, noting that the cancer risks from exposure to these utility HAP emissions are “not low enough to eliminate those metals as a potential concern for public health.”22 Additionally, the agency stated that dioxins, hydrogen chloride, and hydrogen fluoride are of concern from a public health

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perspective, and that emissions of these HAPs are likely to increase from 1990 to 2010.\textsuperscript{23} EPA stated that these risks, as well as the remaining uncertainties, justified a finding that regulating HAP emissions from coal- and oil-fired utility units under section 112 is appropriate and necessary.\textsuperscript{24}

There is now also emerging scientific evidence that metals and acidic particles emitted from power plants may play a significant role in the health effects caused by fine particles.\textsuperscript{25} The high temperature in smokestacks provides the ideal environment for chemical reactions to take place between various substances especially in the presence of metals. Metals are known to function as catalysts, aiding or speeding up chemical reactions, and tend to be concentrated on the surface of fine and ultra-fine particles. For example, metals have been shown to catalyze the conversion of sulfur dioxide to sulfuric acid. Fine particulate matter has a large surface area that provides the ideal platform for these chemical reactions to take place. Even with low concentrations of sulfuric acid, significant damage can occur to critical areas of lung tissue because these particles are carried to the deepest regions of the lung.\textsuperscript{26}

In a recent study, EPA scientists and others concluded that the toxicity of coal fly ash is due primarily to the fine particle fraction and is associated with increased sulfur and trace element content (including zinc and iron).\textsuperscript{27} Furthermore, transition metals (a group which includes mercury, cadmium, chromium and nickel) are able to generate

\textsuperscript{23} Id. at 79,827, 79,829.
\textsuperscript{24} Id. at 79,830.
\textsuperscript{26} Id. at 96.
oxidants in biological systems - causing effects such as alveolitis, airway hyper-reactivity and increased virulence of pulmonary infection leading to enhanced mortality.\textsuperscript{28}

In addition, thousands of organic compounds can adsorb to the surface of fine particles. One such group of organic compounds is polycyclic organic matter (POM). This group of compounds is a concern because these compounds are mutagens and carcinogens and persist and bioaccumulate in the environment. Atmospheric transformation also results in the conversion of polycyclic aromatic hydrocarbons (PAHs) to nitro-PAHs, which are also potent carcinogens and mutagens. EPA did not include either POM or PAHs in the multi-pathway risk assessment conducted for the Utility Air Toxics Study.

Despite EPA’s statements in the Regulatory Finding about the need to further analyze the health effects of additional HAPs emitted from power plants, the agency in its proposed rule has dismissed out of hand the need to do so. In the preamble to the proposed rule, EPA misleads the public by selectively citing excerpts from the Utility Air Toxics Study that misrepresent the conclusions of that body of work. For example, with regard to public health risks from dioxin, the preamble states:

As for dioxins, organic HAP, EPA concluded that the quantitative exposure and risk results from such HAP “d[id] not conclusively demonstrate the existence of health risks of concern associated with exposures to utility emissions either on a national scale or from any actual individual utility.” (Utility RTC at 11-5).\textsuperscript{29} This statement is very misleading, as it reports only a fragment of what EPA actually concluded. The full text of EPA’s conclusions from the 1998 Utility Air Toxics Study is as follows (with omitted statements shown in italics):

\textit{This analysis of non-inhalation exposures to dioxin emission is a screening analysis. Thus, these quantitative exposure and risk results,}

\textsuperscript{28} Wilson and Spengler, \textit{supra}, at 120.
\textsuperscript{29} 69 Fed. Reg. at 4,656.
because of the many modeling and analytic uncertainties, are very
uncertain and do not conclusively demonstrate the existence of health
risks of concern associated with exposures to utility emissions either on a
national scale or from any actual individual utility. The lack of measured
data around these sources precludes a comparison with modeled results.
These results do suggest that exposures and risks of concern cannot at
present be ruled out and that there is a need for development of additional
scientific information to evaluate whether risk levels of concern may
exist.\textsuperscript{30}

EPA’s suggestion now that non-mercury HAPs do not pose risks to public health
therefore conflicts with the conclusions of the Utility Air Toxics Study and Report to
Congress.\textsuperscript{31} EPA’s prior study clearly calls for additional analyses for several of the non-
mercury HAPs, including dioxin, arsenic, nickel and chromium. The Utility HAP report
also cites the need for additional multi-pathway risk assessment, particularly for arsenic
and dioxin, and a further evaluation of short-term, high-end peak releases of hydrogen
chloride and hydrogen fluoride in particular.

In addition, the risk assessment presented in the Utility Air Toxics Study is itself
limited because it did not account for multiple and cumulative exposures associated with
power plant emissions. The failure to account for these types of exposure resulted in an
assessment that generally underestimates the total health risk from power plant
emissions. The assessment also does not address many pollutants for which there are

\textsuperscript{30} Utility Air Toxics Study at 11-5 (italics added).

\textsuperscript{31} Moreover, these distortions are on top of the already watered-down assessments found in the 2000
Regulatory Finding, of the risks posed by exposure to utility HAPs emissions. The original EPA staff draft
of the Finding was revised by the Office of Management and Budget during that earlier review process.
For example, modifying clauses suggesting uncertainty were added by OMB even to simple statements of
accepted scientific fact from the staff draft. See Regulatory Finding, OMB Executive Order 12866 Review
Draft, Docket No. A-92-55 Item No. I-I-6 at 21, and compare staff draft: “Children exposed after birth are
also potentially more sensitive to the toxic effects of methylmercury than adults because their nervous
systems are still developing,” with OMB changes: “It is also possible that children exposed after birth are
also potentially more sensitive to the toxic effects of methylmercury than adults because their nervous
systems are still developing.”
limited health effects data. However, the lack of data does not imply that there is no public health risk.

As discussed elsewhere in these comments, EPA’s conclusion that the risks from utility units’ mercury emissions make regulation “appropriate and necessary” was sufficient to trigger a statutory duty to promulgate maximum achievable control technology (MACT) standards for each HAP utility units emit. Nevertheless, even if one were to accept EPA’s unlawful approach, by which the agency will only regulate those HAPs emitted by utility units that pose a quantifiable danger, it is clear that EPA has ignored important evidence regarding non-mercury HAPs. It is premature and in conflict with prudent public health policy to dismiss potential health risks from the non-mercury HAPs without completely evaluating inhalation and multi-pathway exposures. A complete assessment of power plant emissions must:

- Evaluate the risks from all pathways of exposure to HAP from power plants and quantify the cumulative risks for persistent bioaccumulative pollutants.
- Account for dioxin emissions and their possible generation in electrostatic precipitators.
- Account for exposure to pollutants that disperse more than 30 miles from the power plant – the maximum distance accounted for in EPA’s computer models.
- Take into account the health effects of cumulative exposure to multiple HAPs emitted by power plants, by all routes of human exposure - inhalation, drinking water, food ingestion and skin absorption.
- Account for emission increases that occur during start-up, shutdown or upset operating conditions. These occurrences are a normal part of routine operations and should be represented in emissions testing.
- Account for secondary formation of pollutants in the power plant plume.
- Account for overlapping power plant plumes.
- Account for exposure to power plant wastes and water discharges.
B. The Hazardous Air Pollutants Emitted by Utility Units are Associated with Adverse Environmental and Economic Effects.

In addition to the significant public health effects associated with HAPs emissions from the utility industry, mercury contamination and other adverse effects of HAPs exposure cause environmental damage, and impair regional economies. Mercury contamination is a direct threat to recreational fishing—a vital piece of our national and state economies. Recreational fishing is a multi-billion dollar industry. In 2001, the most recent year for which the data is available, approximately 34.1 million Americans took a total of 437 million fishing trips and spent 557 million days fishing. In 2001, recreational fishing in America:

- Generated more than $35.6 billion in spending on food, lodging, and transportation for fishing trips; fishing and auxiliary equipment; and other items;\(^{32}\)
- Generated more than $116 billion in total economic output;\(^{33}\)
- Supported more than one million jobs;\(^{34}\)
- Created more than $30.1 billion in household income (salaries and wages);\(^{35}\)
- Added more than $1.9 billion in sales tax revenues;\(^{36}\)
- Added more than $470 million in state income tax revenues;\(^{37}\) and
- Generated $4.88 billion in federal income tax revenues.\(^{38}\)

Even a small dent in the recreational fishing industry can mean large economic losses. Of all the money spent on fishing, more than $27.8 billion was spent in states that have issued fish consumption advisories due to mercury. Two of the ten states with the largest number of river miles under advisory -- Florida and Ohio -- are also in the top ten for spending on fishing. Five of the top ten states with the most lake acres (including


\(^{34}\) Id.

\(^{35}\) Id.

\(^{36}\) Id.
statewide) under mercury advisory -- Minnesota, Florida, Michigan, Texas, and
Wisconsin -- are also in the top ten for money spent towards recreational fishing. In
fact, nine of the 19 states with statewide mercury advisories covering all of their inland
lakes or rivers -- Florida, Illinois, Michigan, Minnesota, Missouri, New Jersey, Ohio,
Pennsylvania, and Wisconsin -- also fall in the top twenty states for expenditures on
recreational fishing.

EPA’s Regulatory Finding also recognized that “wildlife consume fish from a
much more limited geographic area than do humans which can result in elevated levels of
mercury in certain fish-eating species in localized geographic areas. Those species can
include kingfisher, river otter, raccoon, loon, as well as some endangered species such as
the Florida panther.” Recent studies confirm the seriousness of the adverse effects of
methylmercury exposure on wildlife. Wildlife viewing is an essential aspect of many

37 Id.
38 Id.
41 See, e.g., Evers, D., O. Lane, C. DeSorbo, and L. Savoy. Assessing the impacts of methylmercury on
Submitted to Maine Department of Environmental Protection, Surface Water Ambient Toxic Monitoring
Program. Biodiversity Research Institute (2003); Evers, D., Status assessment and conservation plan for the
Common Loon (Gavia immer) in North America. U.S. Fish and Wildlife Service (2003); & Evers, D.,
Assessing the potential impacts of methylmercury on the Common Loon in Southern New Hampshire.

See also, e.g., Evers, D. D. Yates, and L. Savoy, Investigation of mercury exposure in Maine’s
mink and river otter. Report BRI 2002-10 submitted to Maine Department of Environmental Protection and
Maine Inland Fisheries and Wildlife. Biodiversity Research Institute (2002); Osowski, S.L., L. W. Brewer,
O.E. Baker and G.P. Cobb, The decline of mink in Georgia, North Carolina and South Carolina – the role
of contaminants. 29 Archives of Environmental Contamination and Toxicology 418 (1995) (assessing
impacts of mercury exposure on mink).

See also, e.g., Evers, D., D. Yates, and L Savoy. Investigation of mercury exposure in Maine’s
mink and river otter. Report BRI 2002-10 submitted to Maine Department of Environmental Protection and
Maine Inland Fisheries and Wildlife. Biodiversity Research Institute (2002); Mierle, G., E.M. Addison,
K.S. MacDonald, and D.G. Joachim, Mercury levels in tissues of otters from Ontario, Canada: Variation
with age, sex and location, 19 Envtl. Toxicology and Chemistry 3044 (2000) (assessing impacts of mercury
exposure on river otter).

See also, e.g., Burger, J. and M. Gochfield, Risk, mercury levels and birds: relating adverse laboratory
effects to field biomonitoring, 75 Env’t. Research 160 (1997); Thompson, D.R. Mercury in birds and
people’s outdoors experience – many hikers, kayakers, and other outdoor enthusiasts pursue these activities in order to view wildlife in its natural habitat. Adverse wildlife effects, including lowered reproductive rates and increased mortality of animals such as the river otter or species of waterfowl, also might be expected to generate long-term economic effects in areas local to populations of the affected wildlife species.

C. Statutory and Regulatory Background.

In 1990, Congress established a detailed, technology-based regulatory scheme for the emissions of 188 listed HAPs emitted by stationary sources. HAPs “present a threat of adverse human health effects . . . or adverse environmental effects.” Congress directed EPA to publish a list of “all categories and subcategories of major sources and area sources” that emit the listed HAPs. For listed categories or subcategories of major sources of HAPs, Congress further directed that EPA must promulgate rules requiring the maximum achievable reduction in HAPs emissions, known as “MACT” regulations.

Although electric utilities are a major contributor of HAPs, and emit a “significant number of the 188 HAP[s] included on the Section 112(b) list,” Congress required EPA to study “the hazards to public health reasonably anticipated to occur as a result of emissions” from electric utilities before regulating them. The statutory scheme further requires that EPA “shall regulate [the] electric utility [industry] . . . under this section”


42 See generally 42 U.S.C. § 7412.
43 42 U.S.C. 7412(b)(2); see also 61 Fed. Reg. at 68,384 (interpreting this statutory definition to include those pollutants “known or suspected to cause cancer, nervous system damage, birth defects or other serious health effects.”).
44 42 U.S.C. § 7412(c). A “major source” of HAPs is defined in the Act as “any stationary source or group of sources located within a contiguous area and under common control that emits or has the potential to emit considering controls, in the aggregate, 10 tons per year or more of any hazardous air pollutant, or 25 tons per year or more of any combination of hazardous air pollutants.” Id. § 7412 (a)(1).
upon finding that “such regulation is appropriate and necessary after considering the results of” the utility health hazards study. EPA interpreted these provisions to prohibit it from listing utility units as a source category subject to MACT requirements until it completed the required studies.

In 1992, after EPA published its list of source categories for which MACT standards were required, the Natural Resources Defense Council (NRDC) sued the agency for refusing to include utility units on the list. NRDC argued that the source category listing obligation applied to “all” industrial categories containing major sources, and thus EPA’s approach to utility units was unlawful. EPA and NRDC settled that litigation, with an agreement that put EPA on a schedule to complete the required studies, make the regulatory finding and promulgate rules in accordance with that finding. Under the agreement, EPA was required to issue its regulatory finding by December 15, 2000, issue a notice of proposed rulemaking by December 15, 2003, and take final action on its proposal by December 15, 2004. On April 27, 2004, in response to EPA’s proposal and the agency’s public suggestions that it would undertake additional analyses of alternative approaches, NRDC notified EPA that it would treat the agreement as satisfied if the Agency completed final rulemaking by March 15, 2004. The parties subsequently modified the settlement agreement to reflect this date.

48 Id.
49 See 42 U.S.C. § 7412(c)(1).
50 On April 28, 2004, moreover, the Izaak Walton League of America, the National Wildlife Federation, and Natural Resources Council of Maine filed suit in the U.S. District Court for the District of Columbia, seeking both a declaratory judgment that a MACT standard is required for utility units, and also a court-ordered and enforceable deadline for the issuance of such standards. Izaak Walton League of America v. Leavitt, Civ. No. 04-694 (D.D.C.).
Pursuant to the agreement with NRDC, in 1998, EPA completed the health hazards study concerning HAPs emissions from utility units as mandated by Congress in section 112(n) of the CAA. 51 Congress, as part of the 1999 EPA appropriations process, further directed the Agency to fund the National Academy of Sciences (“NAS”) to complete an independent study specific to the toxicological effects of one utility HAP, mercury, and prepare recommendations on the establishment of a safe methylmercury exposure reference dose.52 That study found:

The population at highest risk [for adverse effects due to methylmercury exposure] is the offspring of women of childbearing age who consume large amounts of fish and seafood. The committee estimates that over 60,000 children are born each year at risk for adverse neurodevelopmental effects due to in utero exposure to [methylmercury].53

In 2000, relying on the section 112(n) utility health hazards study, the additional study released by the NAS, subsequent peer review analyses, and other available information including public comment, EPA concluded that “the available information indicates that mercury emissions from electric utility steam generating units comprise a substantial portion of the environmental loadings and are a threat to public health and the environment.”54 In particular, EPA stated:

[M]ercury is both a public health concern and a concern in the environment . . . and . . . there is a plausible link between methylmercury concentrations in fish and mercury emissions from coal-fired electric utility steam generating units. Although the degree to which that linkage occurs cannot be estimated quantitatively now, the facts are that: There is a linkage between coal consumption and mercury emissions; electric

51 Utility Air Toxics Study, see supra n.1. In 1997, EPA had completed an additional study, mandated by section 112(n)(1)(B) of the Act. See, U.S. EPA, “Mercury Study: Report to Congress” (Dec. 1997). That study addressed “the rate and mass of [mercury emissions from electric utility steam generating units, municipal waste combustion units, and other sources], the health and environmental effects of such emissions, technologies which are available to control such emissions, and the costs of such technologies.” 42 U.S.C. §7412(n)(1)(B).
53 Id. at 327.
utility steam generating units are the largest domestic source of mercury emissions; and certain segments of the U.S. population (i.e., the developing fetus, subsistence fish-eating populations) are believed to be at potential risk of adverse health effects due to mercury exposures resulting from consumption of contaminated fish.55

Accordingly, the Agency determined that regulation of HAP emissions from utility units under section 112 of the Act is appropriate and necessary.56 EPA justified that determination on the basis, among other things, that utility units “emit a significant number of the 188 HAP included on the section 112(b) list,”57 and that “a number of control options . . . will effectively reduce HAP emissions from” utility units, and “because the implementation of other requirements under the CAA will not adequately address the serious public health and environmental hazards arising from [utility HAP] emissions. . . .”58 The EPA at the same time added utility units to the list of source categories under section 112(c) of the Act, for which MACT regulations must be developed.59

Once EPA determined that regulation of electric utilities was “appropriate and necessary,” and listed electric utilities as a source category, EPA’s obligation to develop MACT standards immediately became effective.60 Moreover, under section 112(c)(2),

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56 Id. at 79,830.
57 Id. at 79,829,
58 Id.
59 Id. at 79,826, 79,830.
60 Section 112(c)(5) states that “. . . the Administrator may at any time list additional categories and subcategories of sources of hazardous air pollutants . . . .” EPA’s regulations, 40 C.F.R. 63.40(c), state that “The requirements of this subpart do not apply to electric utility steam generating units unless and until such time as these units are added to the source category list pursuant to section 112(c)(5) of the Act.” EPA’s actions in requiring case-by-case MACT determinations for new coal fired power plants subsequent to December 2000 demonstrate that the Agency’s listing of coal and oil fired utility units was, in fact, under 112(c)(5). See Memorandum from John Seitz, Director, U.S. EPA Office of Air Quality Planning and Standards, to EPA Regional Office Air Directors, entitled “Case-by-Case MACT for New Oil- and Coal-fired Electric Utility Steam Generating Units” (August 1, 2001); see also Comment Letter on Thoroughbred Generating Station, from Kay T. Prince, Chief, Air Planning Branch, U.S. EPA Region 4 to
“[f]or the categories and subcategories the Administrator lists, the Administrator shall establish emission standards under subsection (d) of this section.”61 Subsection 112(d) echoes this obligation: EPA “shall promulgate regulations establishing emission standards for each category or subcategory of major sources and area sources of hazardous air pollutants listed for regulation pursuant to subsection (c) of this section.”62

The Act goes on to specify that the emission standards must be MACT standards: “Emissions standards promulgated under this subsection [(d)] and applicable to new or existing sources of hazardous air pollutants shall require the maximum degree of reduction in emissions of the hazardous air pollutants subject to this section . . . achievable.”63 The Act neither allows nor requires any “decision” to develop MACT standards pursuant to section 112(d), nor does the Act authorize EPA to “decide” not to develop MACT standards for a listed source category, as will be discussed below.

Having committed the agency to issuing a MACT standard for utility units, EPA established a Utility MACT Working Group (“Working Group”) in the Spring of 2001, as a subcommittee of the Permits, New Source Reviews, and Toxics Subcommittee of the Clean Air Act Advisory Committee, which itself was established under the Federal Advisory Committee Act (FACA).64 The Agency’s Process and Charge to the Working Group defined the scope of the Rulemaking to include “the oil- and coal-fired subset of fossil fuel-fired electric utility steam generating units defined under section 112(a)(8) of the CAA . . . These units are scheduled for regulation under section 112 (NESHAP) after

John S. Lyons, Director Department for Environmental Protection, Kentucky Natural Resources & Environmental Protection Cabinet (February 26, 2002) at 1, 2-4.
61 42 U.S.C. § 7412(c)(2) (emphasis added).
being added to the list of source categories for such regulation in [the Agency’s December 2000 Regulatory Finding] notice . . . .”65 The Working Group was further directed to consider all the pollutants listed under CAA section 112(b) for regulation. EPA asserted that it expected the Working Group to “meet periodically throughout the project.”66

The Working Group included 30 representatives (and additional alternates) from various stakeholder constituencies, as follows: 6 state/local/tribal regulatory agency representatives, 8 environmental organization representatives, and 16 representatives from the regulated industry, fuel suppliers, and labor organizations. The Working Group first met on August 1, 2001, and continued to meet regularly through 2002 for a total of 13 meetings.67 At the October 2002 meeting, the Working Group presented a range of stakeholder recommendations to the Agency for the development of the MACT standard.68,69 The stakeholder recommendations covered several topics, including subcategorization, MACT emission levels (both at and beyond the statute’s so-called “floor”), non-mercury HAPs, methodologies for taking into account variability in emissions, form of the standard, compliance time, and monitoring. The Working Group also recommended approaches for the MACT standard for oil-fired utility units.

66 Id. at 3 & 6, Table 2.
67 See http://www.epa.gov/ttn/atw/combust/utiltox/utoxpg.html#CAAAC.
68 See Working Group Final Report at http://www.epa.gov/ttn/atw/combust/utiltox/utoxpg.html#CAAAC.
69 A diverse group of stakeholders, including representatives of states, industry, and environmentalists, also submitted a consensus document on several key issues, including the MACT floor levels, subcategorization, the form of the standard and a methodology for dealing with variability. See Consensus Positions Concerning the Utility MACT Standard, Submitted to the Utility Working Group by Environmental Stakeholders, Clean Energy Group, New Jersey Department of Environmental Protection, STAPPA/ALAPCO, and the Northeast States for Coordinated Air Use Management (Oct. 2002).
Following a break of several months, the Working Group met again on March 3, 2003, and at that meeting discussed the Agency’s plans for subsequent work, and planned an April 15, 2003 meeting to discuss these efforts. Among the topics planned for discussion at that meeting were the results of EPA modeling of various proposals for MACT floors based on the alternative recommendations the Working Group had developed. That meeting was abruptly cancelled with very little notice to Working Group members, and not rescheduled. Nor did the Agency ever provide the Working Group members, as a body, further information regarding the development of the proposal. But it is notable that during the entire Working Group process, neither the stakeholders in the process nor EPA ever discussed any alternative to the issuance of a MACT standard for coal- and oil-fired utility units.

D. Despite the Statutory Mandate to Promulgate a MACT Standard, EPA has Proposed Two Alternative Regulatory Schemes.

EPA’s regulatory proposal offers three approaches to regulating HAP emissions from utility units. EPA on the one hand proposes establishment of a section 112(d) MACT standard, but only for the mercury emitted by coal-fired utility units, and for the

70 Electronic mail from Bill Maxwell, US EPA to all Utility Working Group members (April 3, 2003) (forwarding an April 1, 2003 electronic mail from Sally Shaver, US EPA to members of a subgroup of the Utility Working Group concerned about modeling various MACT floor scenarios, announcing EPA’s inability to complete model runs in time for the April 15, meeting, and announcing the cancellation of the meeting for that reason (attached as Appendix 2). See also electronic mail from Bill Maxwell to Michael Rossler of EEI (April 3, 2003)(apologizing for late cancellation of meeting and room reservation, and noting that Mr. Maxwell had not “been told of the time frame during which [the meeting] would be rescheduled”) (attached as Appendix 3). The meeting never was rescheduled.

71 Maxwell email to Utility Working Group Members.

72 See letter from John Paul, Regional Air Pollution Control Agency, Dayton Ohio (Utility Working Group Co-chair) to Jeffrey Holmstead, Assistant Administrator for Air and Radiation, U.S. EPA (November 3, 2003) (noting that EPA modeling of stakeholder recommendations was supported by the full CAAAC and approved by Holmstead at the October 2003 CAAAC meeting, yet never performed, and that the April 15, 2003 meeting of the Utility Working Group was not rescheduled after its cancellation) (attached as Appendix 4)

73 Even OMB, despite weakening statements about potential health risks from HAPs exposure, during its review of the EPA staff draft of the Regulatory Finding in 2000 actually strengthened the draft’s language...
nickel emitted by oil-fired units. Moreover, as we will discuss further below, the minimum or “floor” emissions levels EPA proposes are orders of magnitude less stringent than the statute requires. Under this proposal EPA asserts that total resulting mercury emissions from the coal-fired utility sector would be lowered by 2010 to somewhere between 30 and 34 tons annually (from approximately 48 tons currently emitted).

As a second alternative, EPA asserts legal authority under section 112(n) to fashion a mercury trading program, with caps apparently based on the agency’s proposed MACT levels (although that aspect of the alternative is far from clear). This alternative would involve rescinding the decision to list utility units as a source category under section 112(c), which accompanied the December 2000 finding to regulate, but would not rescind the determination that it is appropriate and necessary to regulate such units. Like the MACT approach, EPA claims this would yield total annual mercury emissions from the coal-fired utility sector of between 30 and 34 tons by 2010.74

Third, EPA proposes to “revise” the 2000 Regulatory Finding, conclude that regulating utility units under section 112 is not “necessary,” and instead encourage states participate in a nationwide mercury pollution trading market, with caps becoming effective in 2010 and 2018. The agency claims authority to create such a regime pursuant to section 111 of the Act, which gives EPA authority to promulgate New Source Performance Standards (NSPS), and emission guidelines for states to follow in regulating existing pollution sources. EPA also finds authority for the establishment of a cap-and-

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74 See 69 Fed. Reg. at 4,661-62
trade program for mercury, under the authority of section 111 of the Act. EPA’s caps would be set at approximately 34 tons in 2010 and at 15 tons in 2018.

There are significant flaws with each of the EPA alternative proposals for regulation. The MACT standard is significantly weaker than the statute requires, both because of the mechanics of the MACT floor calculation, in which EPA grossly over-accounts for variability in emissions levels, but also because the Agency completely ignores several HAPs which have been identified as of potential concern. EPA’s attempted justifications for either of its cap-and-trade approaches are blatantly outside the law, and will eventually fail.

We discuss these serious shortcomings of the proposal in the following chapters. In Chapter II we critique the Agency’s MACT standard-setting approach, and demonstrate that faithfully implementing the CAA would result in very strict MACT floors, to say nothing of beyond-the-floor control. Moreover, we show that even if the agency’s basic MACT floor approach were followed, far more stringent emission limits would result. In Chapter III we demonstrate the illegality of EPA’s proposal to regulate listed HAPs under section 111. In Chapter IV we demonstrate the illegality of EPA’s cap and trade proposals. And in Chapter V we demonstrate that a more stringent set of standards than EPA has proposed here is cost effective.