State of New Jersey v. United States EPA on Utility Unit Mercury Emissions

by ***Robert Ferguson*** *and the* ***Washington Legal Foundation***

"All scientific evidence cited in this brief was submitted to EPA during the comment period by the Center for Science and Public Policy and, to a lesser extent, by other parties."

The papers herein credited were authored by SPPI President, Robert Ferguson.

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UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT

No. 05-1097 (and consolidated cases) COMPLEX

STATE OF NEW JERSEY, *et al.*

Petitioners,

v.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY,

Respondent.

On Petition for Review of Final Rules
of The United States Environmental Protection Agency

BRIEF OF AMICUS CURIAE WASHINGTON LEGAL FOUNDATION IN SUPPORT OF RESPONDENT

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CERTIFICATE AS TO PARTIES, RULINGS, AND RELATED CASES

Pursuant to Circuit Rule 28(a)(1), the undersigned counsel for Amicus Curiae Washington Legal Foundation submits this certificate as to parties, rulings, and related cases:

A. Parties and Amici:

All parties and amici are listed in the brief of Government Petitioners State of New Jersey, et al., and the brief of Respondent the United States Environmental Protection Agency.

B. Rulings Under Review:

Reference to the issues under review appear in the brief of Government Petitioners.

C. Related Cases:

Amicus Curiae Washington Legal Foundation is not aware of any related case pending in this or any other Court.

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The Washington Legal Foundation is a non-profit corporation organized under the laws of the District of Columbia. It has no parent company and has issued no stock.

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GLOSSARY

Pursuant to Circuit Rule 28(a)(3), the following is a glossary of all acronyms and abbreviations used in this brief:

CAA	Clean Air Mercury Act
CAIR	Clean Air Interstate Rule
CAMR	Clean Air Mercury Rule
CDC	Center for Disease Control
EPA	Environmental Protection Agency
EPRI	Electric Power Research Institute
FDA	Food and Drug Administration
Hg	mercury
MACT	Maximum achievable control technology
MeHg	methylmercury
NHANES	National Health and Nutrition Examination Survey
PCB	polychlorinated biphenyls
ppm	parts per million
ppb	parts per billion
RfD	reference dose
µg/kg/day	MeHg per kilogram of body weight
WHO	World Health Organization

STATUTES AND REGULATIONS

All applicable statutes and regulations are contained in the Brief for Government Petitioners and the United States Environmental Protection Agency.

STATEMENT OF INTEREST

Pursuant to FRAP 29(c)(3), Amicus Curiae Washington Legal Foundation (WLF) is a national non-profit public interest law and policy center based in Washington, D.C. WLF's supporters include consumers, businesses, and property owners affected by Environmental Protection Agency (EPA) regulation under various environmental statutes such as the Clean Air Act (CAA). WLF's interest and authority to file this brief are further described in WLF's May 27, 2005 unopposed motion for leave to file an amicus brief, which this Court granted on June 29, 2005.

ISSUE STATEMENT

Whether the EPA properly decided it was not "necessary and appropriate" to regulate mercury emissions from utility electric generating units under Section 112 of the Clean Air Act, 42 U.S.C. § 7412,[2] because such utility units cannot reasonably be anticipated to pose a hazard to public health.

STATEMENT OF THE CASE

CAA section 112(n)(1)(A), adopted as part of the 1990 CAA Amendments, required EPA to perform a study of "the hazards to public health reasonably anticipated to occur as a result of emissions by electric utility steam generating

[2] Henceforth, parallel citations of the CAA to the U.S. Code are shown only in the Table of Authorities.

units" of certain hazardous air pollutants, including mercury, "after imposition of the requirements of this Act." CAA section 112(n)(1)(A) also provided for EPA, based on such study, to regulate such utility units if it determined that regulation is "appropriate and necessary."

EPA completed the required study on February 24, 1998, and submitted a report summarizing the results to Congress. Based on the study, and without notice and comment, EPA made a finding on December 20, 2000 that it was "appropriate and necessary" to regulate utility units under CAA section 112. 65 Fed. Reg. 79,825 (Dec. 20, 2000). A petition to review was filed challenging that finding, but this Court held that it lacked jurisdiction to review the finding and that the finding could be reviewed only after EPA issued substantive regulations. *UARG v. EPA*, No. 01-1074, 2001 WL 936363 (D.C. Cir. July 26, 2001).

On March 29, 2005, after notice and comment rulemaking, EPA reversed its December 20, 2004 finding and found that it was not "appropriate and necessary" to regulate utility unit emissions. EPA determined that "the hazards to public health reasonably anticipated to occur as a result of emissions" of mercury from utility units could be adequately addressed by other CAA programs. 70 Fed. Reg. 15,994 (Mar. 29, 2005). Among these programs are the Clean Air Interstate Rule (CAIR), which EPA promulgated under CAA section 110, 70 Fed. Reg. 72,268

(Nov. 22, 2005), and the Clean Air Mercury Rule (CAMR), which EPA promulgated under CAA section 111, 70 Fed. Reg. 28,606 (May 18, 2003).

Certain petitioners challenge EPA's determination that it is not "appropriate and necessary" to regulate power plant mercury emissions under CAA section 112. They argue that CAIR, CAMR and other CAA programs do not adequately protect public health from utility unit mercury emissions. Brief of Government Petitioners, at 22-24, 32-34.

However, scientific evidence supports EPA's position.[3] EPA was more than conservative in concluding that CAIR, CAMR and other CAA programs protect the public from "the hazards to public health reasonably anticipated to occur as a result of emissions" of mercury from utility units.

SUMMARY OF ARGUMENT

EPA properly decided it was not "necessary and appropriate" to regulate mercury emissions from utility units under CAA section 112, because such utility units cannot reasonably be anticipated to pose a hazard to public health. Scientific evidence demonstrates that utility mercury emissions do not pose a public health hazard, particularly after EPA implements the CAIR and CAMR programs. Utility mercury emissions are a tiny fraction of total mercury in the environment, and

[3] All scientific evidence cited in this brief was submitted to EPA during the comment period by the Center for Science and Public Policy and, to a lesser extent, by other parties.

there is no basis to conclude that utility mercury emissions contribute in any meaningful amount to mercury being "biomethylated" and then "bioaccumulated" in fish flesh that is consumed by pregnant women or women of child-rearing age. Finally, EPA's reference dose for the safe level of mercury in human blood is highly conservative and likely overly protective of human health.

ARGUMENT

I. Overview: Utility Unit Mercury Emissions Do Not Pose a Health Threat

Human exposure to mercury (Hg) emitted from utility units is not harmful. To become a potential human health hazard, mercury must undergo a complex chain of bioprocessing and reprocessing (biomethylation) into the compound methylmercury (MeHg), which must be ingested, primarily through fish, in a sufficiently large dose to cause harm.[4]

As EPA has found, the only population at risk of exposure to utility unit mercury emissions are pregnant women or women of child-bearing age who eat large quantities of fish, because high levels of mercury in the fetus can cause developmental disabilities.[5] However, EPA found that "the typical U.S. consumer

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[4] Center for Science and Policy, *White Paper: EPA Mercury MACT Rule Making Not Justified by Science*, at 1 (March 2004) (hereinafter *White Paper*), submitted with the Center's April 30, 2004 comments.

[5] 70 Fed. Reg. at 16,011-12.

eating moderate amounts of a wide variety of low-mercury fish from restaurants and grocery stores is not expected to ingest harmful levels of methylmercury from fish."[6] Thus, according to EPA, only a small subset of pregnant women and women of child-rearing age are even potentially at risk from consuming fish with high levels of mercury. According to EPA, this subset consists of those who are recreational or subsistence fishers eating freshwater fish.[7] But studies demonstrate and EPA concluded that the risk from utility unit mercury emissions to these sub-populations is exceedingly low.[8]

Indeed, after an extensive study of the possible danger posed by utility unit mercury emissions, as required by CAA section 112(n)(1), EPA concluded no more than (1) fish consumption is the primary pathway for human and wildlife exposure to mercury and (2) there is a "plausible link between emissions of mercury from anthropogenic sources (including coal-fired utility units) and methylmercury in fish."[9] EPA admitted that "because of the current scientific understanding of the environmental fate and transport of this pollutant, it is not possible to quantify the contribution of U.S. anthropogenic emissions relative to other sources of mercury, including natural sources and re-emissions from the

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6 70 Fed. Reg. at 16,012.
7 70 Fed. Reg. at 16,012 and 16,021.
8 *Id.* at 16,022-25.
9 65 Fed. Reg. 79,825 and 79,827 (Dec. 20, 2004).

global pool, on methylmercury levels in seafood consumed by the U.S. population."[10] Consequently, EPA found it "*is unable to predict at this time how much, and over what time period, methylmercury concentrations in fish would decline as a result of actions to control U.S. anthropogenic emissions*."[11]

As EPA concluded in the Notice of Proposed Rulemaking, "the relationship between mercury emission reductions from Utility Units and methylmercury concentrations cannot be calculated with confidence."[12] EPA's uncertainty stems from the difficulty of establishing each link from utility emissions of mercury into the atmosphere, to the co-mingling of that mercury with many other anthropogenic and natural sources, to the uncertain global circulation and ultimate deposition of that mercury, to the complex processes by which mercury "methylizes" or is transformed into methylmercury, to the processes by which methylmercury bioaccumulates in fish, to the fish consumption rates of women, and even to the proper reference doses for mercury, that is, the maximum mercury level in human blood considered safe.[13]

It is certain, however, that utility units are a minuscule source of mercury in fish flesh. According to EPA modeling, the median deposition level of mercury is

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[10] Mercury Study Report to Congress, December 1997, Volume I, Executive Summary, p.3-4.
[11] *Id.* (Emphasis supplied).
[12] 69 Fed. Reg. at 4657-58.
[13] *See* discussion *infra* Section V.

only reduced by 8% when utility emissions are zeroed out.[14] Modeling by the Electric Power Research Institute (EPRI) came to roughly the same conclusion: "Computer models run by U.S. EPA and EPRI predict cutting mercury emissions from power plants by 50% will only reduce mercury levels in U.S. waters by an average of 3%."[15] Since only a small fraction of this mercury is transformed into methylmercury (perhaps as low as 0.03%),[16] it is impossible to say that utility unit mercury emissions pose a meaningful threat to health or welfare.

As a result of CAA regulation, utility mercury emissions have declined substantially, from 77 tons in 1995 to about 40 tons in 2003.[17] The CAIR program will result in further, substantial utility unit mercury reductions as a co-benefit to the rule's purpose of reducing utility sulfur dioxide and nitrogen oxide emissions. According to EPA, the CAIR program will reduce "reactive" (or "oxidized") mercury emissions by 70%.[18] Reactive mercury is the type of mercury emitted by U.S. utility units that is likely to be deposited in U.S. waters.[19] CAMR mercury

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[14] 70 Fed. Reg. at 16,019.

[15] L. Levin, EPRI, *A Framework for Assessing the Cost-Effectiveness of Electric Power Sector Mercury Control Policies*, Final Report No. 1005224, Palo Alto, CA (May 2003).

[16] It is estimated that of the 20% local mercury deposits emitted from power plants, 97% goes into sediments, 3% remains in the water, and only 0.03% is converted into MeHg. L. Levin, Valuing Externalities Workshop, U.S. Department of Energy (Feb. 13, 2003) (hereinafter *Valuing Externalities*).

[17] J. Pacyna, E. Pacyna, F. Steenhuisen, and S. Wilson, *Mapping 1995 Global Anthropogenic Emissions of Mercury*, ATMOSPH. ENV. (2003) (hereinafter J. Pacyna, et al).

[18] 70 Fed. Reg. at 16,014/1.

[19] *Id.*

reductions will be in addition to these reductions.

In sum, given that utility units are not a significant contributor to mercury deposition and given that CAIR and CAMR provide for significant utility unit mercury emissions reductions, EPA was more than conservative in its conclusion that utility unit mercury emissions do not pose a hazard to human health warranting even stricter regulation.

II. Utility Emissions Are a Small Part of Total Mercury Emissions

The most prevalent form of utility unit mercury emissions is elemental mercury. Elemental mercury emitted by U.S. utility units in the atmosphere becomes part of a vast global atmospheric reservoir of mercury.[20] As EPA recognizes, U.S. utility units represent a tiny fraction of mercury circulating in the atmosphere:

> Recent estimates (which are highly uncertain) of annual total global Hg emissions from all sources (natural and anthropogenic) are about 5,000 to 5,500 tons per year (tpy). Of this total, about 1,000 tpy are estimated to be natural emissions and about 2,000 tpy are estimated to be contributions through the natural global cycle of remissions of Hg associated with past anthropogenic activity. Current anthropogenic emissions account for the remaining 2,000 tpy. Point sources such as fuel combustion; waste incineration; industrial processes; and metal ore roasting, refining, and processing are the largest point source categories on a world-wide basis. Given the global estimates noted

[20] According to a recent analysis, of the total mercury emitted into the atmosphere from a single source, 20% deposits nearby and 80% disperses into the global atmosphere. *See Valuing Externalities*.

above, U.S. anthropogenic Hg emissions are estimated to account for roughly 3 percent of the global total, and *U.S. utilities are estimated to account for about 1 percent of total global emissions.*[21]

National total mercury emissions from power plants in China, Europe, India, Australia, and Zaire are individually larger than in the United States.[22] With China bringing a new coal-fired powerplant on line every week, it is estimated that China's *annual rate of growth* for mercury emissions (5% per year) will soon equal the current *total yearly* emissions from U.S. power plants (approximately 40 tons).[23] According to a 2003 estimate, China's emissions exceeded 495 tons, as compared with U.S. utility unit emissions of about 40 tons.[24] Annual mercury emissions worldwide from burning vegetation range from 9 to 24 times higher than the amount released from U.S. power plants.[25]

Recent EPRI studies have documented the critical role that intercontinental mercury transport from Asia and other nations plays in determining U.S. mercury

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[21] 69 Fed. Reg. at 4657-58. EPA's estimate that utilities contribute 1% of global mercury emissions may be significantly overstated. Some research indicates that natural mercury emissions may be as much as 58,000 tons per year. Richardson, et al., *Critical Review of Natural Global and Regional Emissions of Six Trace Metals to the Atmosphere,* Final Report, 2001, Table 5.10.

[22] J. Pacyna, et al.

[23] *Id.*

[24] *Id.*

[25] E. G. Brunke, et al., (2001), GEOPHYSICAL RESEARCH LETTERS, vol. 28: 1483-86; H. Friedli, et al., (2003), ATMOS. ENVIRON., vol. 37: 253-67; M. Veiga, et al, 1993, NATURE, vol. 368: 816-17.

deposition.[26] Key findings of this work include:

- "Significant levels" of mercury exit mainland Asia and cross the Pacific to the U.S. In some instances, atmospheric mercury exiting China near Shanghai moved over the Pacific for 400 miles. Other evidence found the same plume crossing the California coast.
- Technical studies show most of the mercury deposited within the U.S. coming from globally distant sources and indicate that most of the mercury appears to originate in Asia, *which releases roughly half of the global human-origin mercury emitted*.
- Research in Florida found evidence that most mercury originates in other countries and is carried west into Florida by the dominant trade winds.
- Another Florida study found that, even though mercury sources in the state (mostly municipal and medical waste incinerators) were controlled to reduce emissions, the mercury levels in Everglades fish did not show a clear pattern of response.[27]

Finally, mercury deposition is not the only source of mercury in the environment available for methylization. The natural mercury emission from Earth's crust is also an important factor controlling mercury distribution in marine waters and the atmosphere. Main sources of mercury to seas are submarine volcanoes, mud volcanoes, and cold gas vents, as are geological processes supplying mercury to soil, oceans, and inland water bodies. When estimates of all

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[26] *See, e.g.*, EPRI Report, *Assessment of Mercury Emissions, Transport, Fate, and Cycling for the Continental United States: Model Structure and Evaluation* (Dec. 2000), *available at* http://www.epri.com\journal\print.as/?id=747.

[27] *Id.*; EPRI, *Research Shows Most Mercury Deposited in U.S. Originates Outside the Country*, EPRI JOURNAL ONLINE (Dec. 22, 2003).

natural sources are considered, including geothermal events under oceans and lakes, U.S. power plants may account for as little as 0.02% of the entire annual world mercury emissions budget.[28]

III. Utility Unit Emissions Do Not Correlate With Natural Methylation

The process by which mercury becomes a human health hazard, biomethylation, is not controlled by the amount of mercury in the environment. Compared to the annual emissions of U.S. power plants, estimated to be about 40 tons, the oceans contain an estimated 100 million tons of naturally occurring mercury, yet only a minute fraction is biomethylated.[29]

The rate of biomethylation in oceans depends upon many complex, interacting processes and species in the ecosystem that have little to do with mercury. For example, single-celled plant algae and zooplanktons, which may contain MeHg, are consumed by other organisms that consequently accumulate MeHg. The size of algae and zooplankton populations, and hence how much MeHg they transmit into the ecosystem, in turn depends on ecosystem factors unrelated to the availability of mercury. Moreover, the processes by which mercury is converted into MeHg are often inefficient, and competing processes can result in the conversion of MeHg back to less toxic forms of mercury compounds.

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[28] P. Rasmussen, (1994), ENVIRON. SCI. & TECH. vol. 28: 2233-41.
[29] *Id.*

If the conversion to MeHg were more favorable, all available mercury in the ocean and aquatic systems might already have been converted into MeHg, leading to lethal concentrations in large, long-lived fishes and marine mammals.[30]

The same natural pathways of mercury biomethylation and bioaccumulation occur in ocean and fresh waters, with similar complex ecosystem dynamics controlling MeHg levels in ocean and fresh waters.[31]

In addition, available statistical evidence does not show a positive association between mercury concentrations in fish and mercury deposition. Concentrations of methylmercury in fish vary and are dependent on four factors: 1) the trophic level of the fish (level of the fish in the aquatic food web); 2) age of the fish; 3) whether the fish is wild or farm-raised; and 4) whether the fish is a freshwater, marine, or estuarine species.[32]

Several studies demonstrate the lack of correlation between mercury emissions and the methylation and bioaccumulation processes. In 2004, the Maryland Department of Natural Resources conducted a study which found that the most important source of mercury for methylation is not external but instead is

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[30] P. Pickhardt, et al., (2002), *Algal blooms reduce the uptake of toxic methylmercury in freshwater food webs*, PROCEEDINGS ON THE NATIONAL ACADEMY OF SCIENCES, vol. 99: 4419-23.

[31] *White Paper*, at 4.

[32] Weiner, et al., *Ecotoxicology of Mercury*, in HANDBOOK OF ECOTOXICOLOGY 409-63 (D. J. Hoffman, et al. eds. 2003).

in situ mercury production within aquatic systems by sulfate-reducing bacteria.[33] The Maryland Study also found that the concentration of mercury varies among ecosystems and is not directly related to the amount of atmospheric mercury deposition, as the ability of the ecosystem to convert mercury to methylmercury, and for the methylmercury to bioaccumulate, depends on many physicochemical variables.[34]

An earlier review similarly found that U.S. power plants contribute in only small amounts to methylmercury level in the Chesapeake Bay.[35] This study found that most (72.5%) of the methylmercury in the system comes from in-situ production annually. Remote transport from rivers contributes about another 20%, and atmospheric deposition only about 7.5%. Thus, factors other than power plant emissions dominate the production of methylmercury in the Bay's ecosystems.

Another recent study of microbial cycling of mercury in sediments of San Pablo Bay, California, noted that "sediment geochemistry (redox, sulfide, pH, organic content, etc.) is a much more important control on methylmercury production than is the absolute mercury concentration."[36] It was found that despite

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[33] Maryland Dep't of Nat'l Resources, Online Publications, *Final Report – Methylmercury Concentrations in Fish from Tidal Waters of the Chesapeake Bay*, at 7 (Nov. 2004), *available at* http://www.dnr.state.md.us/streams/pubs/ad04-1.pdf (hereinafter *Maryland Study*).

[34] *Maryland Study*, at 7-8.

[35] Mason, et al., MARINE CHEMISTRY vol. 65: 77-96 (1999), cited in CPS Comments, at *13.

[36] M.C. Marvin-DiPasquale, et al., ENVIRONMENTAL GEOLOGY vol. 43: 260-67 (2003), cited in CPS Comments, at *13-*14.

the constant level of total inorganic mercury available in all four sampling sites in that study of three open water and one salt-marshland sites, the production and concentrations of methylmercury are enhanced at the biologically active and organically rich marsh wetland site. This study again emphasizes the important fact that production of methylmercury in the natural ecosystems is dominated by factors other than the available amount of inorganic mercury, such as that from utility units or other industrial emissions.

Similarly, a group of geochemists from Princeton University and the University of Louis Pasteur found no significant increase in the mercury found in similar cohorts of yellow-fin tuna caught off Hawaii in 1971 and 1998, despite the continued and increased use of coal-fired power plants around the world (particularly Asia) over this 27-year period.[37] Likewise, in a paper for Science of the Total Environment, experts from the San Francisco Estuary Institute and California Department of Fish and Game reported no obvious increasing trend for mercury (Hg) concentration in tissues of the popular and long-lived sport fish species of striped bass (Morone saxatillis) caught off three widely dispersed locations throughout the San Francisco Bay area over the 1970-2000 period.[38]

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[37] R. Ferguson, Center for Science and Public Policy, *How Safe Are We From the Fish We Eat?* at 6-7 (Sept. 2004).

[38] *Id.* at 7.

IV. Historical and Pre-Historical Data Show Equal or Higher Mercury Levels

Research into historical and pre-historical mercury levels demonstrates that man-made freshwater mercury deposits are not cause for concern. Lakebed sediment measurements of elemental mercury over the past 11,000 years in Minnesota's Elk Lake show that the addition of recent man-made mercury emissions is neither exceptional nor alarming. Today's mean mercury level of about 140 parts per billion (ppb) was exceeded, owing to natural causes, at least seven times in the last 8,000 years, measuring about 350 ppb 8,000 years ago.[39]

Examination of ancient human remains confirms significant natural exposures to MeHg through fish and marine mammals in the diet. For example, eight Alaskan mummies dated to 550 years ago show a mean value in the four infant mummies of 1.44 parts per million (ppm) and a mean value of 1.2 ppm in the four adults, with one value as high as 4.6 ppm.[40] In contrast, Alaska's current population has a range of MeHg exposure with a mean of 0.6 ppm.[41]

Dated fish (1878–1909) indicate elevated MeHg levels associated with natural exposure. These samples, from the Smithsonian Museum, show a mean

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[39] W. Cannon, et al., (2003), GEOLOGY vol. 31: 187-90.

[40] John Middaugh, Testimony to the FDA Advisory Committee on Methylmercury (July 24, 2002) (hereinafter *Middaugh FDA Testimony*).

[41] State of Alaska ENGINEERING BULLETIN No. 29 (Dec. 11, 2002).

level of 0.38 ppm. This compares to 1978 samples with a mean level of about 0.16.[42]

V. Utility Mercury Emissions Are Not a Human Health Concern

There is no convincing scientific evidence to support the assertion of adverse health effects on adults or children from consuming large quantities of fish containing low traces of MeHg; no studies considered by EPA found signs of mercury poisoning. John Middaugh, the Alaska state epidemiologist, in testimony to the Food and Drug Administration concluded:

> Data linking low-level methylmercury exposure to adverse health outcomes are weak. Adverse neurodevelopmental outcomes documented are subclinical [i.e., indirect], detectable only by sophisticated tests of unknown, long-term significance. Results may be limited by potential confounding factors; leading studies have not found similar results, and ongoing studies hold the promise of providing important information in the near future.[43]

After 27 years of experience, a University of Rochester research team concluded in 2000 that clinical studies in American Samoa, Peru, and the Seychelles provided "no evidence that consuming large quantities of fish is associated with adverse effects on adults or children."[44] In a 2003 update to their

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[42] G.E. Miller, et al., *Mercury Concentrations in Museum Specimens of Tuna and Swordfish*, (1972), SCIENCE vol. 75: 1121-22; C. Carrington et al., *A Risk Assessment for Methylmercury in Tuna*, 1997, WATER, AIR AND SOIL POLLUTION vol. 97: 273-83.

[43] *Middaugh FDA Testimony* (July 24, 2002).

[44] G. Myers, et al., 2002, ENVIRONMENTAL RESEARCH vol. 83: 275-85.

ongoing study (the most recent publication at the time of the EPA rulemaking),[45] Myers and his fellow researchers again reported no detectable risk from low levels of MeHg exposure to children whose mothers had consumed large quantities of seafood. The Seychelles study, one of the longest longitudinal studies of children, followed 643 children from before birth in 1989 to nine years of age in the Republic of the Seychelles, an island nation in the Indian Ocean whose population consumes far more fish than the average American diet. The Seychelles children have been evaluated five times – each time, with no harmful effects being detected. Scientific commentary published in The Lancet on the study concludes:

> On balance, the existing evidence suggests that methyl mercury exposure from fish consumption during pregnancy, of the level seen in most parts of the world, does not have measurable cognitive or behavioral effects in later childhood . . . . For now, there is no reason for pregnant women to reduce fish consumption below current levels, which are probably safe.[46]

Gary J. Myers, the senior author of the report noted, "[t]his study indicates that there are no detectable adverse effects in a population consuming large quantities of a wide variety of ocean fish. These are the same fish that end up on

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[45] *See generally* G. Meyers, et al., *Prenatal Methylmercury Exposure From Ocean Fish Consumption in the Seychelles Child Development Study*, THE LANCET vol. 361: 1686-92 (May 17, 2003) (hereinafter *Seychelles Study*).

[46] C.G. Lyketsos, *Should Pregnant Women Avoid Eating Fish? Lessons from the Seychelles,* THE LANCET vol. 361: 1667-68 (May 17, 2003).

the dinner table in the United States and around the world."[47]

Because of the tenuous link between methylmercury concentrations in fish and health risk, EPA narrowed its regulatory focus to select subpopulations that might be of concern. In the first place, EPA eliminated concern as to the consumption of salt water fish and focused on possible concern as to fresh water fish.[48] Yet most Americans do not consume significant quantities of fresh water fish. Nationally, consumption of fresh water fish is less than .05% of total fish and fish products consumed annually in the United States.[49] EPA thus limited its regulatory focus to recreational fishers and subsistence fishers.[50] But EPA was able to dismiss concern as to recreational fishers. Even hypothesizing high levels of consumption of the highest level of methylmercury fish tissue concentrations possibly attributable to utility units assumed by EPA, exposures were well within the range EPA considered safe (even at EPA's conservative reference dose level, as discussed in the next section).[51]

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[47] Press Release, Univ. of Rochester Med. Ctr., No Detectable Risk from Mercury in Seafood, Study Shows (May 16, 2003) (quoting Meyers on his Lancet paper).

[48] 70 Fed. Reg. at 16,012/2.

[49] Total fresh water catch in the United States is reported as 27.4 thousand metric tons. The total food supply from fish and fish products in the United States is reported as 5657 thousand metric tons. Even assuming that all the fresh water catch is consumed domestically, domestic fresh water fish accounts for less than .05% of consumption. Food and Agriculture Organization of the United Nations, *Trade in Fish and Fishery Products, Fish Consumption, Fishers and Fleet Information*, tables CM 1.1 and CM 1.2.

[50] 70 Fed. Reg. at 16,021/2.

[51] 70 Fed. Reg. at 16,022/2-3.

Subsistence fishers posed a more difficult issue because of the lack of adequate data as to the level of fish consumption. But even assuming high fish consumption rates, and even using EPA's conservative reference dose, EPA concluded that, after CAIR, the probability that subsistence fishers would consume fish with unsafe mercury levels was very low. Most subsistence fishers are Native Americans who live in areas not significantly affected by utility-attributable mercury deposition.[52]

VI. EPA's Reference Dose Is Overly Protective

EPA uses a reference dose (RfD) of 0.1 micrograms of MeHg per kilogram of body weight per day (0.1 µg/kg/day). This is based principally on epidemiological data from a study in the Faroe Islands. So far, the Faroe study is the only study suggesting "discernible, insidious [neuropsychological] effects from human consumption of mercury from fish,"[53] but that study has well-known confounding factors. In particular, the Faroe study contained known unique dietary consumption patterns of meat and blubber from pilot whales, which Americans do not consume. The whale meat contained unusually high MeHg concentrations (about 5 times the amount in different species of ocean fish, of

[52] 70 Fed. Reg. at 16,022/1-2.
[53] P. Grandjean, et al., 1997, NEUROTOXICOLOGY AND TERATOLOGY, vol. 19: 417-28.

about 0.3 ppm for both the U.S. and Seychelles Island populations),[54] and the blubber contained polychlorinated biphenyls (PCB) concentrations of ten times the burden level of the U.S. population. PCBs are believed to cause serious neurological problems and would distort the detection of neurological or neuropsychological problems arising from MeHg consumption. In contrast, no PCB contamination is present or confounds the Seychelles study discussed above.[55]

In clinical studies, laboratory rats exposed to PCBs (but not MeHg) showed adverse neurological effects, while rats exposed to MeHg (but not PCBs) showed no effects.[56] Rats exposed to both PCBs and MeHg (as in the Faroe study and the U.S. Great Lakes) showed greater adverse effects than those rats exposed to PCBs only.[57]

The Food and Drug Administration (FDA) has set allowable levels of PCBs that contaminate food and food packaging. Notably, the FDA regulation recognizes that PCBs are "toxic, industrial chemicals … [which] have become a persistent and ubiquitous contaminant in the environment." 21 C.F.R. § 109.30(a).

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[54] *See Seychelles Study*.
[55] *White Paper*, at 7.
[56] J. Bemis & R. Seegal, *Polychlorinated Biphenyls and Methylmercury Act Synergistically to Reduce Rat Brain Dopamine content in Vitro*, ENVIRONMENTAL HEALTH PERSPECTIVES, vol. 107: 879-85.
[57] *Id.*

Yet the FDA has set acceptable and tolerable limits of PCBs that contaminate milk, poultry, eggs, fish, and even "*infant and junior foods*." 21 C.F.R. § 109.30(a)(1)-(9) (emphasis added).

Dr. Kenneth Poirier and Dr. Michael Dourson, former EPA RfD/Reference Concentration Work Group co-chairs, informed EPA that "[t]he Faroe Islands studies are not the proper choice for the critical study for a methylmercury RfD." They also stated that the Seychelles study, discussed above, was a more appropriate basis to establish a reference dose and concluded that "[t]he primary methylmercury exposure in the Faroe Islands is from pilot whale meat, which is eaten infrequently and when eaten tends to be by binge consumption, whereas the fish consumption in the Seychelles is more continuous."[58]

Even using the Faroe study, the 0.1 µg/kg/day reference dose established by EPA is highly conservative and set the reference dose at one-tenth the level of the level deemed to be protective of human health.[59] In contrast, the World Health Organization uses a reference dose of 0.23 µg/kg/day.

Recent and comprehensive research by the Centers for Disease Control and Prevention (CDC), which measured mercury in the blood of women, indicates that people in the U.S. are not being exposed to levels of mercury considered to be

[58] Letter from Drs. Kenneth Poirier and Michael Dourson to Technical Information Staff, EPA (Nov. 29, 2000) (on file with authors).
[59] 70 Fed. Reg. at 16,024/2.

harmful to fetuses, children, or adults. According to the CDC, "[t]he levels reported in this NHANES [National Health and Nutrition Examination Survey] 1999-2000 subsample for maternal-aged females were below levels associated with *in utero* effects on the fetus, or with effects in children and adults (National Academy of Sciences, 2000)."[60]

[60] Ctr. for Disease Control & Prevention, Nat'l Ctr. for Envtl. Health, SECOND NATIONAL REPORT ON HUMAN EXPOSURE TO ENVIRONMENTAL CHEMICALS (2003).

CONCLUSION

For the foregoing reasons, EPA properly decided it was not "necessary and appropriate" to regulate mercury emissions from utility units under CAA section 112, because such utility units cannot reasonably be anticipated to pose a hazard to public health.

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CERTIFICATE OF COMPLIANCE WITH FRAP 32(a)(7)

Pursuant to FRCP 32 and this Court's Order, dated November 29, 2006, counsel hereby certifies that the foregoing Brief of Amicus Curiae Washington Legal Foundation in Support of Respondent contains 4,999 words, as counted by counsel's word processing system and has been prepared in a proportionally spaced typeface using Microsoft Word in 14 point Times New Roman font.

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