

# WHITE PAPER



EPA Mercury MACT Rulemaking Not Justified by Science



The Center for Science  
and Public Policy

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## OVERVIEW

**T**he U.S. Environmental Protection Agency (EPA) is developing a rulemaking that will require the electric utility industry to install controls to reduce mercury emissions from coal and oil-fired power plants.

The action, which is unwarranted, follows congressional direction to EPA (in Section 112(n) of the 1990 Clean Air Act Amendments) to study the reasonably anticipated public health hazards caused by Hazardous Air Pollutant (HAP) emissions from electric utility steam-generating units and to promulgate control standards for utilities *only* if the study showed that such standards were “appropriate and necessary.”<sup>1</sup> However, the scientific facts indicate that the Maximum Achievable Control Technology (MACT) rulemaking is unnecessary. Therefore, EPA’s failure to provide a reasonable and “comprehensive” scientific basis for its rulemaking contravenes the explicit intent of Congress that science should govern a determination to regulate mercury emissions from the utility sector.

Human exposure to elemental mercury (Hg) directly emitted from power plants 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.

EPA completed and released its utility HAP study to Congress and the public in February 1998. In a December 20, 2000, *Federal Register* notice, after an extensive study process, EPA found only that fish consumption is the primary pathway for human and wildlife exposure to mercury and that there is a “plausible link between emissions of mercury from anthropogenic sources (including coal-fired utility units) and methylmercury in fish.”<sup>2</sup> EPA thus decided, pursuant to Clean Air Act Section 112(n), that regulation of utility HAP emissions, including mercury, is “appropriate and necessary.” However, EPA, in its Notice of Regulatory Finding, admits that it is not possible to quantify how much MeHg in fish results from electric utility plants, which is an important issue, as it speaks directly to justification of harm. At the same time, EPA asserts that such information is “not necessary” for the purposes of the finding.

According to a recent estimate,<sup>3</sup> even imposing the strictest mercury-emissions control scenarios would result in only a 2.7% to 3.4% reduction in total U.S. mercury deposition (see chart 1). On June 12, 2003, EPA’s Office of Air & Radiation admitted for the first time that no matter how strictly EPA regulates mercury from coal-fired electricity generation plants, it cannot meet regulatory standards for mercury because too much domestic mercury deposition originates externally.<sup>4</sup> These external sources include natural emissions (55% of total world annual mercury budget) having nothing to do with human activities.

As discussed in this report, there appears to be no scientific basis to conclude that utility power plant emissions of elemental mercury are significant or endanger the public health or welfare upon grounds of emissions sources, transport and deposition patterns, or toxicity and epidemiological studies. In light of EPA’s finding and review of the scientific literature, regulation of emissions of elemental mercury from U.S. power plants more stringent than co-benefits—the amount of mercury concurrently removed by the processes that remove sulfur dioxide (SO<sub>2</sub>) and nitrous oxide (NO<sub>x</sub>), varied by coal type—seems arbitrary and capricious on the basis of health risks.

## MERCURY EMISSIONS: CONSIDER ALL THE SOURCES

U.S. power plants emit little Hg compared to natural processes and non-U.S. man-made sources that dominate the world emissions budget. Furthermore, as a result of co-benefits from recently installed pollution control technologies, U.S. total annual man-made emissions of mercury from power plants have declined significantly, from 77 tons in 1995 to about 40 tons today. At the same time, there has been a significant increase in emissions from other countries<sup>5</sup> (see charts 2 and 3). Consider the following facts about the nature of mercury emissions:

- Worldwide release of mercury from the oceans, volcanoes, wildfires, and non-U.S. man-made sources significantly exceeds emissions from U.S. power plants, which emit less than 1% of the world total<sup>6</sup> (see chart 4).
- National total mercury emissions from power plants in China, Europe, India, Australia, and Zaire are individually larger than in the United States. In two to five years, it is estimated that the annual rate of growth of China's mercury emissions (5% per year) will equal the current total yearly emissions from U.S. power plants (approximately 40 tons).<sup>7</sup> Already, China's emissions exceed 495 tons (see chart 5).
- Annual mercury emissions worldwide from burning vegetation range from 9 to 24 times higher than the amount released from U.S. power plants according to three, new independent estimates.<sup>8</sup>

## THE FAR AND WIDE TRAVELS OF MERCURY

Once released into the air, mercury vapor travels long distances and deposits at distant locations. It is estimated that only 20% of the relatively small amount of mercury emitted by U.S. power plants is deposited locally.<sup>9</sup> The overwhelming sources of mercury deposits in the United States are natural and non-U.S. anthropogenic sources (see chart 6).

The many processes that are involved in the transport and deposition of mercury and its transformation to MeHg remain uncertain.<sup>10</sup> Thus, despite increased mercury emissions from some countries, scientists still do not know how mercury is deposited into the environment.<sup>11</sup> EPA agrees that more research is necessary to address the considerable questions about the load and transport of mercury in the environment.

Data quality problems compound these uncertainties. For example, many non-governmental reports, especially those concerning lake deposition of mercury by rain water<sup>12</sup>, are of little policy relevance because of scientific and statistical errors. These errors include, but are not limited to, consistent adoption of highest data outliers, failure to make corrections for volume dilution factors, and confusion over mercury compounds and their relationship to the biomethylation and bioaccumulation processes.

## WHAT WE DON'T KNOW ABOUT MERCURY AND FISH

EPA has no idea whether reducing power-plant emissions of Hg will reduce MeHg levels in fish. The agency is on record as stating:

*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*

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*relative to other sources of mercury, including natural sources and re-emissions from the global pool, on methylmercury levels in seafood [the same dynamics and uncertainties apply to fresh water fish] consumed by the U.S. population. Consequently, the U.S. EPA is unable to predict at this time how much, and over what time period, methylmercury concentrations in fish [ocean or fresh water] would decline as a result of actions to control U.S. anthropogenic emissions.<sup>13</sup>*

Even though EPA admits that it cannot determine how much, if any, of the MeHg in both ocean and freshwater fish derives from coal and oil combustion, EPA argues that such a determination is “not necessary.”<sup>14</sup> Such an argument seems to vitiate the clear legislative requirement on “appropriate and necessary” controls based on reasonable and “comprehensive” findings.

Consider the following facts on biomethylation and bioaccumulation, the accumulation of substances by living organisms:

- 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 the most minute fraction of this is biomethylated.
  - The rate at which biomethylation occurs in oceans depends upon many complex, interacting processes and species in the ecosystem that have little to do with mercury itself. For example, single-celled plant algae and zooplanktons, which may contain MeHg, are consumed by other organisms that consequently accumulate MeHg.<sup>15</sup> 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. If the conversion of mercury 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.
  - The same natural pathways of mercury biomethylation and bioaccumulation occur in both ocean and fresh waters, with similar complex ecosystem dynamics controlling MeHg levels in the ocean and fresh waters.
  - Research has demonstrated 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.<sup>16</sup>
  - It is estimated that out of the 20% local deposition of mercury emitted from power plants, 97% goes into sediments, 3% remains in the water, and only 0.03% is converted into MeHg.<sup>17</sup>
  - 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
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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.<sup>18</sup> In contrast, Alaska's current population has a range of MeHg exposure with a mean of 0.6 ppm (see chart 7).

- Dated fish (1878–1909) indicate elevated MeHg levels associated with natural exposure. These samples, from the Smithsonian Museum, show a mean level of 0.38 ppm. This compares to 1978 samples with a mean level of about 0.16<sup>19</sup> (see chart 8).

Completely eliminating mercury emissions from U.S. power plants would likely result in no change in MeHg exposure from ocean fish, and a maximum exposure reduction of only 20% or less from freshwater fish. A reduction of human exposure to fish, as discussed on the following pages, is not necessary, as recent findings indicate that current levels of mercury in fish consumed by Americans are not a cause for concern.<sup>20</sup>

## WHY MERCURY IS NOT A HUMAN HEALTH CONCERN

The primary source of exposure to MeHg for most people is through eating fish. (Exposure from fresh water fish consumption is statistically insignificant at less than .05% of total fish and fish products consumed annually in the United States).<sup>21</sup> 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 his 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.*<sup>22</sup>

It is difficult to conduct a suitable MeHg-health epidemiological study in the U.S. population because exposure to MeHg is extremely low, and there are many other confounding risk factors. Other studies have been conducted in communities with high fish consumption. After 27 years of experience, researcher Gary Myers and his research team<sup>23</sup> concluded in 2000 that clinical studies by the University of Rochester researchers 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.” In spite of MeHg exposure, a positive, beneficial association was found in a host of child neurodevelopment tests such as the McCarthy Scales of Children's Abilities memory tests and other language and cognitive tests.

In the most recently released 2003 update to their ongoing study,<sup>24</sup> Myers and his fellow researchers again reported no detectable risk from low levels of MeHg exposure to children whose mothers had consumed relatively 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 is found in the American diet. The Seychelles children have been evaluated five times—each time, with no harmful

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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.*<sup>25</sup>

Gary J. Myers, the senior author of the report noted, *“This 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 the dinner table in the United States and around the world.”*<sup>26</sup>

Moreover, there are well-known public health benefits from fish consumption. Eating fish provides high nutritional value such as vitamins A, E, and C, protein, energy, omega-3 fatty acids, monolipids, iron, and zinc. Omega-3 fatty acids have proven benefits in preventing complications from, for example, diabetes, plus preventing coronary heart disease<sup>27</sup> and arteriosclerosis and complications from arthritis. Seen in this light, what is ill-advised is discouraging fish consumption.

EPA's extreme reference dose (RfD, the amount of a substance that can be safely consumed over a lifetime) of 0.1 micrograms of MeHg per kilogram of body weight per day is derived from the flawed Faroe Islands study (discussed in the next section). This RfD is prompting widespread fish advisories, which, in fact, result in unintended negative impacts on public health, especially for pregnant women who are increasingly afraid of eating fish.

Alaska State Epidemiologist John Middaugh and scientist Grace Egeland said, “Severely limiting consumption of fish and seafood may do more harm than good by reducing the consumption of health benefits and by increasing the consumption of alternative foods that have potential health risks.”<sup>28</sup> They have not been alone in voicing these concerns.<sup>29</sup>

## THE EPA REFERENCE DOSE: CREATING FEAR OF FISH

In 1995, EPA established a reference dose (RfD) of 0.1 micrograms of MeHg per kilogram of body weight per day, based upon the study of 81 children poisoned in Iraq in 1971 because their mothers while pregnant directly consumed seed treated with MeHg (used as a fungicide) instead of planting the seed as intended.<sup>30</sup> However, the aberrant Iraq case is not representative of possible health problems for the U.S. population, as it involves an acute, direct dose of MeHg through grain consumption versus chronic exposure through fish consumption. Realizing that examples of direct poisoning have little relevance to the consumption of fish in the United States, EPA shifted the basis of its RfD justification to epidemiological studies.

When considering more recent and complete epidemiological studies of fish consumption in large populations, EPA could have reasonably developed an acceptable RfD by choosing a safety factor in the range of 3 to 5 rather than using a safety factor of 10 (lowering its calculated value to a value 10 times lower still). At a reference dose of 0.3 or 0.5  $\mu\text{g}/\text{kg}/\text{day}$  recommended by the Agency for Toxic Substances and Disease Registry and the World Health Organization, respectively, there would be far less concern over mercury exposure from fish consumption today and less rush to regulation.<sup>31</sup>

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Instead, EPA now bases its Rfd value of 0.1 µg/kg/day on epidemiological data from the Faroe Islands study. So far only the Faroe Islands study is able to suggest “discernible, insidious [neuropsychological] effects,”<sup>32</sup> but that study has the well-known confounding factors.<sup>33</sup>

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 an unusually high concentration of MeHg (about 5 times higher than the mean concentration found in different species of ocean fish of about 0.3 ppm for both the U.S. and Seychelles Island populations<sup>34</sup>), and the blubber contained a concentration of polychlorinated biphenyls (PCB) 10 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.)

In clinical studies,<sup>35</sup> laboratory rats exposed to PCBs (but not MeHg) showed adverse neurological effects, while rats exposed to MeHg (but not PCBs) showed no effects. 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.<sup>36</sup> In a letter to EPA, Dr. Kenneth Poirier and Dr. Michael Dourson, former EPA RfD/Reference Concentration Work Group co-chairs, provided these scientific findings to the Technical Information Staff at EPA.

In their letter, Poirier and Dourson alerted EPA, “The Faroe Islands studies are not the proper choice for the critical study for a methylmercury RfD.” They then listed three reasons.<sup>37</sup> Poirier and Dourson wrote, in part: “Comparisons of fish consumption between the U.S. and the Faroe Islands should not be used, in part, as a basis of the choice of critical study. The RfD is a daily consumption and choice of study should reflect this exposure whenever possible. In this regard, studies from the Faroe Islands are inferior when compared to studies from the Seychelles. The 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.”<sup>38</sup>

Based on findings issued prior to publication of the new Seychelles Islands study published in *The Lancet*,<sup>39</sup> a recently released World Health Organization (WHO) report<sup>40</sup> suggests a provisional MeHg reference dose that is one half of WHO’s previously suggested value. (The newly revised WHO recommendation is a provisional tolerable daily intake of 0.2 micrograms per kilogram of body weight.)

The WHO panel that issued this provisional recommendation notes the availability of the most recent Seychelles Islands data ahead of its publication in *The Lancet*. However, while the WHO panel is clearly aware of the new Seychelles data, there is no evidence that, at the time that WHO issued its provisional recommendation, it took into account the conclusions ultimately published in *The Lancet*. The new *Lancet* study found that there is no observable health effect associated with fish consumption in which MeHg is present and that the Faroe Islands study should not be the sentinel study upon which assessment of MeHg intake via fish consumption should be gauged. Thus, the urgent need still exists to reexamine what is an acceptable dose in view of the new Seychelles Islands study results.

On the basis of these scientific facts, EPA has no “appropriate and necessary” justification for a rulemaking to limit mercury emissions from U.S. power plants.

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## **UNINTENDED HARM TO PUBLIC HEALTH**

Regulations intended to reduce certain health-threatening risks should be expected to provide benefits in the form of safer, healthier, and longer lives. However, the economic costs of mercury regulation of U.S. power plants—particularly the impacts on income and employment—could worsen individual health and safety and shorten life spans. To the extent that mercury regulation leads to replacing coal and oil-fueled power in the United States, harm to the public health could result.

A recent study suggests that just replacing coal-fueled power in the United States could reduce household incomes by \$125 billion to \$225 billion, increase unemployment by 2.2 million to 4.5 million workers, and induce 14,000 to 25,000 premature adult deaths because energy costs would rise.<sup>41</sup> Minorities, the elderly, and the poor could be hurt the most as they would suffer disproportional impacts. The study did not factor in the collateral impacts on children.

## **ABOUT THE AUTHORS**

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## END NOTES

- 1 Section 112(n) was based on the House version of the Clean Air Act Amendments and was adopted in conference in preference to the Senate provision. Rep. Oxley, one of the House conferees, explained to the House that the conferees did not want EPA to regulate utility HAP emissions unless a comprehensive study showed there was a rigorous scientific basis to do so:

As we all know, the utility industry has been singled out for regulation under the acid rain provisions. The utility industry may also face additional controls for NO<sub>x</sub> emissions for ozone control, and revised PM-10 controls. All of these programs will result in substantial reductions in emissions of conventional and potentially hazardous air pollutants. Even without all of these reductions in air pollution, the health risks from emissions of hazardous air pollutants from power plants are vanishing small, as EPA has repeatedly recognized.

Under the existing section 112 of the Clean Air Act, EPA has addressed the question whether additional regulation of power plants is necessary to control air toxic emissions to protect the public health. EPA, thus far, has studied several substances for which emissions data and some indicator of toxicity exist: arsenic, beryllium, cadmium, hexavalent chromium, formaldehyde, and radionuclides. EPA found that additional regulation of emissions of these substances from power plants was unnecessary. For some other substances listed in S. 1630, such as mercury and other volatile substances, little scientific evidence exists about either emissions rates or effects on public health or welfare. Under the conference agreement adopting the approach that the House included in its bill, these and other scientific issues will be examined, and regulations will be imposed only if warranted by the scientific evidence.

1990 Clean Air Act Legislative History 1177 (Oct. 26, 1990) (Statement of Rep. Oxley).

Thus, section 112(n) of the Clean Air Act was intended to allow EPA to regulate HAP emissions from utilities only if a scientific study “clearly establish[es] that emissions of any pollutant, or aggregate of pollutants, from such units cause a significant risk of serious adverse effects on the public health.” *Id.* To make clear that EPA could regulate utility HAP emissions only if a scientific study demonstrated that these emissions caused a significant risk to public health, § 112(c)(6), which allows EPA to promulgate MACT standards for sources of seven specific pollutants including mercury, was revised to specifically exempt utilities. *Id.*

Rep. Oxley also described the differences between the Senate and House versions of §Section 112(n):

With respect to air toxics generally, the Senate and House bills included provisions that differed substantially with respect to scientific studies, timing, and regulatory requirements. The House provision required that the EPA Administrator perform a 3-year study of the hazards to public health reasonably anticipated to occur as a result of emissions by electric utility steam generating units and report the results of that study to the Congress.

On the other hand, the Senate provision was the result of a complex, and ultimately unsatisfactory, set of negotiations. Unlike the House provision, scientific studies were not to serve as the basis for regulation, but simply were to be included in the docket of the regulatory process leading to regulations. Under the Senate provision, regulations for the control of particulates and mercury would have had to be promulgated no sooner or later than 5 years after enactment.

Rather than accept the Senate provision, the conference favored an approach that adopted the basic House provision. The provision did contain two constructive elements found in the Senate provision; a direction to the National Institute of Environmental Health Sciences to conduct a study on a mercury threshold below which adverse effects on human health are not expected to occur and the requirement that EPA study mercury emissions from all sources. The conferees agreed to the House provisions because of the logic of basing any decision to regulate on the results of scientific study and because of the emissions reductions that will be achieved and the extremely high costs that electric utilities will face under other provisions of the new Clean Air Act amendments. *Id.*

- 2 12/20/2002, 65 Federal Register 79825.
- 3 L. Levin, Valuing Externalities Workshop, U.S. Department of Energy, Feb. 21, 2003, p.28.

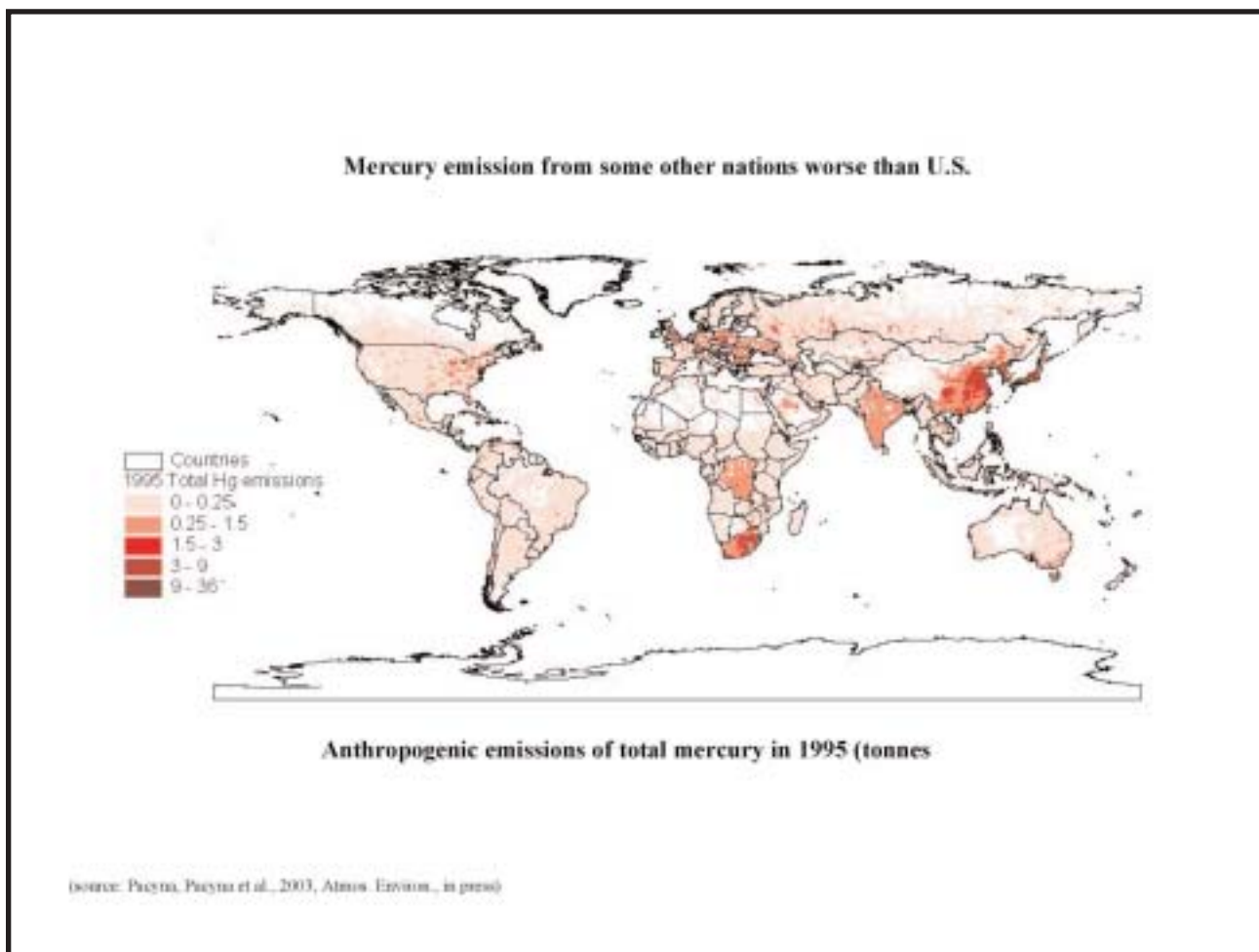
- 4 Energywashington.com (June 19, 2003), an Internet trade publication reports Terry Keating of EPA's Office of Air & Radiation making these remarks to a June 12 Clean Air Act advisory panel. Keating reportedly based his comments on the findings of new NASA satellite data that tracks the international transport of air pollutants.
  - 5 J. Pacyna, E. Pacyna, F. Steenhuisen, S. Wilson, 2003, *Atmospheric Environment*, in press.
  - 6 Ibid.
  - 7 Ibid.
  - 8 E. G. Brunke et al., 2001, *Geophysical Research Letters*, vol. 28: 1483-1486; H. Friedli et al., 2003, *Atmospheric Environment*, vol. 37: 253-267; M. Veiga et al., 1993, *Nature*, vol. 368, 816-817.
  - 9 According to a recent analysis, of the total mercury emitted into the atmosphere from a single source, 20 percent deposits nearby and 80% disperses into the global atmosphere. For most of the United States, a regional model suggests that approximately 60% – 100% of the mercury deposited into the U.S. environment originated as anthropogenic emissions in other countries or from background and natural sources (L. Levin, Feb. 21, 2003, *Valuing Externalities Workshop*, U.S. Department of Energy).
  - 10 “A growing interest during the last decade concerning atmospheric transport, transformation and deposition processes of mercury and the development of new measurement techniques have led to much improved understanding of its occurrence and cycling in the environment. However, there are many processes that are still not well known. Such less known include the removal of gaseous elemental mercury (Hg<sup>0</sup>) from the atmosphere, due to both deposition and chemical transformation (Seigneur et al., 1994; Schroeder and Munthe, 1998). Other processes that are poorly understood are those determining the magnitude and spatial and temporal variability of natural emissions fluxes (Schroeder and Munthe, 1998; Ebinghaus et al., 1999).” (p. 191-192; T. Bergan and H. Rodhe, 2001, *Journal of Atmospheric Chemistry*, vol. 40: 191-212).
  - 11 “Although it is accepted that atmospheric mercury burdens have increased substantially since the pre-industrial period, it is uncertain whether overall atmospheric mercury levels are currently increasing, decreasing, or remaining stable. Measurements over remote areas of the Atlantic Ocean show increasing level until 1990 and a decrease for the period 1990-1994 (Slemr 1996). Measurements of deposition rates suggest decreased deposition at some localities formerly subject to local or regional deposition (see Section 2.1.2.2 below). However, other measurements at remote sites in northern Canada and Alaska show deposition rates that continue to increase (Lucotte, et al. 1995; Engstrom and Swain, 1997). Since these sites are subject to global long-range sources and few regional sources, these measurements might indicate a still increasing global atmospheric burden. More research is necessary; a multi-year, worldwide atmospheric mercury measurement program may help to better determine current global trends.” (p. 2-4 of *Mercury Study Report to Congress*, volume III: Fate and transport of mercury in the environment, EPA 1997).
  - 12 National Wildlife Federation report, *Cycle of Harm*, <http://www.nwf.org/nwfwebadmin/binaryVault/CycleofHarmFinalJ.pdf>.
  - 13 p.3-4, volume I, Executive Summary, Mercury Report to Congress, 1997.
  - 14 Notice of Regulatory Finding, p.10.
  - 15 P. Pickhardt et al., 2002, *Proceedings on the National Academy of Sciences*, vol. 99: 4419-4423.
  - 16 W. Cannon et al., 2003, *Geology*, vol. 31: 187-190.
  - 17 L. Levin. op. cit., p.13.
  - 18 John Middaugh testimony to the *FDA Advisory Committee on Methylmercury*, 7/24/02.
  - 19 Miller et al., 1972 *Science*, V. 175, 1121—1122, “Mercury Concentrations in Museum Specimens of Tuna and Swordfish”; Carrington et al. 1997, *Water, Air and Soil Pollution*, V. 97, 273-283.
  - 20 Comments on NRDC's Testimony on Power Plant Regulation, J. Schwartz, CEI, 5/30/03.
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- 21 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. (Source: 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).
  - 22 J. Middaugh, FDA testimony, 7/24/2002.
  - 23 G. Myers et al., 2000, *Environmental Research*, vol. 83: 275–285.
  - 24 G. Myers et al., 2003, *The Lancet*, "Prenatal Methylmercury Exposure From Ocean Fish Consumption in the Seychelles Child Development Study," vol. 361: 1686–1692 (May 17, 2003).
  - 25 C. G. Lyketsos, *The Lancet*, vol. 361: 1667–1668 (May 17, 2003).
  - 26 G. Meyers quoted in the University of Rochester Medical School's May 16, 2003's press release on his *Lancet* paper.
  - 27 P. Kris-Etherton et al., 2002, *Circulation*, vol. 106: 2747–2757.
  - 28 *Science*, 1997, vol. 278, 1904–1905.
  - 29 "The typical U.S. consumer eating fish from restaurants and grocery stores is not in danger of consuming harmful levels of methylmercury from fish and is not advised to limit fish consumption. The levels of methylmercury found in the most frequently consumed commercial fish are low, especially compared to the levels that might be found in some non-commercial fish from fresh water bodies that have been affected by mercury pollution." (p. O-1 to O-4 of *Mercury Study Report to Congress*, volume I: Executive Summary, EPA 1997).  
  
Concerning the FDA's 2001 revised advisory on MeHg and fish consumption to follow the EPA's "safe" level, Charles Lockwood, in his testimony to the FDA Advisory Committee on Methylmercury, 7/24/02, said: "Almost every obstetrician I talk to knows about the FDA mercury recommendations.... It is tough enough because we terrify pregnant women with every conceivable turn of the page. They are worried about having babies with Down Syndrome or chromosomal abnormalities; they go through all these screening tests, which result in patients being scared out of their wits for no reason, and here's yet another thing coming down the road: you can't eat fish anymore....we are concerned that, in fact, we don't know enough about the neurodevelopmental effect of mercury. The literature has been, at best, unconvincing. We would like to urge the NIH and other federal agencies to support research to establish in a much more rigorous way what mercury does to the developing infant's brain." (Charles Lockwood, chairman of the Obstetrics and Gynecology department at the Yale University School of Medicine).
  - 30 D. C. Rice et al., 2003, *Risk Analysis*, vol. 23: 107–115.
  - 31 EPA's reference dose is overly stringent when compared to those by the World Health Organization (WHO) and the Health and Human Service's Agency for Toxic Substances and Disease Registry (ATSDR) – three times more stringent than ATSDR and five times more stringent than the WHO.
  - 32 P. Grandjean et al., 1997, *Neurotoxicology and Teratology*, vol. 19: 417–428.
  - 33 As a side note, Faroese children were found to have excellent visual contrast sensitivity, plausibly because of "ample supply of dietary omega-3 fatty acids."
  - 34 G. Myers et al.'s May 17, 2003 *Lancet* paper op. cit.
  - 35 J. Bemis and R. Seegal, 1999, "Polychlorinated Biphenyls and Methylmercury Act Synergistically to Reduce Rat Brain Dopamine content in Vitro," *Environmental Health Perspectives*, vol. 107: 879–885.
  - 36 Ibid.
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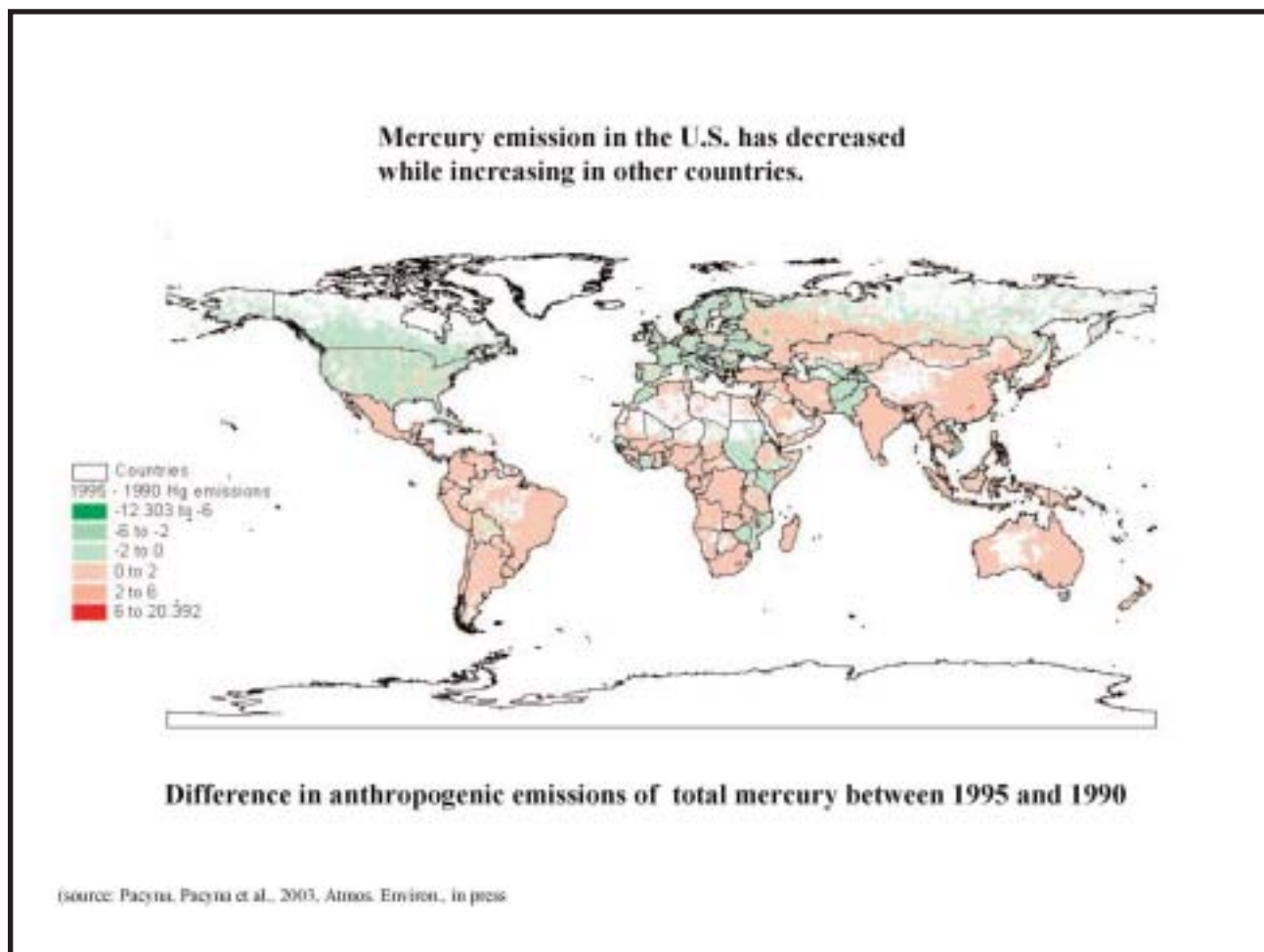
- 37 Choice of study: The Faroe Islands studies are not the proper choice for the critical study for a methylmercury RfD for the following 3 reasons:
1. Methylmercury exposures were confounded with exposures to PCBs. These PCB exposures are in excess of the RfD for Aroclor 1254 by 600-fold, and in excess of the monkey LOAEL on EPA's IRIS by 2-fold. NAS did not account for such excess exposures (TERA, 2000). PCBs are a known neuro- and developmental toxin, similar to methylmercury (Jacobson and Jacobson, 1996). Also, PCBs and methylmercury have been shown to act synergistically for neurotoxicity endpoints (Bemis and Seegal, 1999).
  2. Bolus dose differences between the Faroe Islands fish and pilot whale were approximately 15-fold based on average consumption and mercury concentration (TERA, 2000). Bolus dose differences can have a dramatic developmental effect. It is well established that developmental toxicity differences between alcohol doses over a short time are more harmful than that which occurs by gradual ingestion (Bonthius and West, 1990, as cited by EPA, 2000). Fish consumption in the Seychelles is expected to result in a much smaller mercury bolus dose when compared to pilot whale consumption in the Faroes Islands. This may be a Page 2 Toxicology Excellence for Risk Assessment 1/5/01 2 principal reason why effects may be seen in the Faroes Islands and not the Seychelles.
  3. Comparisons of fish consumption between the United States and the Faroe Islands should not be used, in part, as a basis of the choice of critical study. The RfD is a daily consumption, and choice of study should reflect this exposure whenever possible. In this regard, studies from the Faroe Islands are inferior when compared to studies from the Seychelles. The 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. The complete letter can be found at this Internet address:  
<http://216.239.39.100/custom?q=cache:zcgrA1ohMhAJ:www.tera.org/news/Final%2520letter%2520to%2520EPA%2520Hg.pdf+Bemis+and+Seegal+mercury&hl=en&ie=UTF-8>.
- 38 Ibid.
- 39 G. Myers et al., 2003, *The Lancet*, "Prenatal methylmercury exposure from ocean fish consumption in the Seychelles child development study," vol. 361: 1686–1692 (May 17, 2003).
- 40 <ftp://ftp.fao.org/es/esn/jecfa/jecfa61sc.pdf>.
- 41 Mortality Reductions from Use of Low-cost Coal-fueled Power: Analytical Framework, Klein and Keeney, Dec. 2002.
-



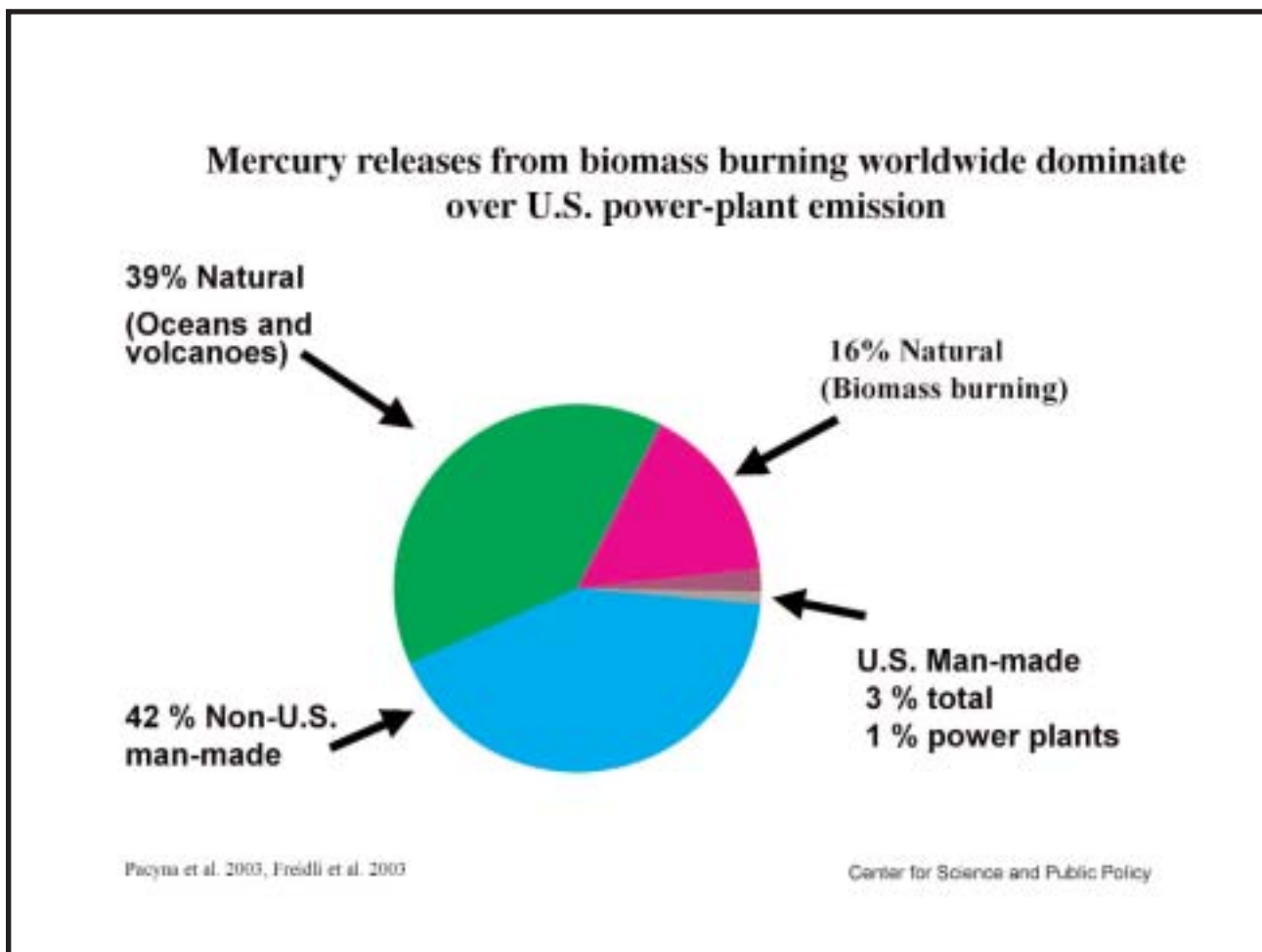
# CHART 2



## CHART 3



# CHART 4



## CHART 5

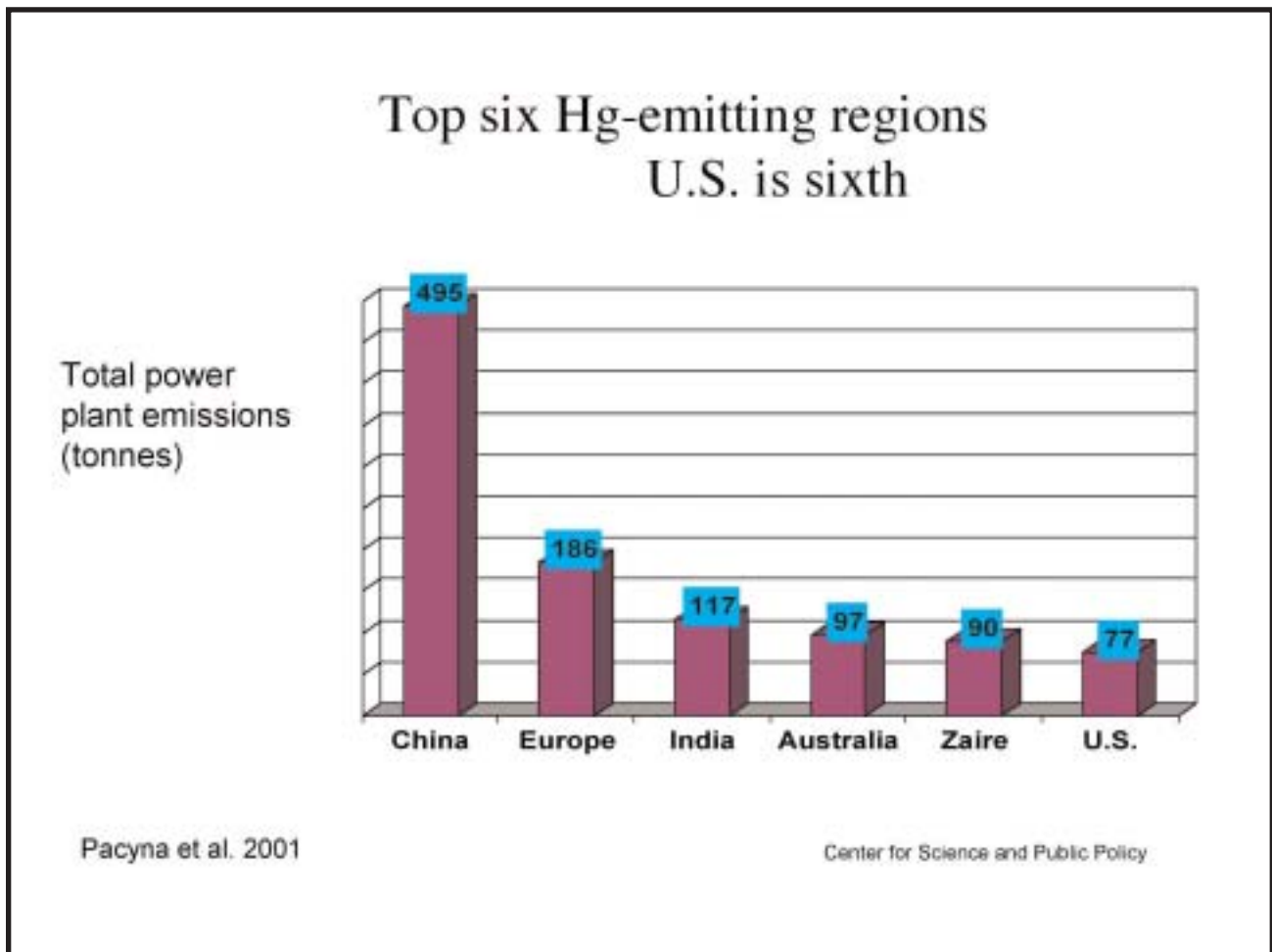
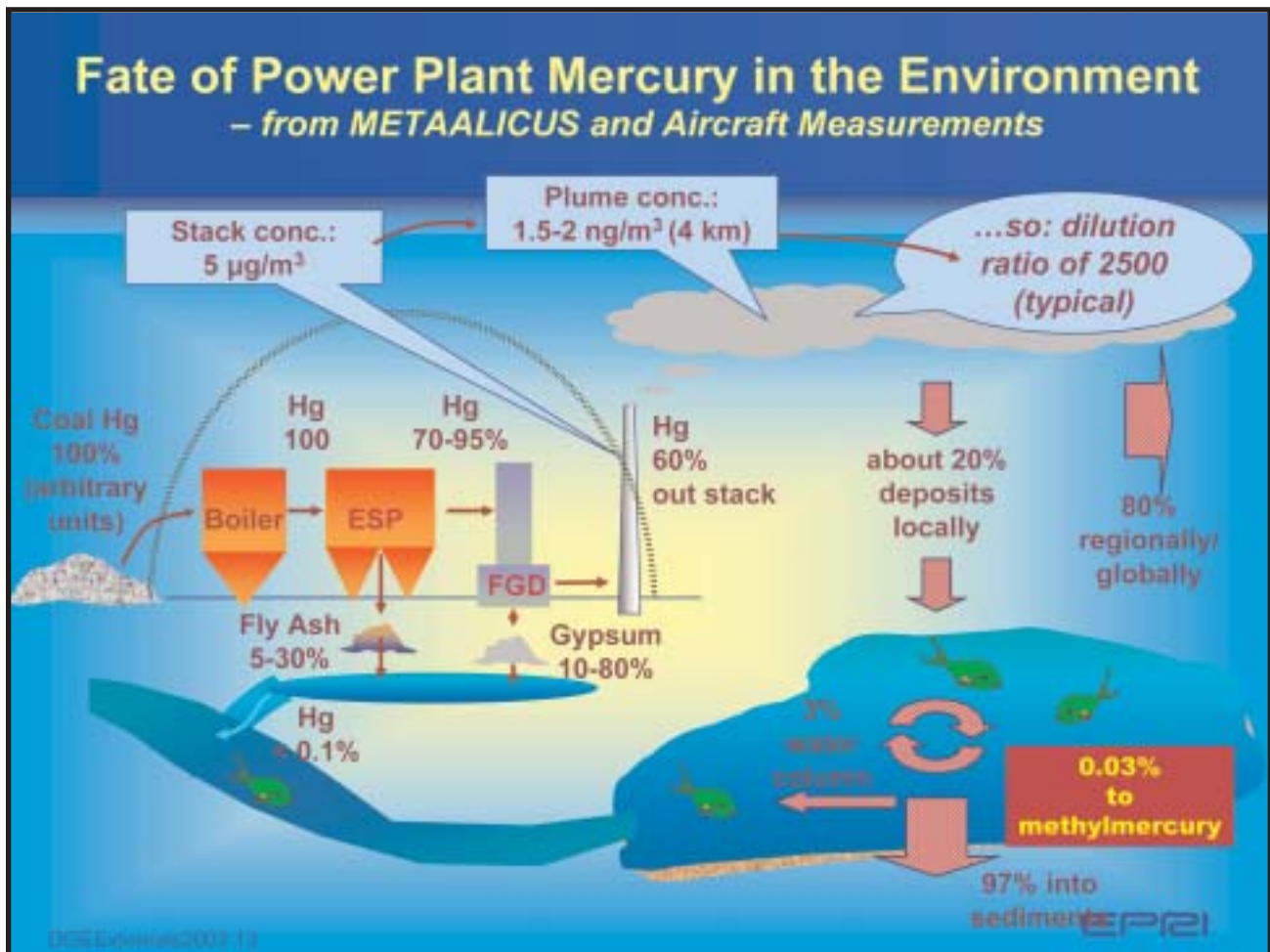
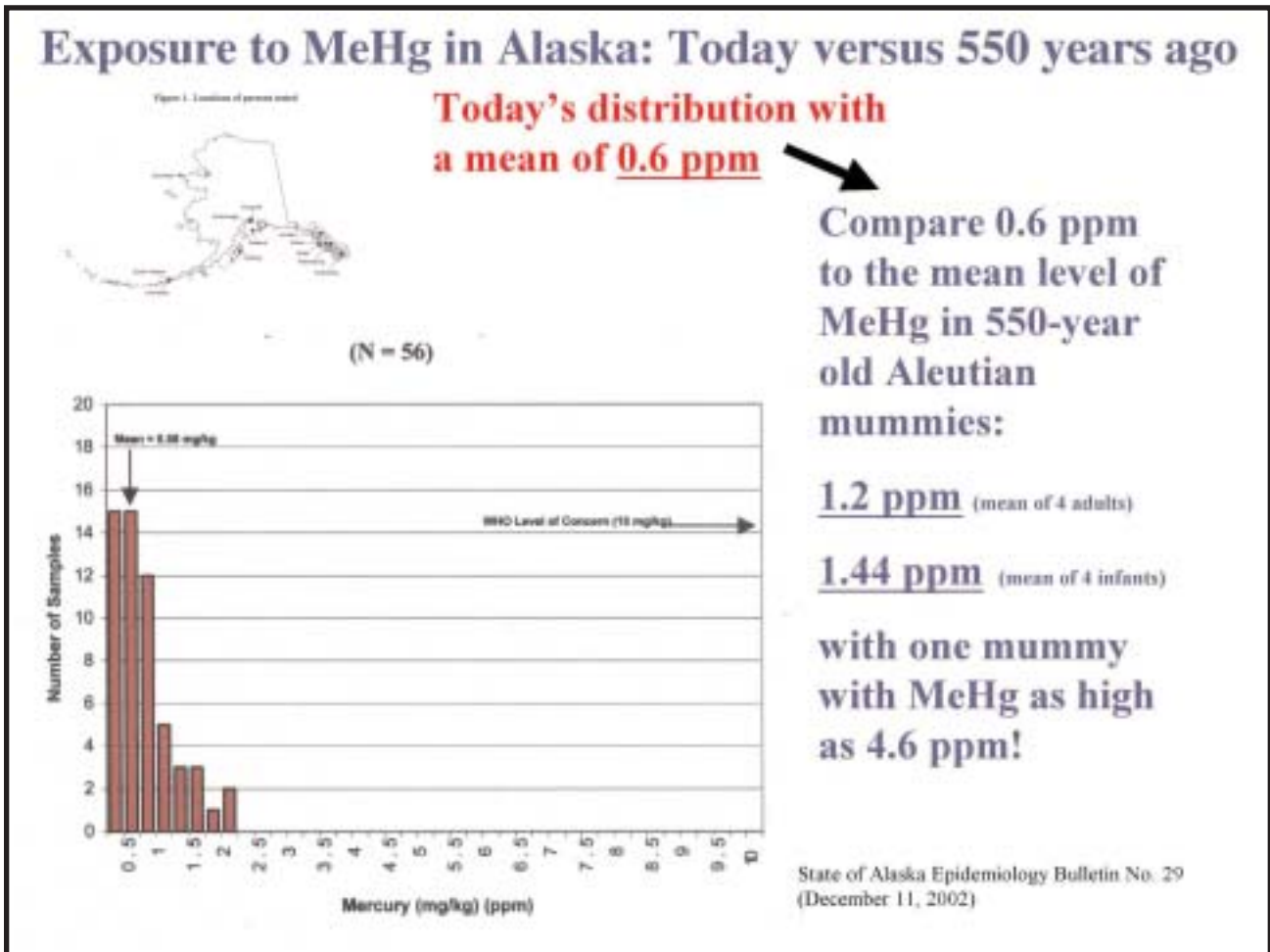


CHART 6

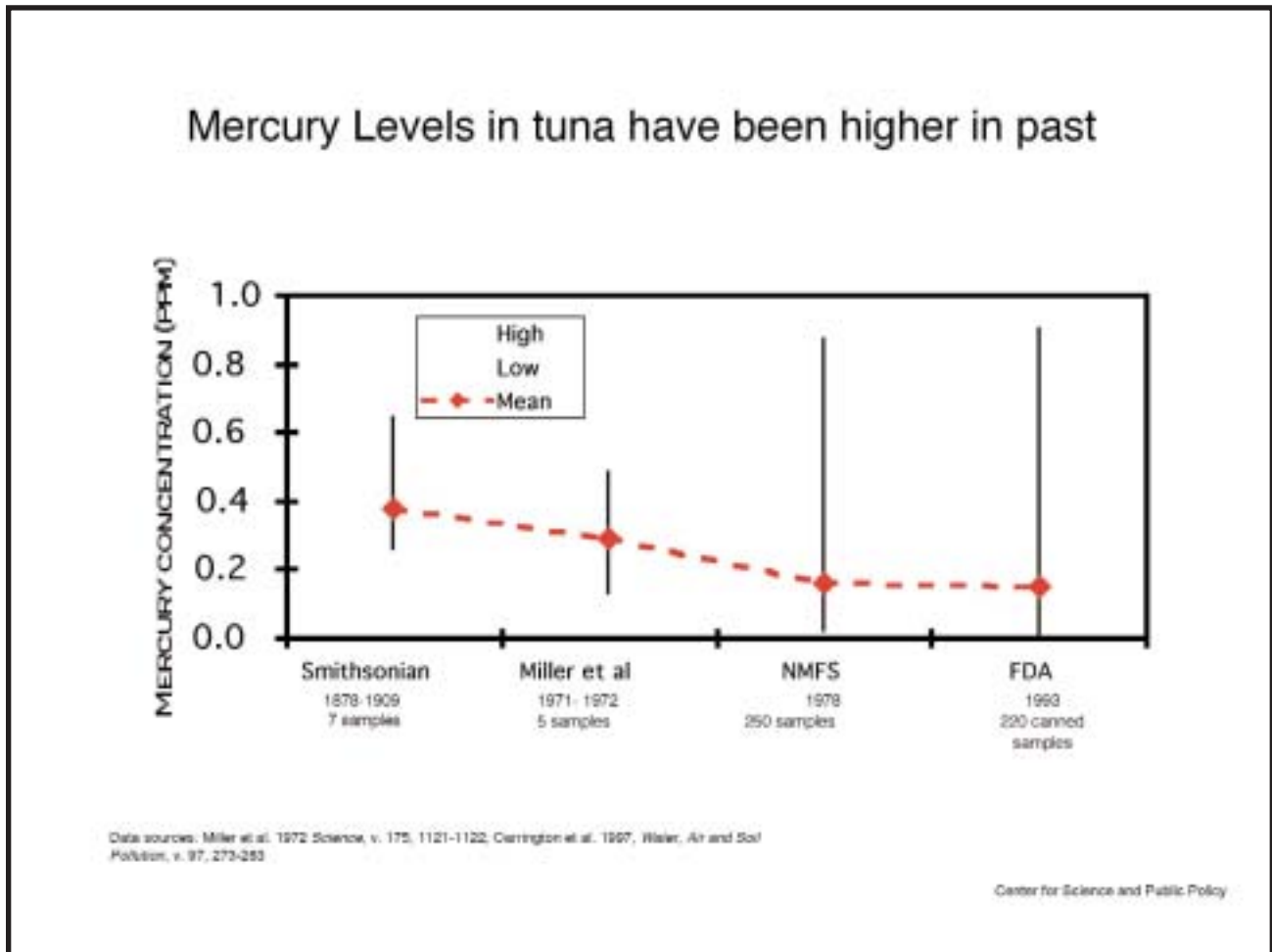


Source: Levin, Valuing Externalities Workshop, U.S. Department of Energy, Feb. 21, 2003, p.13.

CHART 7



# CHART 8



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