UNITED STATES
ENVIRONMENTAL PROTECTION AGENCY
(December 29, 2004)

COMMENTS OF THE CENTER FOR SCIENCE AND PUBLIC POLICY

EPA's Proposed National Emission Standards for Hazardous Air Pollutants; and, in the Alternative, Proposed Standards of Performance for New and Existing Utility Steam Generating Units: Notice of Data Availability (as issued in Federal Register, vol. 69, no. 230, December 1, 2004, 69864-69878)

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Critical Comments on EPA's Proposed National Emission Standards for Hazardous Air Pollutants; and, in the Alternative, Proposed Standards of Performance for New and Existing Utility Steam Generating Units: Notice of Data Availability (as issued in *Federal Register*, vol. 69, no. 230, December 1, 2004, 69864-69878)

Center for Science and Public Policy (CSPP)
(December 2004)

On December 1, 2004, EPA solicited additional public comments on both the methodology and feasibility in its proposed "Clean Air Mercury Rule (CAMR) under the Clean Air Act concerning coal- and oil-fired electric utility steam generating units (power plants)" which represents "the first-ever Federal action to regulate mercury from this source category."

It is clear that the foundational premise for regulatory action by EPA is health protection for all Americans - especially women of childbearing age and their fetuses, infants and young children. Therefore, it is critical to point out that EPA's supposed public health "benefits" from reducing levels of trace mercury in fish via reductions in power plants emissions are largely hypothetical, unverifiable, counterproductive and potentially dangerous.

As demonstrated both here and elsewhere[1], a critical review of the multidisciplinary literature on mercury and related health issues finds that both epidemiological and clinical data suggest no actual danger to average Americans from consuming a wide variety of fish from our restaurants and grocery stores, but there *is* much potential harm from avoiding or restricting fish consumption.

Again, **it is increasingly obvious that EPA does not possess the capability to measure or detect the "benefits" from its CAMR in any actual epidemiological or clinical settings**, except through several artificially imposed links in its current modeling efforts. These are plagued with both unjustified and unverifiable assumptions concerning how inorganic mercury (Hg) from U.S. power plants would ultimately be tracked and directly linked to trace levels of methylmercury (MeHg) in fish, and to finally affect neurodevelopment of fetuses, infants and young children, and cardiovascular health in adult Americans.

In its current Notice of Available Data (NODA), EPA seeks specific comments on two particular components concerning its regulatory options and cost-benefit analyses:

(1) Electric Utility Sector Modeling and Hg Speciation, and
(2) EPA's Proposed Revised Benefits Assessment.

CSPP herein focuses specific criticisms on component (2) of the new NODA in the form of detailed science-based comments on several selective statements (quoted in bold-faced letters) in "EPA's Proposed Revised Benefits Assessment" (pp 69872-69878 of *Federal Register*, vol. 69, no. 230, December 1, 2004).[2]

(A) "As stated in the proposed CAMR [Clean Air Mercury Rule], Hg exposure is both a domestic and a global issue. From a domestic perspective, power plants are one source of Hg air emissions, but there are other domestic sources of man-made Hg. Mercury also enters the atmosphere from a variety of natural processes, including, for example, volcanic eruptions, groundwater seepage, and evaporation from the oceans. EPA currently does not have an inventory of natural or re-emitted sources suitable for modeling purposes." (p. 69873)

Natural sources of mercury emissions dominate the small amount of mercury emissions from U.S. power plants. This fact brings into stark reality that any meaningful control of mercury emissions toward a realistic "reduction" in mercury deposited on U.S. soils will be almost impossible. There are well over 5,000 surface and submarine volcanoes in the world, with about 50 to 60 eruptions each month, according to the Smithsonian Institution.[3] Volcanic degassing may be the single largest source of ocean and atmospheric mercury. For example, at Roaming Mountain, Wyoming, researchers measured mercury emanating from the clay hillside at up to 2,400 nanograms per square meter per hour. By comparison, background levels away from geothermal areas range from zero to 10. So Hg emissions from active geothermal areas could be tens and hundreds times more than from other background areas.

For obvious reasons, volcanic degassing and other geothermal activities as dominant sources of mercury have not received much attention or have been downplayed. For example, EPA staff provided the Administrator with an outdated volcanic accounting study. **Figure A1** clearly shows that EPA's current adopted value for the annual contribution of atmospheric mercury by volcanic eruptions and degassing is significantly *under* accounted for by about a factor of 6 to 7. When adjusted to reflect a more accurate accounting of volcanic Hg emission (**Figure A2**), U.S. power plant contributions to the annual estimated mercury budget world-wide fall to an insignificant 0.8% or less.

Figure A3 maps the range of potential volcanic activity in the Western U.S. These are also potential sources of enormous mercury degassing and deposition, especially Yellowstone National Park (see below).

Figure A4 emphasizes that the pools of mercury stored in U.S. forests and peat lands (covering less than 2% of U.S. area) swamp the 100-150 tons total annual anthropogenic Hg emission from U.S. sources.

Taken together, these figures support EPA's admission of poor accounting for (a) natural sources of mercury emission and (b) the large pool of background mercury at all times available for emission from the natural ecosystems and geological settings within the U.S.

Yellowstone National Park is just one such geological reservoir of mercury. A report issued last fall by the Idaho National Engineering and Environmental Lab[4] showed that several places in Yellowstone Park have higher levels of airborne mercury than power plants. It went on to say that Yellowstone could emit or exceed as much mercury as all of Wyoming's eight coal-fired power plants combined.

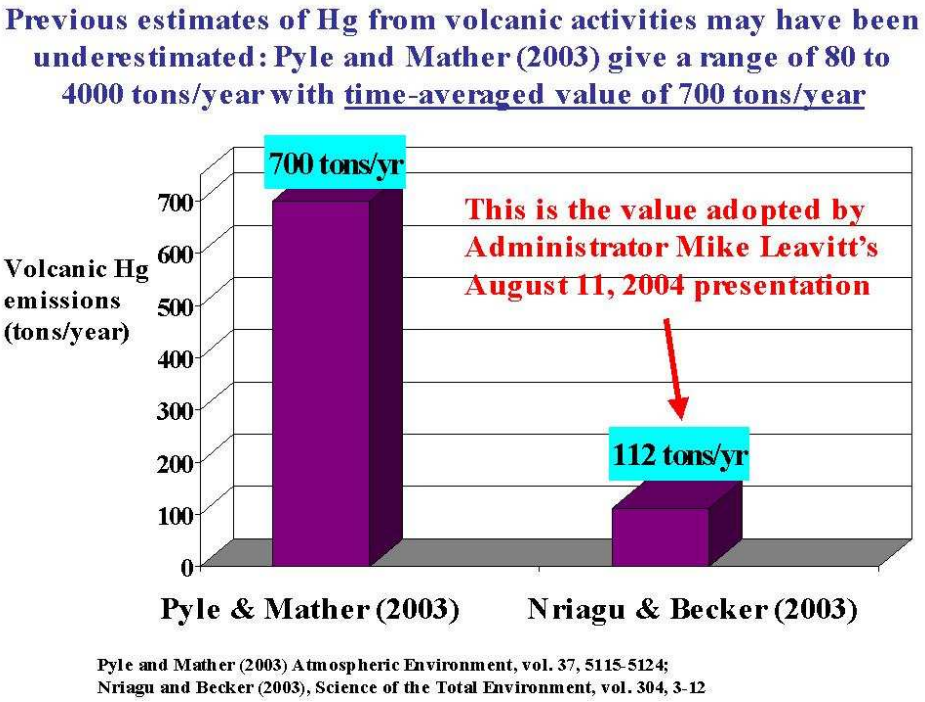
At Yellowstone Lake, researchers have discovered submerged faults, explosion craters, domal features, hydrothermal vents, lava flows extending far out into the lake and much more. Mercury may propagate from these natural features up through the food chain transforming into MeHg in native cutthroat trout.

And since Yellowstone is the headwaters of important tributaries to the Missouri (Yellowstone River) and Columbia (Snake River), no one knows how far the natural contamination carries through the Earth's air and water systems.

However, the mercury presence and emissions were noted by experts to pose no danger to park rangers or visitors. Even native grizzly bears who consume up to 400 lbs. of spawning cutthroat trout exhibit no ill effects, according to researchers with the Interagency Grizzly Bear Study Team.

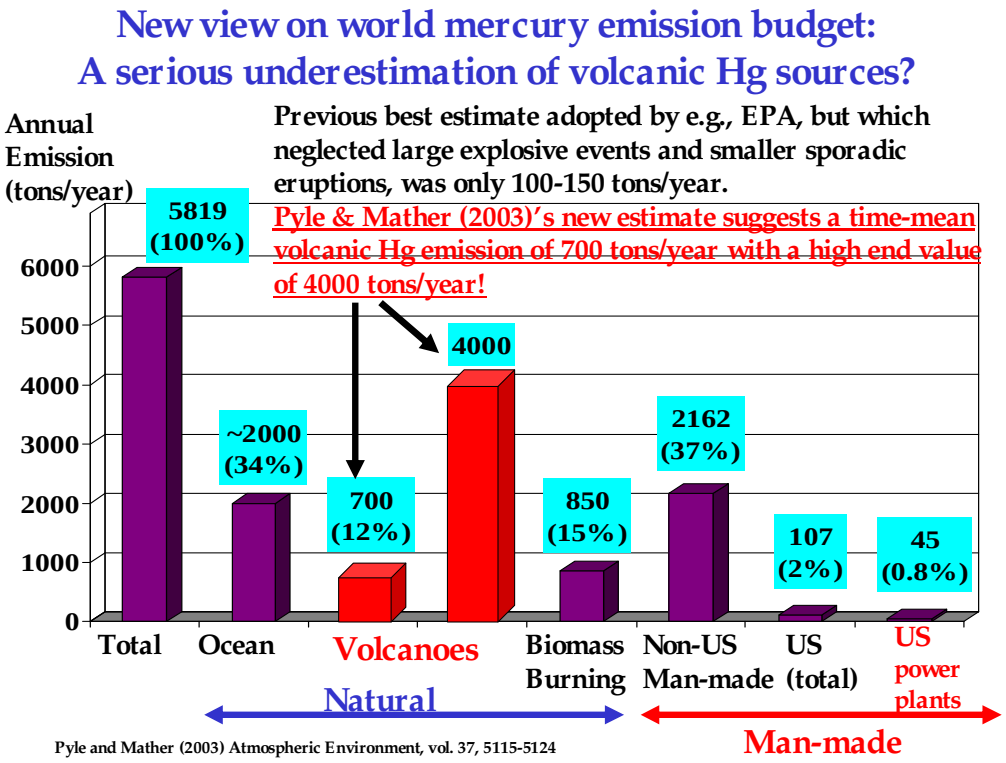
Thus, the most important question for EPA's Hg emission and deposition modeling team to answer confidently is whether the proposed CAMR to control mercury emissions from U.S. power plants can assure *any* consequential "reduction" of mercury deposition in U.S. soils, leading to *any* reputed public health "benefits."

Figure A1



Pyle and Mather (2003) Atmospheric Environment, vol. 37, 5115-5124;
Nriagu and Becker (2003), Science of the Total Environment, vol. 304, 3-12

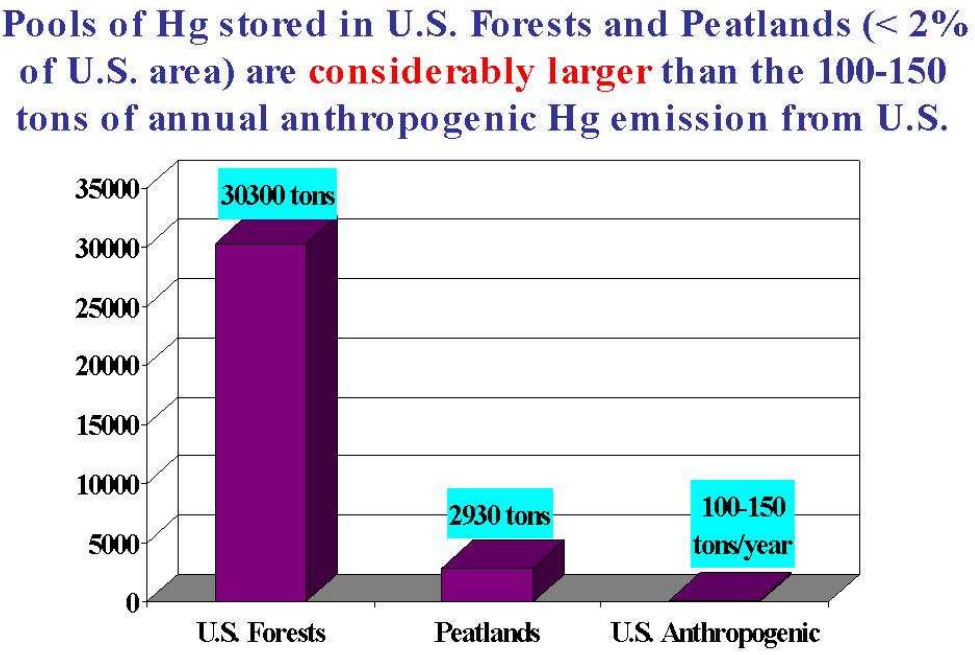
Figure A2



Pyle and Mather (2003) Atmospheric Environment, vol. 37, 5115-5124

Figure A3

Figure A4



Grigal (2003), J. Environmental Quality, vol. 32, 393-405

(B) "These measurements may improve our understanding of the atmospheric reactions that … ultimately impact fate and transport of emissions originating in Asian countries and other international sources. This research is, therefore, directly relevant to the first step of our preliminary proposed revised benefits methodology, as it affects our ability to estimate the U.S. power plant contribution to total Hg deposition within the U.S. " (p. 69873)

Figure B1 provides another important challenge for EPA's Hg emission and deposition modeling team. It shows the results from recent measurements of Hg content in Illinois soils clearly suggesting that atmospheric deposition from any past or current U.S. power plants is insignificant compared to the large quantity of background Hg already resided in soils across the state. This is why it is extremely difficult for EPA to convincingly show that the Hg from U.S. power plant emissions will be selectively filtered by the ecosystem components in Illinois (or anywhere else) to bring about increased levels of MeHg in freshwater fish while ignoring the large pool of natural Hg in the native ecosystem. It is therefore extremely difficult, if not impossible, for EPA to plausibly demonstrate the assertion that its proposed CAMR can/will bring about any direct, measurable improvement in public health.

Figure B2 provides a recent Hg emission and deposition budget analysis for the northeastern Chinese city of Changchun. Scientists found that of that 7.1 tons of Hg emitted by the city of Changchun, only less than 12% of coal-fired power plant Hg was deposited back into the local area, while most escaped as contributions to regional and global cycling of Hg.

This scenario of the local Hg emission and deposition budget at Changchun, China may serve as a useful model verification target for the EPA's Hg emission and deposition modeling team under a wide range of meteorological and climatic conditions and settings.

Figure B1

What Edward Krug & Derek Winstanley confirmed:

Man-made atmospheric deposition of Hg is a VERY SMALL contributor to the huge amount of natural Hg in Illinois and US soils

(1) It has been estimated that "anthropogenic activities could have increased world soil Hg content by [only] 0.02 percent."

(2) From the measured high Hg content in Illinois soils, it would take 9000 years at the currently measured atmospheric deposition rate to dump all the Hg to the top 380-cm of Illinois soils.

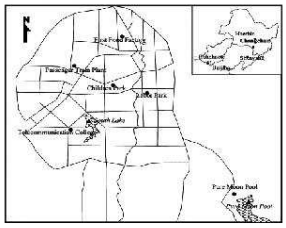
(3) If assuming the average Hg in the top 140-cm of US soils to be about 10 ppb, it would take 14,000 years at the current atmospheric deposition rate to do it.

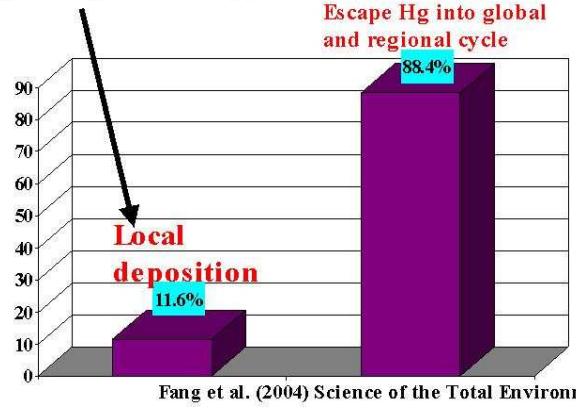
Krug and Winstanley, 2004, Hydrology & Earth System Sciences, vol. 8, 98-102

Figure B2

No local Hg hotspot in Changchun, NE China:
The urban district of Changchun
is a net Hg source for regional and global Hg cycle

"Only 11.6% of Hg, emitted from coal combustion, deposited into land surface in urban district and the rest [of Hg] participated in regional or global cycle."





The metropolitan city of Changchun has 2.92 million people, covers an area of 158 km^2 and emits 7.1 tons of Hg in 2001

Fang et al. (2004) Science of the Total Environment, in press

(C) "The HYSPLIT [Hybrid Single Particle Langrangian Integrated Trajectory] model has also been used extensively in the Great Lakes and Chesapeake Bay watersheds to analyze source-receptor relationships for Hg deposition in these areas." (p. 69873)

Figure C1 represents still another important observational target for EPA's modelers. First, it is important to point out that the mercury in rainwater or moist air exists mostly in the dissolved ionic form of mercury (Hg^{2+}) rather than methylmercury (MeHg) - the biologically active form of mercury that may affect human health at extraordinary dose levels. **Figure C1** shows that most (72.5%) of the MeHg in the Chesapeake Bay ecosystems comes from *in-situ* production. Remote transport of MeHg from rivers contributes about another 20%, and atmospheric deposition sources may contribute toward production of as little as 7.5% of MeHg in the Chesapeake Bay's ecosystems. Such a scenario of the MeHg budget clearly emphasizes the need for better scientific understanding of the complex physical, chemical and biological factors controlling the production *and destruction* of MeHg, and why the levels of MeHg in an ecosystem *do not* depend directly on available amounts of inorganic mercury (i.e., from background or power plant emissions).

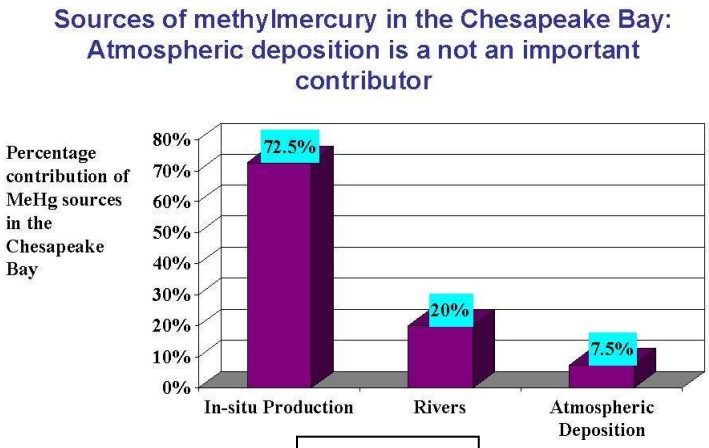


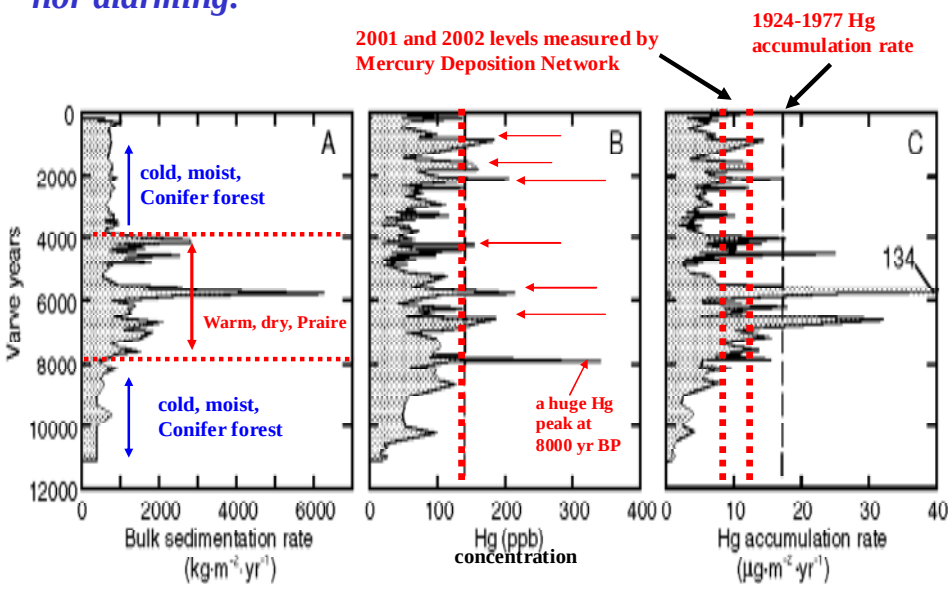
Figure C1

Reference: Mason et al. (1999)

(D) "EPA is seeking comment on its proposed use of REMSAD [Regional Modeling System for Aerosols and Deposition] and CMAQ [Community Multiscale Air Quality] to evaluate how Hg moves through the atmosphere and how it ultimately would be deposited. … We recognize the need to complement the MDN [Mercury Deposition Network] wet deposition measurements with dry deposition measurements because it is not clear how significant dry Hg deposition is to total ecosystem deposition. Currently, there is no recognized field method of measuring dry deposition. " (p. 69874)

CSPP agrees with EPA's call for increased understanding about how dry Hg deposition adds to the total ecosystem deposition. **Figure D1** shows a new set of Hg deposition data obtained from sedimentary cores representative of the past 11,000 years in Elk Lake in Minnesota. These data might serve as an important benchmark in wet and dry depositions under a very wide range of meteorological and climatic conditions as well as a variety of mercury sources (for example, Hg-enriched dusts and sands from nearby Nebraska sand hills or Hg from local and regional forest fires) for EPA's REMSAD and CMAQ models to demonstrate both the correctness and robustness of their atmospheric transport, chemistry and deposition modules.

Figure D1 **Measurements of Hg over the past 11,000 years in Elk Lake, MN show that today's Hg level *is neither exceptional nor alarming*.**



Cannon et al. (2003), *Geology*, vol. 31, 187-190 (figure courtesy of Bill Cannon, USGS)

(E) "As we explained in the proposed CAMR, the main route of human exposure to MeHg is through consumption of fish containing elevated levels of MeHg. Accordingly, to estimate the changes in human exposure to MeHg that may result from reductions in Hg emissions from U.S. coal-fired power plants, we must first quantify how changes in Hg deposition from U.S. coal-fired power plants (forecasted using the models described above) translate into changes in MeHg concentrations in fish." (p. 69874)

As noted in **comment (E)** here and in **comments (A)-(D)** above and **comment (F)** below, EPA's current assertion that a reduction in Hg emissions from U.S. power plants will lead to a reduction in the MeHg concentrations in fish are not scientifically convincing or correct, and hence the effort is almost certainly wrong-headed, wasteful of the limited resources and potentially harmful to public health generally, and women and children specifically.

Figure E1

No evidence of increasing trend or any change in Hg of deep-sea fish (blue hake): 1970s versus 1880s fish

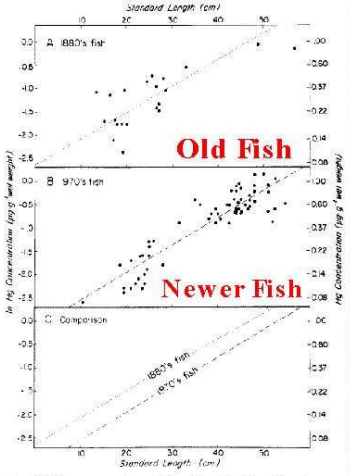


Figure 2. Linear regression analysis of the natural logarithm of mercury concentration vs. length in the deep-sea fish *Antimora rostrata*. (A) Fish collected in the 1880s; (B) fish collected in the 1970s; (C) comparison of regression lines for the 1880s fish and 1970s fish.

"To test for a change in mercury content in the last century, two samples of the deep-sea fish named blue hake (*Antimora rostrata*) were analyzed. *Antimora rostrata* is resident throughout the world's oceans at depths of 1000-3000 m but does not venture into depths shallower than 800 m [actually about 200 m in the cold waters of the polar region]; therefore, this deep-sea species is not exposed to local estuarine, coastal, or atmospheric inputs of mercury. A sample of 21 specimens collected in the 1880s was compared with a sample of 66 specimens collected in the 1970s in the western North Atlantic Ocean. In both recent and old fish mercury increased as a function of length, but comparison of the two concentration vs. length relationships shows that there has not been an increase in mercury concentration in deep-sea fish in the last century. This result supports the idea that the relatively high concentration of mercury found in marine fish that inhabit the surface and deep waters of the open ocean result from natural processes, not 20th century industrial pollution."

Barber et al. (1984) Environmental Science & Technology, vol. 18, 552-555; Barber et al. (1972) Science, vol. 178, 636-639

Figure E1 shows that although one can find a clear increase of mercury concentration in the tissue of the deep sea fish (blue hake) caught from western Atlantic waters as the size of the fish increases, one can hardly see any significant changes in the fish tissue mercury-size relation for fish samples caught in 1880s when compared to the modern samples caught in the 1970s. This research clearly suggests that mercury concentration in world ocean fish is *not likely to be changed or modified* by any amount of alteration of inorganic Hg sources (either anthropogenic or natural). This is why the claim that the current EPA CAMR will lead to a measurable reduction in MeHg accumulated in world ocean fish or even fish from local U.S. lakes is *factually misleading*. Additional evidence and comments by CSPP follow.

Figure E2 shows recent results by Kraepiel et al. (2003, Environmental Science & Technology, vol. 37, 5551-5558) which found no clear increase in the mercury levels of Yellowfin tuna caught in 1998 relative to a similar cohort caught in 1971. The theoretical expectation (similar to EPA's) was that the methylmercury concentration "should have increased by 9 to 26%" over the interval "if methylation occurred in the mixed layer or in the thermocline [of the Pacific oceans]." The theory was *not* proven. We further note that Zhang et al. (2002, Ambio, vol. 31, 482-484) has recently estimated that China's mercury emissions from coal combustion are increasing at the rate of 5% per year (from available data from 1978 through 1995), which is consistent with the theoretical expectation of increase in the amount of methylmercury in the waters of the Pacific Ocean *if* the Hg-to-MeHg conversion process is sensitive to industrial emissions. To the contrary, Kraepiel et al. (2003) clearly concluded that "[s]uch an increase is statistically inconsistent with the constant mercury concentrations measured in tuna. We conclude tentatively that mercury methylation in the oceans occurs in deep waters or in sediments." (p. 5551). This is why the relatively small man-made sources of mercury emissions can neither overwhelm nor directly alter the natural cycling of mercury in the environment and biosphere.

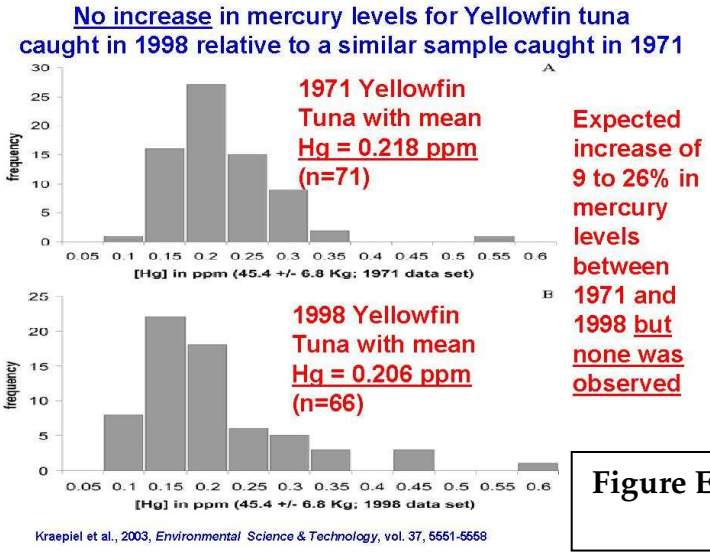
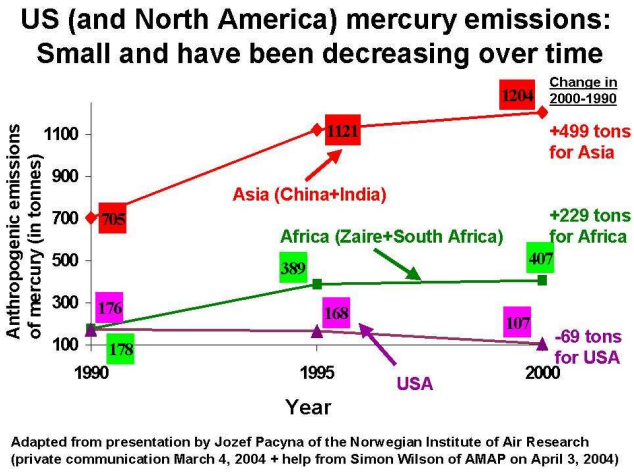


Figure E2

Independent results shown in **Figure E3** support and update the finding of Zhang et al. (2002) that industrial Hg emissions from China (and India) are increasing significantly from 1990 to 2000 and that amount, both in the absolute amount and the rate of increase, dwarfed the rather small amount of industrial Hg emissions from the U.S. EPA's CAMR should seriously consider

Figure E3



and weigh-in this important fact if there is to be any effective Hg emission management rulings.

Figure E4 shows additional new evidence against any increasing trend in mercury levels in fish by examining concentrations in tissue of striped bass from the San Francisco Bay area over the period 1970-2000. The study's findings also clearly show that in any given year there is at least one striped bass sample containing mercury values above EPA's consumption advisory threshold value of 0.5 ppm. Perhaps even more significant, those striped bass with mercury concentration values above 0.5 ppm had no apparent connection to power plant Hg emissions.

Figure E4

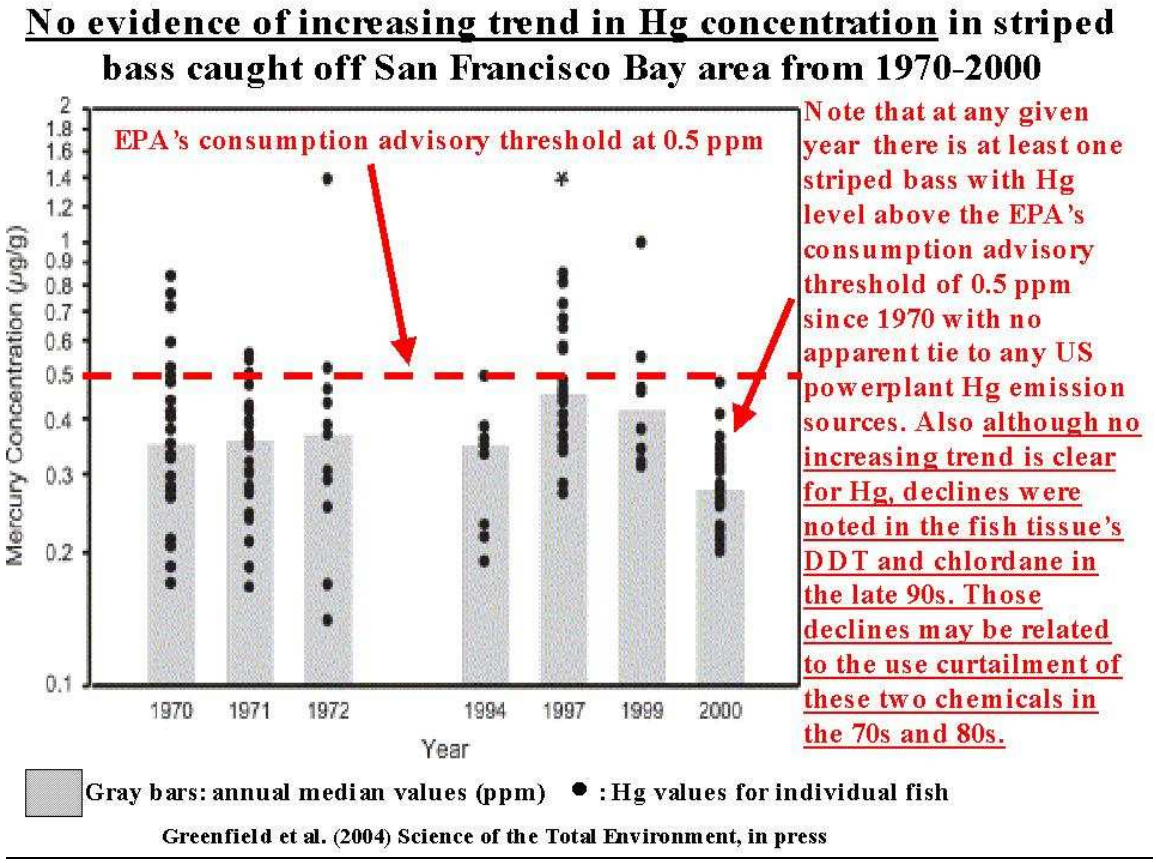


Figure E4 reveals another important finding from this new study. Even though no accumulation trend was noted for mercury in striped bass in the 1970-2000 period, significant declines in the late 1990s were noted for other contaminants like DDT and chlordane in San Francisco Bay's fish tissues. The authors suggest that the declines may be linked to known curtailed usage of the two chemicals in the 1970s and 1980s. Thus, the combined findings suggest a more complicated

and complex chain of biomethylation and bioaccumulation for mercury in fish. That is, compared to other contaminants it appears that the pathway and behavior of mercury transformation and accumulation in fish differs significantly.

A similar tendency was recently reported (Yamaguchi et al., 2003, Chemosphere, vol. 50, 265-273) for levels of contaminants in fish from upper River Thames in Britain by a group of zoologists from Oxford University and Cornell University. These authors concluded that although the recent decrease in environmental contamination level of PCBs may be partly associated with industrial and human activities, it was difficult to find such associations for mercury.

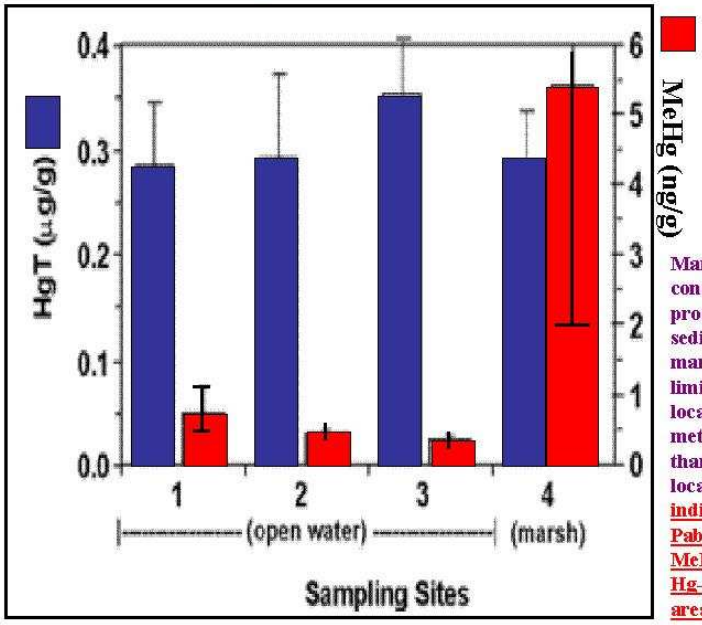
(F) "To effectively estimate fish MeHg concentrations in a given ecosystem, it is important to understand the behavior of Hg in aquatic ecosystems is a complex function of the chemistry, biology, and physical dynamics of different ecosystems." (p. 69874)

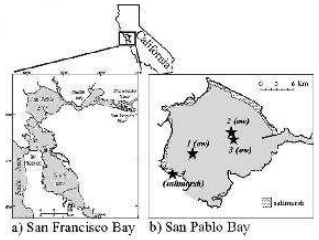
Figure F1 confirms admission by EPA that trace levels of MeHg in fish depend on the complex physical, chemical, and biological factors within each unique ecosystem. More importantly, it evidences that despite the relatively constant level of total inorganic mercury available in *all four* (3 open water and 1 salt-marshland) of the sampling sites (the four blue bars in **Figure F1**) in this study, the production and concentration levels of MeHg were significantly enhanced at the biologically active and organically rich marsh wetland site (the tallest red bar marked "marsh" in **Figure F1**). The authors concluded that "sediment geochemistry (redox, sulfide, pH, organic content, etc.) is a much more important control on MeHg production than is the absolute total mercury concentration" (p. 266 of Marvin-DiPasquale et al., 2003, Environmental Geology, vol. 43, 260-267).

The San Francisco Bay findings add to the body of evidence showing that either adding or reducing Hg atmospheric deposition from any coal-fired power plant would not measurably affect MeHg levels in the San Francisco Bay ecosystems. To the contrary, *MeHg levels are naturally self-limited* by specific ecosystem dynamics, water quality variables like dissolved sulfate, parameters like the population of algae and/or zooplankton, availability of nutrients and/or sunlight and so on.

Figure F1

Methylmercury (MeHg) production DOES NOT depend on the amount of elemental mercury (including those from power plants) available:
Mircobial MeHg production in marsh wetlands are 25-50 times more than in open-water locations around San Pablo Bay area





Marvin-DiPasquale et al. (2003) concluded that "Microbial MeHg production ... in 0-4 [cm] surface sediments was also the highest in the marsh [3.1 ng/g/day] and below detection limit [< 0.06 ng/g/day] in open-water locations. The marsh exhibited a methylation/demthylation ratio more than 25X than of all open-water locations. ... These preliminary data indicate that wetlands surrounding San Pablo Bay represent important zones of MeHg production, more so than similarly Hg-contaminated adjacent open-water areas." (p. 260)

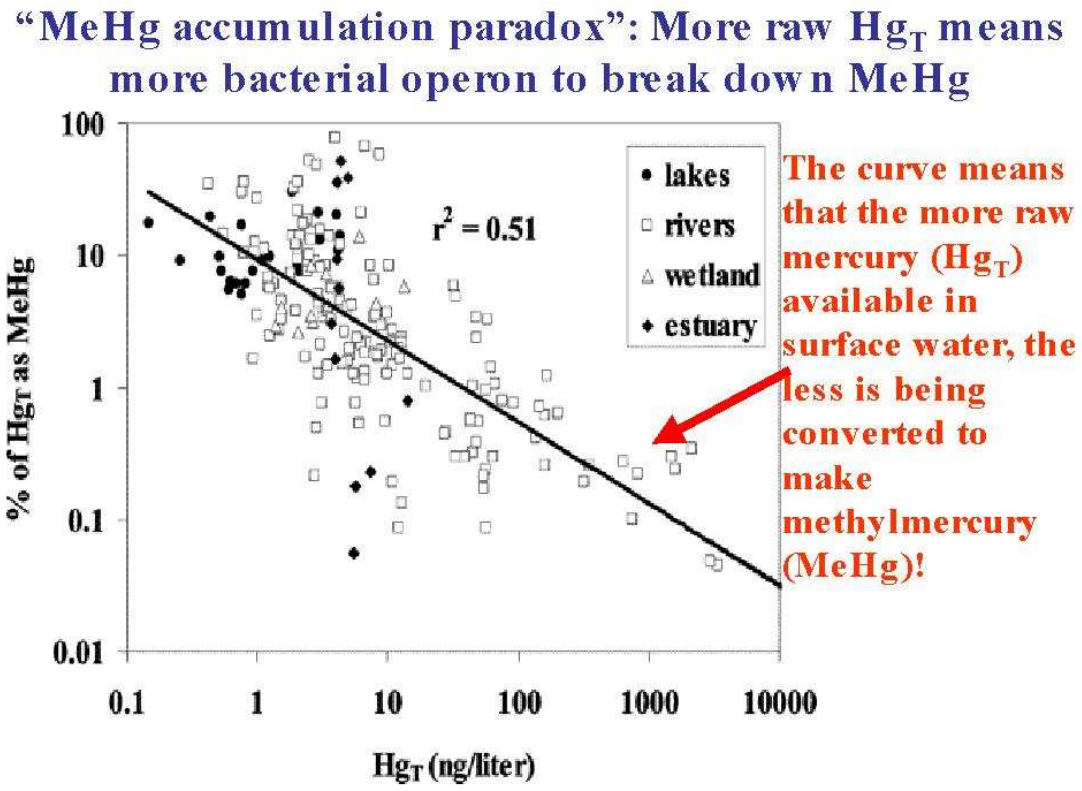
Reference: Marvin-DiPasquale et al. (2003) Environmental Geology, vol. 43, 260-267

(G) "Microbes convert a small fraction of the pool of inorganic Hg in the water and sediments of these ecosystems into the organic form of Hg (MeHg). MeHg both bioconcentrates and biomagnifies. In the environment this process is referred to as bioaccumulation. MeHg is the only form of Hg that biomagnifies in organisms." (p. 69874)

Generally agreeing with EPA's assessment, we offer an additional new and important model validation target for EPA's modeling team to factor. **Figure G1** shows a very important observation concerning the accumulation of MeHg in various watershed systems. It shows a particular "MeHg accumulation paradox" in that the relative percentage of MeHg converted from raw Hg available actually *decreases* as the amount of raw Hg available *increases*. The authors of these important findings suggest that as the amount of raw Hg increases in a watershed system, there are actually more bacterial operons (i.e., bacterial enzymes encoded by the mercury resistance (*mer*) operon) available to significantly break down the MeHg produced, and thus explaining the observed

"MeHg accumulation paradox." Again, the proposed EPA's CAMR needs to fully account for the underlying science before making costly and ineffectual compliance rulings.

Figure G1



Schaefer et al. (2004) Environmental Science & Technology, vol. 38, in press

(H) "To analyze the link between Hg deposition and MeHg concentrations in fish in aquatic ecosystems across the U.S., EPA currently is considering using EPA's Office of Water's Mercury Maps (MMaps). … The MMaps model suggests that changes in steady-state concentrations of MeHg in fish will be proportional to changes in Hg inputs from atmospheric deposition if air deposition is the only significant source of Hg to a water body; and if the physical, chemical, and biological characteristics of the ecosystem remain constant over time." (p. 69875)

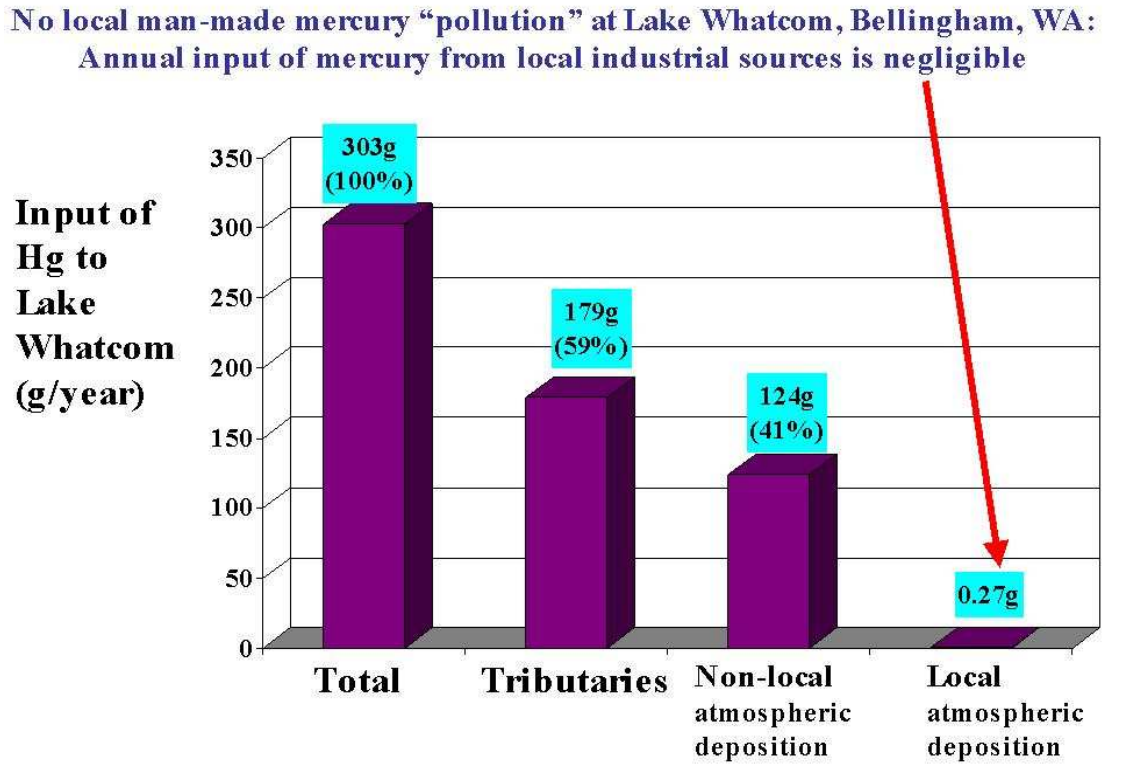
It is very important to emphasize that the best available science suggests repeatedly that the stated assumptions in EPA's MMaps model are likely to be wrong. Therefore, they cannot be meaningfully applied for a realistic assessment

of how a change in Hg emissions from power plants can possibly affect concentrations of MeHg in fish.

First, **Figure D1** above provides real data that "the physical, chemical, and biological characteristics of the ecosystem(s)" never remained constant over time. That alone invalidates the critical model assumption.

Figure H1 offers another important target for validating the assumptions in EPA's MMaps model. It shows that local atmospheric deposition of Hg has negligible contribution to the annual budget of mercury to the Lake Whatcom ecosystems, thus directly challenging EPA's MMaps assumption that "air deposition is the only significant source of Hg to a water body."

Figure H1



Paulson (2004) Sources of mercury in sediments, water, and fish of the Lakes of Whatcom County, Washington, U.S. Geological Survey Scientific Investigations Report 2004-5084 (August 2004)

The author of the new Lake Whatcom study further noted:

> "Concerns about mercury (Hg) contamination in Lake Whatcom, Washington, were raised in the late 1990s after a watershed

> protection survey reported elevated concentrations of Hg in smallmouth bass. The USGS ... cooperated to develop a study to review existing data and to collect new data that would lead to a better understanding of Hg deposition to Lake Whatcom and other lakes in Whatcom County. Of all the lakes examined, basin 1 of Lake Whatcom would have been most affected by the Hg emissions from the chlor-alkali plant and the municipal sewage-sludge incinerator in the City of Bellingham. The length-adjusted concentrations of Hg in largemouth and smallmouth bass were not related to estimated deposition rates of Hg to the lakes from local atmospheric sources. Hg concentrations in dated sediment core samples indicate that increase in Hg sedimentation were largest during the first half of the 20th century. Increases in Hg sedimentation were smaller after the chlor-alkali plant and the incinerators began operating between 1964 and 1984. Analysis of sediments recently deposited in basin 1 of Lake Whatcom, Lake Terrell, and Lake Samish indicates a decrease in Hg sedimentation. " (p. 1 of Paulson, *Sources of mercury in sediments, water, and fish of the Lakes of Whatcom County, Washington*, U.S. Geological Survey Scientific Investigations Report 2004-5084, August 2004)

(I) "Despite these limitations of this model [MMaps], EPA is unaware of any other tool for performing a national-scale assessment of the change in fish MeHg concentrations resulting from reductions in atmospheric deposition of Hg. " (p. 69875)

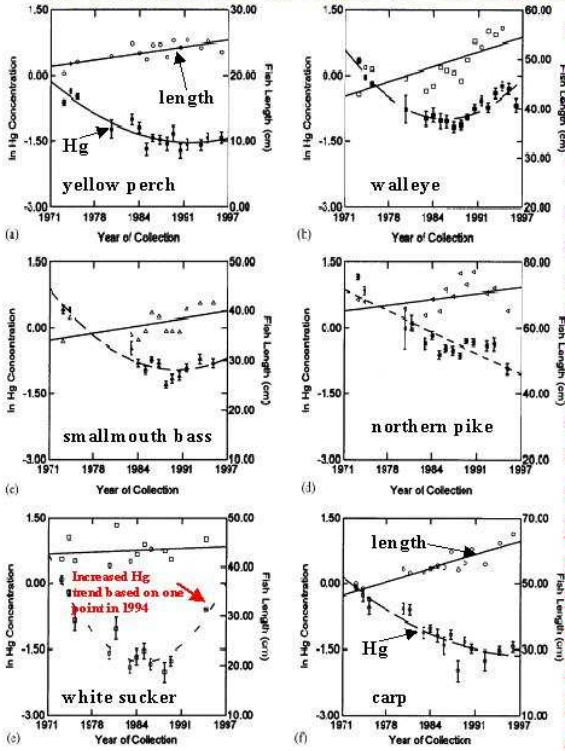
It should be fairly clear from the peer-reviewed literature exhibited in **comments (A) through (H)** that EPA's MMaps isn't simply suffering from "limitations," but is instead *terminally overwhelmed* by numerous *demonstrably flawed assumptions* in its irrational determination to claim a reduction of Hg emissions from U.S. power plants can or will lead to a reduction in accumulation of MeHg in ocean or U.S. freshwater fish.

Figure I1 presents for EPA's modelers yet additional, recently published fish mercury data sets of various sport fish species caught from 17 "areas of concern for mercury contamination" in the Canadian Great Lakes from 1971 to 1997. The results again evidence that historical changes in mercury concentrations *are not* simply to be expected from local industrial Hg emissions. In fact, the author concluded that, "Differences observed [among different areas of concern] did not consistently parallel expectations associated with historical presence of chlor-alkali plants in the vicinities of some locations." More importantly, the author

also noted that "An attempt to correlate the fish tissue mercury with the frequency of occurrence of infantile cerebral palsy at AOC [areas of concern] was unsuccessful." Further mercury-related health issues are addressed in **comments (K) through (Q)** below.

Figure I1

Hg concentration in sport fish from Canadian Great Lakes areas of concern: No link to occurrence of infantile cerebral palsy



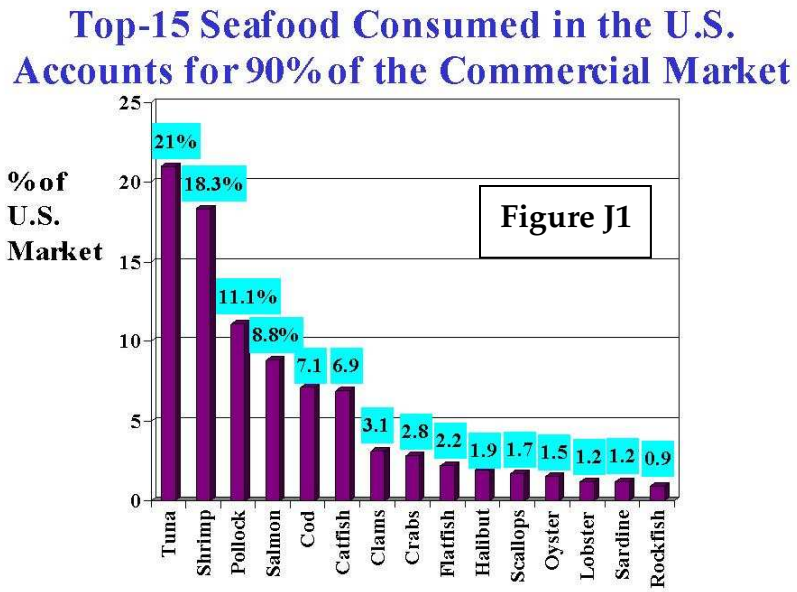
"The tissue mercury concentration in six species of fish collected at the 17 Areas of Concern [AOC] ... were analyzed. A linear increase in Hg concentration with fish length was found, but slopes differed among locations. The temporal pattern over the period 1971-1997 differed across species in fish collected in Lake St. Clair; in at least two species there was evidence of increased mercury concentration during the 1990s that had been suggested in an earlier analysis. AOC differed significantly in observed tissue concentrations. Differences observed did not consistently parallel expectations associated with historical presence of chlor-alkali plants in the vicinities of some locations. An attempt to correlate the fish tissue mercury with the frequency of occurence of infantile cerebral palsy at AOC was unsuccessful."

Weis (2004) Environmental Research, vol. 95, 341-350

(J) "MMaps is designed to simulate natural freshwater systems. We currently do not have an appropriate method for assessing how a change in the deposition of Hg relates to a change in the concentration of MeHg in fish tissue in fish found in marine environments and/or farm-raised species. We recognize, however, that marine and farm-raised species comprise a large proportion of the fish consumed by the U.S. population and, likely account for a significant fraction of the overall exposure." (p. 69875-69876)

This partial admission on the highly limited use of MMaps by EPA goes a long way toward illustrating our concerns regarding the scientifically questionable direction of EPA's modeling effort. **Figure J1** confirms that the top-15 fish and

sea foods consumed in the U.S., representing about 90% of the U.S. commercial market, is accounted by marine and farm-raised species. (According to UN statistics, domestic fresh water fish may account for as little as .05% of total U.S. consumption.[5]) This alone renders EPA's MMaps modeling results on MeHg levels in freshwater fish almost irrelevant or largely insignificant.



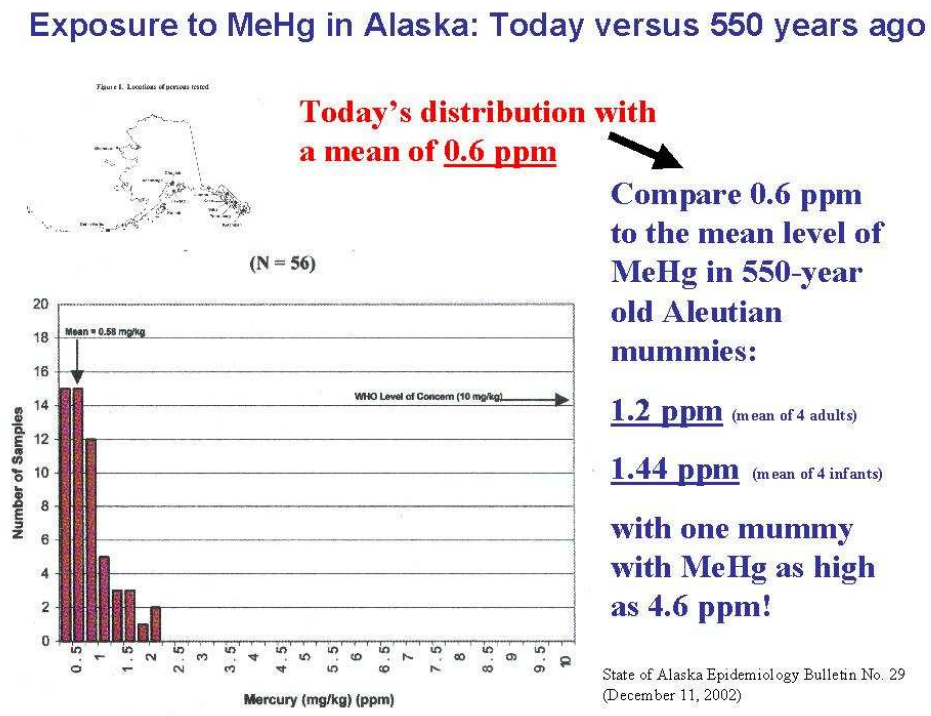
Source: Carrington and Bolger (2002) Risk Analysis, vol. 22, 689-699 + updates in Carrington and Bolger (2003)'s Intervention Analysis Draft Report

Considering the insignificant Hg emissions from U.S. coal-fired power plants (i.e., less than 1% of annual global emissions budget) and the millions of tons of natural Hg available in world oceans from deep venting, it is clear that there will **be no detectable change in trace MeHg in oceanic fish even if EPA were to impose *zero emission* standards for all U.S. Hg sources**. Evidence provided in **comment (E)** (i.e., **Figures E1, E2 and E4**) alone should be adequate for our hypothesis. (If trace levels of MeHg did not increase in a wide variety of fish along with rapidly growing worldwide anthropogenic emissions (See figure E3), what rationale is there that fish MeHg levels would *drop* in response to falling U.S. emissions?) Meanwhile, the strictest burden of proof for EPA rule making demands a clear demonstration (not invalid modeling assumptions) that its CAMR rulings can deliver a clear and meaningful reduction in MeHg in world ocean fish.[6]

(K) "We are also collecting information on fish consumption rates by different affected populations, particularly in the eastern half of the U.S. We recognize that many Americans consume seafood or freshwater fish; however, some subpopulations in the U.S. (e.g., Native Americans, Southeast Asian Americans, and lower income subsistence fishers) may rely on fish as a primary source of nutrition and/or for cultural practices." (p. 69877)

We present here an important source of information. **Figure K1** shows the distribution of hair mercury values of 56 pregnant women sampled from 12 different native communities across Alaska. This 2002 survey by the State of Alaska Epidemiology Office confirms that on average Alaskan native pregnant women (with a mean hair mercury value of 0.6 ppm) consumed more fish than other average U.S. women (who have a mean hair mercury value of about 0.2 ppm based on the ongoing CDC's NHANES database). It is also clear from **Figure K1**, based on the examination of 8 Aleutian mummies dated to about 550 years ago, that the native Alaskans had long been naturally exposed to significantly large levels of MeHg through fish and marine mammals in their traditional diets without any plausible "contamination" by power plant Hg emissions.

Figure K1



Regarding other native populations in Eastern North America, field records from Nunavik, Quebec (**Figure K2**) suggest that the prenatal exposure level of MeHg, lead and persistent organic pollutants (or POPs) in Inuit infants born between 1994 and 2001 has *declined* significantly. The authors of this new research paper concluded that "A significant reduction of lead and mercury concentrations was found, but there was no clear linear or exponential trend. The decreases observed could be explained by a decrease in food contamination, by changes in dietary habits, or, most likely by a combination of both. Although questions remain as to the exact causes of decline, it is encouraging to observe such an improvement in prenatal exposure for this highly exposed population."[7]

Figure K2

Concentrations of mercury, lead and persistent organic pollutants in umbilical cord blood of Inuit infants born in Nunavik, Quebec have been decreasing from 1994 to 2001

"A significant reduction of lead and mercury concentrations was found, but there was no clear linear or exponential trend. The decreases observed could be explained by a decrease in food contamination, by changes in dietary habits, or, most likely by a combination of both. Although questions remain as to the exact causes of decline, it is encouraging to observe such an improvement in prenatal exposure for this highly exposed population."

POPs

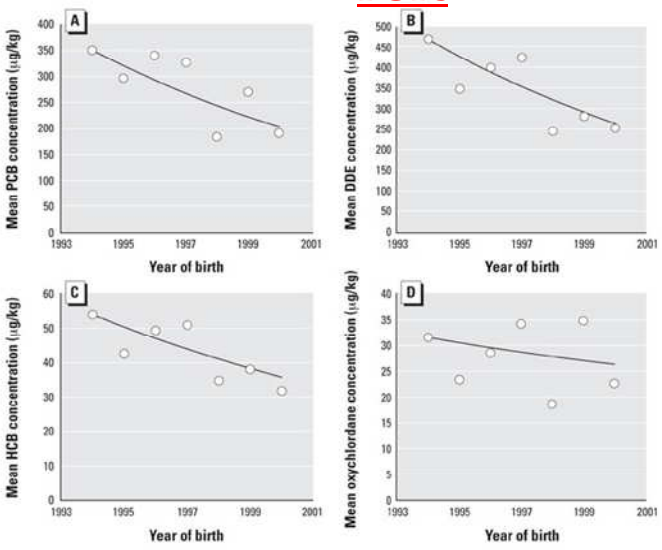
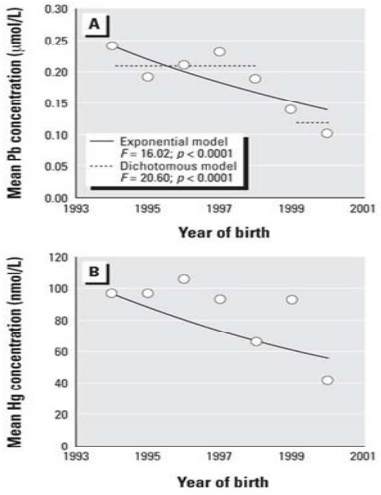


Figure 2. Adjusted mean OC concentrations according to the year of birth: (*A*) PCBs; (*B*) DDE; (*C*) HCB; (*D*) oxychlordane. The lines represent the slope estimates presented in Table 2.

Lead and Mercury



Mercury level in cord blood

← 20 µg/L

← 8 µg/L

Figure 3. Adjusted mean heavy metal concentrations according to the year of birth for (*A*) lead and (*B*) mercury. The solid lines represent the slope estimates presented in Table 2. The dotted lines represent the adjusted mean lead concentrations before and after January 1999.

Dallaire et al, 2003, Environmental Health Perspectives, vol. 111, 1660-1664

Equally important are explanations and cautions from this team of Laval University Medical Center researchers in an earlier publication (Dewailly et al., 2001, Archives of Environmental Health, vol. 56, 350-357):

> "According to recommendations formulated by the World Health Organization (WHO), no more than 5% of individuals in a population should display a methylmercury concentration that exceeds 1000 nmol/L [or converted to 200 μg/L MeHg in blood]. Concentrations of total mercury noted in present study did not exceed 560 nmol/L [112 μg/L]. … WHO issued more stringent recommendations for pregnant women, stating that not more than 5% of this subgroup should exhibit methylmercury concentration above 400 nmol/L [80 μg/L]. In our survey, no women of child-bearing age exhibited concentrations of this magnitude … Recent data from Faroe Island suggest that the neurologic status of children can be affected by low-level prenatal exposure to mercury. … There are, however, major differences between the diet of Faroese and the diet of Inuits, and care must be exerted before one concludes that Inuit children are at risk. … [I]n view of the high selenium intake [in the diets of the Inuit population from consumption of mattak (beluga whale skin) which is about 2.4 times higher than that measured in the Farose], which may counteract methylmercury-induced toxicity, local public health authorities did not recommend reducing seafood consumption. "

If EPA has serious concerns for native populations, it should focus on the fact that instead of advancing health and safety for these peoples, mercury warnings are already causing harm. John Middaugh, State Epidemiologist of Alaska, recently warned FDA:

> "Advisories based upon risk assessment without consideration of well-established public health benefits of fish consumption have great potential to harm public health if reductions in fish consumption occur."[8]

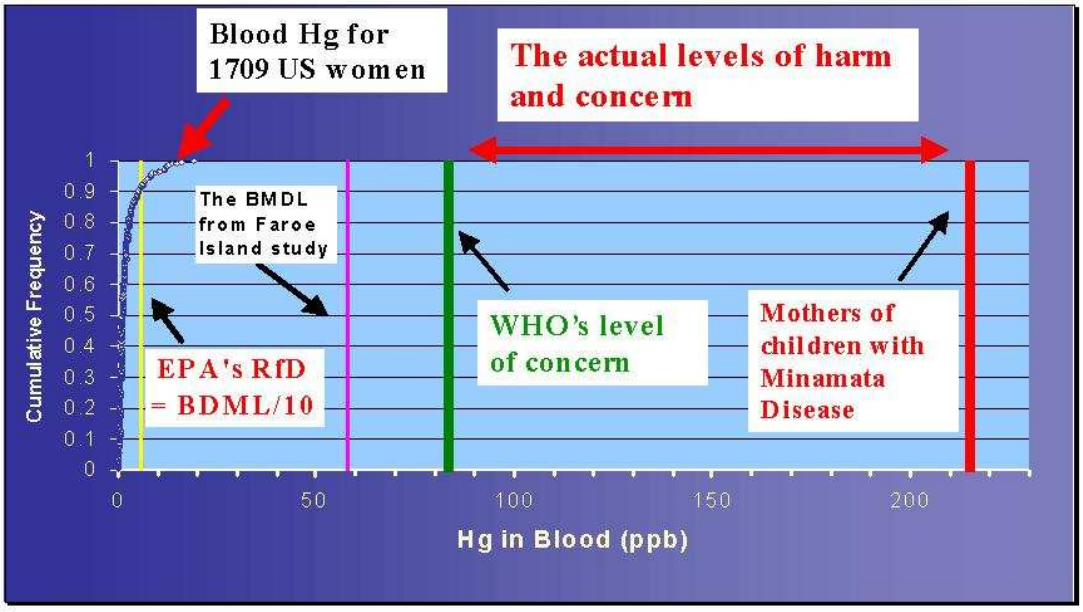
Middaugh reported that many native Alaskan communities abandoned traditional fish diets since the FDA's 2001 mercury advisory, with a subsequent increase in diabetes, heart disease, and vitamin A and D deficiencies.[9]

(L) "We intend to use the following consumption data to complete our analysis concerning the relationship between reductions in MeHg concentrations in fish tissue and reductions of human exposure to MeHg. a. *Women of childbearing age*-The National Health and Nutrition Examination Survey (NHANES) provides information based on the women who participated in the study. b. *Children*-Exposure Factors Handbook and NHANES provide information." (p. 69877)

CSPP supports EPA's intention to properly apply information from the ongoing CDC's NHANES database (which some at EPA have not done), but must stress that *no women (**Figure L1**) or children (**Figure L2**) in the current NHANES survey are actually harmed by the levels of mercury in their blood from fish consumption*.

Figure L1

No woman in the NHANES survey has blood mercury higher than EPA's chosen Benchmark Dose Lower Limit (BMDL) value and the actual level for triggering an actual health concern or harm is much higher than EPA's RfD level of 5.8 ppb



Adapted from chart shown in the July 2003 EPA/FDA stakeholder meeting

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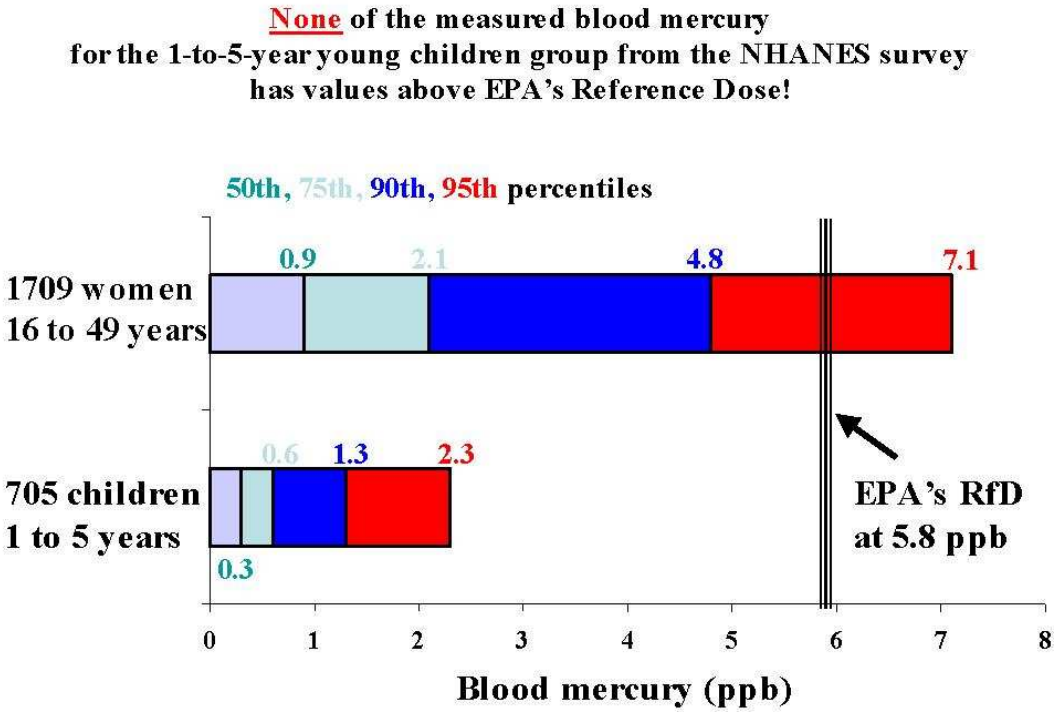
We will delay comments on the ultra-conservative nature of the MeHg RfD value set by EPA, as shown in **Figures L1** and **L2,** to **comment (O)** below. We will also confirm in comment (O) that EPA's MeHg RfD was derived from the Faroe

Islands Children Study that was plagued by contaminants like PCBs and DDT through consumption of pilot whale products and hence is widely recognized in professional circles as incompatible with or irrelevant to the U.S. consumption profile of a wide variety of fish (i.e., excluding whale products).

Figure L2



(M) "Published MeHg research suggests there may be neurological effects during fetal and child development, including intelligent quotient (IQ) decrements and more subtle effects on the ability to learn." (p. 69877)

As will be shown in **comment (O)**, EPA's premise for negative child neurodevelopment is based essentially on one endpoint from the Faroe Island study that is representative of exposure to a cocktail of toxic chemicals like PCBs, DDT and MeHg rather than MeHg alone as demanded by EPA's CAMR power plant emission controls. *CSPP suggests that EPA's claim for health "benefits" from its CAMR is hypothetical or almost impossible to demonstrate because the suggested health concerns were drawn on either flawed or irrelevant epidemiological data.*

In direct contrast to claims of health concerns from consuming fish with trace amounts of MeHg, we offer findings from recent scientific studies supporting claims of significant children health-related *benefits* derived through adequate consumption of fish or fish oil containing omega-3 polyunsaturated fatty acids.

Helland et al. (2003, Pediatrics, vol. 111, e39-e44) recently stated that:

> "Pregnant women [of Oslo, Norway] were recruited in week 18 of pregnancy to take 10 mL of cod liver oil [with about 2g of DHA+EPA] or corn oil until 3 months after delivery [in a randomized and double-blinded study]. Children who were born to mothers who had taken cod liver oil (n=48) during pregnancy and lactation scored higher [by 4 points] on the Mental Processing Composite of the K-ABC [Kaufman Assessment Battery for Children] at 4 years of age as compared with children whose mothers had taken corn oil. This study indicates that maternal supplementation with very-long-chain n-3 PUFAs during pregnancy and lactation improves the intelligence of children at 4 years of age."

Daniels et al. (2004, Epidemiology, 15, 394-402) found that:

> "Fish intake by mother during pregnancy and by the infant postnatally, was associated with higher mean [child] development scores [in a cohort of 7421 British children]. For example, the adjusted mean MacArthur [vocabulary] comprehension score for children [15 months old] whose mothers consumed fish four or more times per week was 72 ... compared with 68 among those whose mothers did not consume fish. Although total cord mercury levels increased with maternal fish intake, **our data did not suggest adverse developmental effects associated with mercury**. In a small study of subjects in [this] ALSPAC study, maternal DHA levels were associated with improved visual stereoacuity among offspring at 3.5 years of age. ... Fish intake during pregnancy has the potential to improve fetal development because it is a good source of iron and long chain omega fatty acids, which are necessary for proper development and function of the nervous system." (Emphasis added)

Finally, in another new study, Smuts et al. (2003, Obstetrics and Gynecology, vol. 101, 469-479) explains:

> "[Our] study was a randomized, double-blind, controlled, clinical trial to determine the effects of increasing docosahexaenoic [DHA] acid intake during the third trimester of pregnancy on pregnancy and birth outcomes. Subjects were supplied with [DHA-] enriched eggs (mean of 133 mg of [DHA] per egg) or ordinary eggs (mean of 33 mg of [DHA] per egg). Eighty-three percent of subjects completed the study (291 of 350 enrolled). No subject was discontinued for an adverse event. No safety concerns were raised by the study. The current study found **a 6-day longer period of gestation** when [DHA] intake was increased ... Olsen et al. suggested that higher [DHA+EPA] intake from fish by Faroe Islanders compared with Danes was the reason for longer gestation in Faroe Islanders. The authors subsequently demonstrated increases in gestation of 4 and 8.5 days, respectively, in randomized clinical trials that provided 2.7 g per day of [DHA+EPA] to a group of healthy pregnant women and to healthy pregnant women with a previous pre-term delivery. " (Emphasis added)

Claims of concern for fetal and child health by EPA and mercury activists appear disingenuous because they largely failed to emphasize to the public the *benefits* of fish consumption. This activism could unnecessarily terrorize expectant mothers into not eating a food that promotes better fetal development and child health.

Premature birth is a striking example. So serious is this outcome that the March of Dimes organization has adopted it as a primary cause.[10] More than 470,000 babies are born prematurely every year in the U.S. These infants aren't just small; they're developmentally "unfinished."[11]

The March of Dimes provides these facts on prematurely born babies:

- Has increased by 29 percent since 1981
- Accounts for 12 percent of all live births
- Can happen to any pregnant woman
- Is the leading killer of babies in their first month of life
- Is a major cause of long-term health problems, including cerebral palsy, mental retardation, blindness, chronic lung problems, Respiratory distress syndrome and bleeding in the brain
- Is the number one obstetrical problem in the country
- Robs families of the full potential of their children, society of their future leaders and our nation of strong and healthy citizens
- Places tremendous financial burdens on everyone. Hospital charges for infants with a principle diagnosis of prematurely average $75,000, and add up to billions of dollars each year.[12]

Recognizing the role of fish nutrition plays in helping prevent the tragedy of premature births, the March of Dimes is funding a Danish and Chinese research team to further clarify the issue. One of the researchers, Dr. Sjurdur Olsen of Denmark reported that Danish women who consumed fish or seafood at least once a week during the first 16 weeks of pregnancy have three times less risk of low-birth weight or premature births.[13] But a closer look at the literature will reveal ample evidence already available that women who avoid fish in their diets during pregnancy are at increased risk for delivering their babies early, which increases risk for their babies being born small, sick and dying.

(N) "However, research also raises the possibility that MeHg in fish can reduce the cardioprotective effects of fish consumption in adult males." (p. 69877)

The postulated but unconfirmed effects of methylmercury (MeHg) on cardiovascular health in the NRC (2000) report appear to have contributed to EPA's RfD for MeHg.

Alan Stern, of the New Jersey Department of Environmental Protection and member of the NRC (2000) MeHg committee, recently revealed:

> "In 2000, the National Research Council's Committee on the Toxicological Effects of Methylmercury issued a report (NRC, 2000) in which it considered the various adverse health effects associated with the exposure to methylmercury (MeHg). Among the effects considered were cardiovascular effects. The committee concluded that 'Given the limits of the available data, neurotoxicity is the most sensitive, well-documented health endpoints[14]. ... However, there is emerging evidence of potential effects on both the immune and cardiovascular systems at low doses of exposure. Although these effects are not well understood, emerging data underscore the need for continued research and raise the possibility of adverse effects ... at or below the current levels of concern for developmental neurotoxicity.' The committee recommended that an overall uncertainty factor of adjustment of 10 be applied to the neurodevelopmental point of departure to derive a MeHg reference dose (RfD). This uncertainty factor, in part, addressed the possibility that cardiovascular effects may ultimately prove to be a more sensitive endpoint than neurodevelopment effects. The US EPA, in its derivation of an RfD for methylmercury, followed the lead of the NRC committee in applying a similar rationale for its 10-fold uncertainty factor adjustment (US EPA 2004)."

CSPP suggests that EPA should be more critical in providing an independent assessment on this potentially dangerous and poorly documented claim. To that end, we offer several concise criticisms on the two main published studies (as cited by EPA's NODA) suggesting a connection between MeHg exposure from fish and cardiovascular disease (CVD), coronary health disease (CHD) and even death in adults. (A longer and more thorough review on this recent alarmism about the negative impacts of fish intake on cardiac health can be found in **"Fish, Mercury and Cardiac Health"** by CSPP.[15]) But it should be pointed out that the third study cited by the EPA's NODA, the Yoshizawa et al. (2002, New England Journal of Medicine, vol. 347, 1755-1760) paper, actually reported their *inability* to confirm an association of total mercury exposure and risk of CVD based on a 5-year follow-up of 33,737 U.S. male health professionals. The results of Yoshizawa et al. (2002) clearly did *not* raise the **"possibility that MeHg in fish can reduce the cardioprotective effects of fish consumption in adult males**" as incorrectly implied by EPA's citation.

First, some background on the two studies claiming negative cardiac health associations with fish consumption.

(1) The Finnish Study by Salonen et al. (1995, Circulation, vol. 91, 645-655; 2000, Atherosclerosis, vol. 148, 265-273) + Virtanen et al. (2002, poster presentation in the April 23-26, 2002 American Heart Association, Asia Pacific Scientific Forum at Honolulu, Hawaii): A study of 2005 men from Kuopio, eastern Finland found that men in the highest quarter (> 2.5 ppm) had a 1.6-fold risk of CVD death and 1.7-fold risk of CHD death when compared to men in the lowest three quarters *after adjusting* for numerous risk factors including age, LDL (bad) cholestrol+triglyceride, intakes of saturated animal fatty acids and etc.

(2) The European/Israeli Study by Guallar et al. (2002, New England Journal of Medicine, vol. 347, 1747-1754): A case-control study of 684 men with 724 controls reported increasing toe nail mercury level from 0.11 to 0.66 ppm (about 0.34 to 2 ppm in equivalent hair mercury levels) is associated with a doubling of the risk of myocardial infarction *after adjusting* for numerous risk factors like age, family history of heart attack, smoking status, alcohol intake, diabetes, history of hypertension, selenium intake and etc.

However, as explained in CSPP's **"Fish, Mercury and Cardiac Health"** report, numerous risk factors other than MeHg in fish will more likely explain most of the findings in Salonen et al. (1995, 2000) and Guallar et al. (2002).

Figure N1

Statistics of mortality from Coronary Heart Diease: Men of Eastern Finland are especially vulnerable

"[T]he intake of diary products, potatoes, butter, and sugar products was very high in Finland. A similar but lower intake pattern was observed in The Netherlands. Fruit, meat and pastry consumption was high in the USA. Cereals and wine consumption was high in Italy, while bread consumption was high in Yugoslavia with the exception of the Belgrade cohort. In Greece, the intake of olive oil and fruit was very high, while the Japanese cohorts were characterized by a high consumption of fish, rice, and soy products."

Table 1: Age-standardized 25-year death rates per 1000 from CHD in 16 cohorts of the Seven Countries Study. Standard error of rate in parenthesis.

Cohorts	N	CHD (death rates/1000)	Cohorts	N	CHD (death rates/1000)
US Railroad, USA	2571	160 (7)	Crete, Greece	686	25 (6)
East Finland, Finland	817	268 (15)	Corfu, Greece	529	48 (9)
West Finland, Finland	860	180 (13)	Tanushimaru, Japan	508	30 (8)
Zutphen, The Netherlands	878	169 (13)	Ushibuka, Japan	502	36 (8)
Crevalcore, Italy	993	93 (9)	Velika Krsna, Serbia	511	43 (9)
Montegiorgio, Italy	719	60 (9)	Zrenjanin, Serbia	516	116 (14)
Rome Railroad, Italy	768	87 (10)	Belgrade, Serbia	536	106 (13)
Dalmatia, Croatia	671	54 (9)	Slavonia, Croatia	696	89 (10)

Menotti et al., 1999, European Journal of Epidemiology, vol. 15, 507-515

Our criticisms on Salonen et al. (1995, 2000) include the following points:

(1) Salonen et al. (1995)'s own admission: "Theoretically, our findings **could be specific only for men in Eastern Finland**, who traditionally have a high intake of meat, fish, and saturated animal fat and a low intake of selenium and vitamin C and, most likely, other vegetable-derived antioxidants."

(2) The Kupio population **has one of the highest recorded rates of CHD and high consumption of animal fat with high measured levels of LDL (bad) cholesterol**. [See **Figure N1**]

(3) Stern (2005, Environmental Research, in press) pointed out that even in Salonen et al. (1995) as long as 9 years already elapsed between the collection of hair+urine samples and the recording of a CVD+CHD+death event. Updated report of KIHD Hg-related results in Virtanen et al. (2002) extends the elapse time to 16 years or so and hence contributing to a serious potential misclassification of causes and effects.

(4) Clarkson (2002) noted that highest recorded hair level is 15.7 ppm and more than 6 standard deviations from the mean and only a small % of the population has high hair Hg. Yet high-value points may play a major role in this type of study, "it would have been of

> interest to see if these correlations persisted when the very high mercury levels were excluded."
> (5) No clear accounting for stress—which is believed to be a major risk factor.

Our criticisms on Guallar et al. (2002) include the following points:

> (1) Contradicted by the negative results of Yoshizawa et al. (2002) 5-year follow-up study of 33.737 US male health professionals that covers a wider range of toenail mercury from 0 to 14.6 ppm (or about 45 ppm in equivalent hair mercury level)
> (2) Why is LDL cholesterol not measured and identified as a risk factor (while HDL and total cholesterol were measured)?
> (3) Serious challenges and questions from Plante and Babo (2003) in New England Journal of Medicine (vol. 348, 2151-2152): "**Patients with Minamata disease and hair mercury levels above 100 ppm did not have a higher rate of death from heart disease than controls, nor did they have a higher degree of arteriosclerosis**. In the Minamata region of Japan, a population of approximately 50,000 with an average hair mercury level of 50 ppm did not have a higher rate of death from heart disease than a reference population of 800,000 with an average level of 9 ppm. ... **Cree Indians with an average hair mercury concentration of 10 ppm have a lower risk of death from circulatory disease than the rest of the population in Quebec**, in which the average hair mercury level is 0.5 ppm. If, as Guallar et al. suggest, mercury increases the risk of myocardial infarction by more than 100 percent when the hair mercury level reaches 2 ppm, **how can one explain the absence of effects at doses greater than 100 ppm**?" (Emphasis added)

Concerning "sudden death" the clinical evidence continues that fish nutrition can lower the risk:

> "The n-3 fatty acids found in fish are strongly associated with a reduced risk of sudden death among men without evidence of prior cardiovascular disease. As compared with men with levels of long-chain n-3 fatty acids in the lowest quartile, those with levels in the highest quartile had an 81 percent lower risk of sudden death." (Albert et al., 2002)[16]

> "[W]e have summarized the growing clinical evidence that these n-3 fatty acids are antiarrhythmic and can prevent sudden cardiac death in humans. These n-3 fatty acids have been part of the human diet for some 2 to 4 million years ... They are safe and have been listed on the GRAS ('generally regarded as safe") list according to the Food and Drug Administration in amounts up to 3.5 g of fish oil per day." (Leaf et al. 2003)[17]

> "Alexander Leaf and colleagues ... suggest a hypothesized cellular mechanism through which n-3 PUFAs affect ion channels to reduce the risk of arrhythmia. The messages ... are clear. For clinicians, it is time to implement the current American Heart Association dietary guidelines that recommend the dietary intake of 1 to 2 fish meals, particularly fatty fish, each week. For policymakers, there is a need to consider new indication for treatment with low-dose n-3 PUFAs supplements ..." (Siscovick et al., 2003)[18]

Speaking on cardiac risk concerns, Professor Tom Clarkson, Distinguished Professor of Environmental Medicine at the University of Rochester has commented that:

> "Eating lots of ocean fish isn't much of a hazard compared to missing out on the benefits from not eating fish. **A slew of scientific reports have shown that eating fish helps protect against cardiovascular disease and enhances brain development before and after birth**. Fish is a rich source of low-fat protein and is full of fatty acids known to lower cholesterol. **Overstating the almost negligible risk of mercury could adversely affect millions of people who face the risk of heart disease**." (Emphasis added)

Dr. Eric Rimm, Professor of Epidemiology and Nutrition at Harvard School of Public Health agreed:

"The message of fish being good has been lost and people are learning more about the hypothetical scare of a contaminant than they are of the well-documented benefits of coronary disease reduction. The danger of the tuna fish is not well documented compared to the potential dangers for a 50-year-old male or female who are at a much higher risk of coronary health."[19]

(O) "EPA subsequently established a reference does (RfD) of 0.0001 milligrams per kilogram of body weight per day (mg/kg/day) derived from a neurodevelopmental endpoint based on the NAS review." (p. 69877)

Figure O1 shows the rarely seen "evidence" that both EPA and the "NAS review" in 2000 (see more criticisms of the "NAS" [actually NRC (2000)] review under **comment P** below) had adopted to support their claims of negative neurodevelopmental impacts from prenatal exposure to MeHg through maternal consumption. The result was drawn from the Faroe Island children study originally published by Grandjean et al. (1997, Neurotoxicology and Teratology, vol. 19, 417-428) and the particular endpoint test (see additional criticisms by CSPP in **comment Q** below) is the so-called cued Boston Naming Test (note that this is not to be equated to "IQ" as represented in the final section of EPA's NODA).

Figure O1

Evidence for neuropsychological problems in the Faroe islands children study is not strong

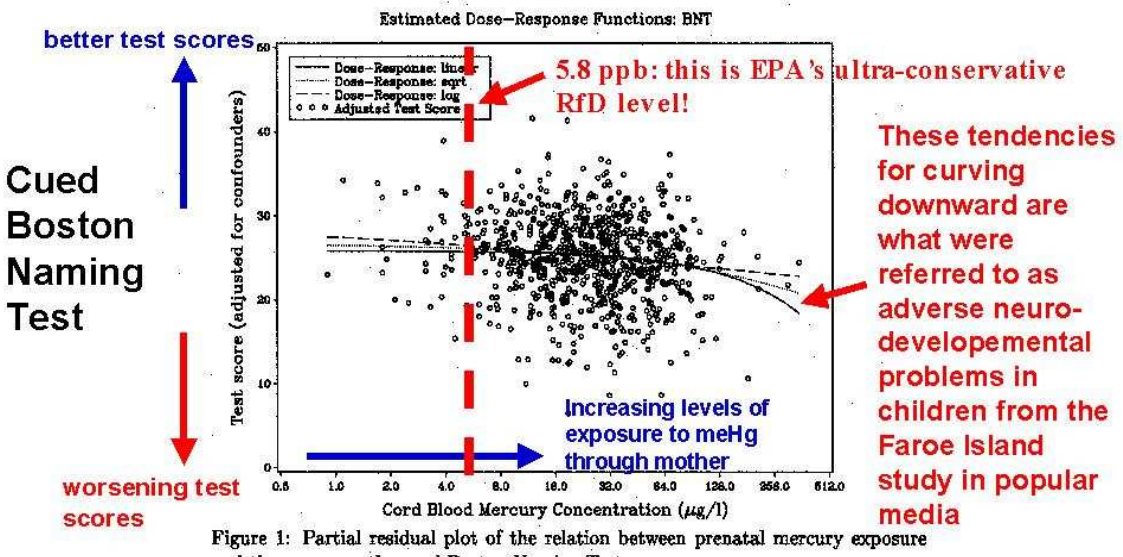


Figure 1: Partial residual plot of the relation between prenatal mercury exposure and the scores on the cued Boston Naming Test.

Budtz-Jorgensen et al. (2003) *Environmetrics*, vol. 14, 105-120

Figure O1 clearly suggests a significant scatter in the test scores as MeHg exposure level changes. It is also worth reminding that this particular endpoint is indeed the *best* evidence allowing the EPA and "NAS" suggestions of negative

impacts with increasing MeHg exposure—as guided by various statistical fitting lines in **Figure O1** despite the large scatter. More important to note in **Figure O1** is the relative position of the EPA adopted level of MeHg RfD in the equivalent blood mercury levels of 5.8 ppb (marked as red dashed vertical line in **Figure O1**). The superposition of the EPA's adopted MeHg RfD shows a clear *disconnect* to the underlying data which forms the original claim for negative impacts linked to MeHg exposures. *This result makes clear the distinction between the actual levels of harm or concern for MeHg and the hypothetical and ultra-precautionary level of safety set by EPA's RfD shown in* ***Figure L1*** *above*.

There are even more serious issues in the underlying epidemiological data from the Faroe Island children study which exposes its selection for an RfD level as *highly inappropriate*. Not the least of which is the **refusal of the Faroe Islands researchers to release their raw data** to allow independent statistical analyses violates EPA's own data quality guidelines.[20]

The MeHg exposure profile for the Faroe Island study is *neither compatible nor directly applicable to U.S. fish consumers*. By admissions of the original Faroe Island study researchers (mainly Dr. Philippe Grandjean and Dr. Pal Weihe) and several published scientific evaluations of the Faroe Island study, the Faroe Island results should best be considered as a study assessing exposures to a mixture of chemicals like PCBs, DDT and MeHg rather than MeHg alone. It has long been noted and admitted that the Faroe Island study cohorts were contaminated by maternal exposure to high levels of DDT and PCBs via consumption of pilot whale meat and fat. The PCBs levels were evaluated to be about 600 times the so-called Aroclor 1254 RfD level established by EPA's own Integrated Risk Information System (Dourson et al., 2001, Neurotoxicology, vol. 22, 677-689).

In addition, it should not go unnoticed that in a letter to the EPA, Drs. Kenneth Poirer and Michael Dourson, both as former EPA's RfD/Reference Concentration Work Group co-chairs, had previously provided their scientific findings to the Technical Information Staff at EPA, advising that: "The Faeroe Island studies are not the proper choice for the critical study for a methylmercury RfD."[21] EPA continues to ignore this and more recent scientific evaluations.

Finally, in a February 9, 2004 open letter there is the crucial clarification by Faroe Island Children Study's Chief Physician, Dr. Pal Weihe, that the Faroese children are exposed to mercury by consumption of pilot whale meat only, *not fish*. In contrast, says Dr. Weihe, the fish consumption most likely is beneficial to their health. Dr. Weihe's letter follows:

To whom it may concern:

Faroe Islands women do not eat mercury-tainted fish and fish consumption does not harm Faroese children.

In the Boston Herald, Friday, February 6, 2004, p. 20 the following was stated about a mercury study in the Faroe Islands conducted in the cooperation with the Harvard University: "A fish industry spokesman said that the Harvard study was flawed because Faroe Islands women typically eat far more mercury-tainted fish than do Americans"

As the researcher in charge of the mercury studies on children in the Faroe Islands since 1985 I want to correct this statement.
The Faroese children are not exposed to methylmercury by eating fish. They are exposed to mercury by the traditional consumption of pilot whale meat. Fish normally consumed in the Faroes, e.g. Cod and haddock, are low in mercury and **do not, to my opinion constitute any threat to the health of the Faroese children.** In the contrary the fish consumption **most likely is beneficial** to their health.

The Faroese authorities in 1998 recommended women who plan to become pregnant within months, pregnant women, and nursing women to abstain from eating **pilot whale meat.** The mercury concentration in the blood of pregnant women has **declined dramatically since and are now below the US-EPA limit.**

Yours sincerely,
Pal Weihe, Chief Physician [Emphasis added]

How can EPA or the National Research Council seriously cling to the Faroe study for its RfD for fish consumption when the lead author states the study has nothing to do with MeHg in fish, but only in whale meat? How can Dr. Grandjean claim associations between IQ levels in Faroese children and fish consumption (see further discussion under **comment Q** below) when Dr. Weihe reports that those children (a) are not exposed to MeHg by eating fish, (b) are exposed to no health threat from fish, and (c) actually benefit from maternal fish consumption?

CSPP urges EPA to discontinue relying on the very weak (and inappropriate) scientific foundation to base its claim of negative impacts on children neurodevelopments using the results of the inferior Faroe children study.

CSPP further documents here additional evidence of the extremism or ultra-precautionary nature of the current EPA RfD for MeHg.

First, it is clear from the ethical guidelines established by the Institutional Review Board of the National Center for Health Statistics of the CDC that approved the NHANES study that cautions are issued to NHANES participants *only if* their total hair mercury levels are above 15 ppm or total blood mercury above 200 ppb (McDowell et al., 2004, Environmental Health Perspectives, in press, available online May 27, 2004). The ultra-precaution by EPA is connected to its RfD which considers a blood mercury level to be safe only at levels below 5.8 ppb, which is dramatically lower than the ethical guideline established by the Institutional Review Board of CDC.

Second, it is obvious from the latest results of the Japanese hair mercury measurements for 8665 individuals collected in 10 different locations over 1999 to 2002 by Yasutake et al. (2004, Journal of Health Science, vol. 50 (2), 120-125) that **the overwhelming majority of the Japanese population, i.e., 87%, has hair mercury levels *exceeding* the mercury "safety" level set by EPA's RfD**. Since there is no detectable epidemics of any defective mental capability of both the Japanese adult and children populations, we suggest that such reality confirms the ultra-precautionary nature of the current EPA RfD level for MeHg, and conclude that the actual levels of concern from MeHg exposure occurs at much higher levels (see for example, various values identified in **Figure L1**) than an the RfD value of 5.8 ppb in blood mercury adopted by EPA.

(P) "The RfD was based on three epidemiological studies of prenatal MeHg exposure in the Faroe Islands, New Zealand, and Seychelles Islands." (p. 69877)

This particular statement by EPA is both confusing and misleading. The statement all by itself appears contradictory to what was stated in **comment (O)** above that EPA's RfD was derived from a single neurodevelopmental endpoint.

In addition, any reference to the NRC (2000) report, *Toxicological Effects of Methylmercury*, about the review and assessment of these three epidemiological studies would be more complete in noting that:

> "The committee concludes that there do not appear to be any serious flaws in the design and conduct of the Seychelles, Faroe Islands, and New Zealand studies that would preclude their use in a risk assessment. However, because there is a large body of

> scientific evidence showing adverse neurodevelopmental effects [unfortunately, **the NRC did not provide any precise citation for such evidence**] ... the committee concludes that an RfD should not be derived from a study, such as the Seychelles study, that **did not** observe an association with MeHg." (p. 6) (Emphasis added)

Therefore, we conclude that the high-quality results from the Seychelles study (with additional assessment noted below) were ignored by NRC (2000) not for any scientifically defensible reasons, but because of a direct bias to recommend only results that show evidence for "adverse neurodevelopmental effects." This situation is truly unfortunate because it is relatively well-known that the results of the Faroe Island study had been contaminated by simultaneous exposures to other toxic chemicals like PCBs and DDT (see **comment O** above).

A post NRC (2000) analysis by Dourson et al. (2001, Neurotoxicology, vol. 22, 677-689) recommended that "The Faroe Islands data are from exposures to a mixture of chemicals. The Seychelles Island data are from exposures to primarily one chemical, methyl Hg.... We would ... encourage EPA to use the Seychelles Island data as the basis of its methyl Hg RfD."

This is why **it is scientifically appropriate to challenge** the biased conclusions of NRC (2000) and hence EPA's basis for its MeHg RfD. The Seychelles Island results are clearly superior for deriving RfD exposure to methylmercury for the U.S. population. This is so simply because that study is without toxic confounders and the Seychelles Island mothers consumed ocean fish containing MeHg concentrations comparable to those consumed by the general U.S. population.

In contrast to the Faroe Island study, the Seychelles Child Development Study (SCDS), "was specifically designed to test the validity of [the] hypothesis [of adverse neurodevelopmental effects] in a well-nourished population exposed to MeHg only from high consumption of unpolluted ocean fish." The research authors recently concluded:

> "[SCDS] longitudinal assessment at 9 years of age indicates no detectable adverse effects in a population consuming large quantities of a wide variety of ocean fish. These results are consistent with our earlier findings in the same children examined at 6, 19, 29 and 66 months of age. In Seychelles, fetal exposure was continuous through frequent consumption of ocean fish containing concentrations of MeHg comparable to those consumed by the general population in the USA. We recorded effects from covariates known to affect child development, but did not find an association

> with prenatal mercury" (p. 1692 of Myers et al., 2003, The Lancet, vol. 361, 1686-1692).

Constantine Lyketsos of the John Hopkins Hospital (2003, *Lancet*, vol. 361, p. 1668) in offering a professional overview on the implications of the Seychelles study concluded:

> "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 behavioural effects in later childhood. This conclusion is especially true against the background of the several other variables that affect cognitive-behavioural development. The positive findings from the Faeroe Islands and New Zealand studies may be related to the fact that pilot-whale blubber and shark muscle contain 5-7 times the concentrations of methyl mercury than the fish consumed in the Seychelles. While higher concentrations in seafood do not necessarily lead to higher levels in maternal hair, consumption of much larger boluses by the mother could lead to greater difficulty on the part of the developing fetus to detoxify the mercury by natural mechanisms, as Meyers and colleagues propose. Whatever the answer, **the discrepant findings from the various studies need explaining**. Whilst there is always an issue of power to detect an effect in a study reporting null findings, this is not likely to be the case in the Seychelles study with the sample size involved. If there is subtle association that could only have been detected in a much larger sample or through the use of more sensitive tests, it can reasonably be argued that the effect would be small enough to be essentially meaningless from the practical point of view. For now, there is no reason for pregnant women to reduce fish consumption below current levels, which are probably safe." (Emphasis added)

(Q) "These studies examined neurodevelopmental outcomes through the administration of numerous tests of cognitive functioning. These tests provided partial or full assessments of IQ, problem solving, social and adaptive behavior, language functions, motor skills, attention, memory, and other functions." (p. 69877-69878)

It must first be noted that the primary author the Faroe Island study, Dr. Philippe Grandjean, was found to have admitted in the May 20, 2002 Mercury Forum held

at Mobile, Alabama (http://www.masgc.org/mercury/) that "In conclusion, we have obtained evidence of subtle adverse effects on neurobehavioral functions, blood pressure, and growth. At age 7 years, a doubling of the mercury exposure corresponds to a developmental delay of up to 2 months. **Although IQ tests were not done, such delays would be comparable to a loss of about 1.5 IQ points**." (Emphasis added)

Another relevant notice is the statement in Dr. Grandjean's written testimony at the Mercury MACT Rule Hearing at Maine State House on March 1, 2004 that "Even though the children that we examined were all **basically normal**, we have documented detectable deficits that appear to be permanent." CSPP takes the statement to suggest that Faroese children are essentially all normal with normal functioning capability despite the permanent "detectable deficits" of the sort described by Grandjean. [Emphasis added]

Finally, it was also clear that the cued Boston Naming Test conducted by the Faroe study (i.e., shown in **Figure O1** above) does not constitute a proper IQ test.

In short, Dr. Grandjean's statements are contradictory at best. They would not hold up in a rigorous scientific evaluation by other experts or his peers. They should not go unchallenged by EPA either, especially as his raw data are not transparent.

But the really relevant question to assessing the statement in (Q) is whether if any or all of these reputed neurodevelopmental outcomes and tests can be shown to be related to MeHg exposure. In that regard, it is important to consider that for a total 17 neuropsychological tests conducted by the Faroe study to search for associations with prenatal exposure to MeHg, only 3 tests (both the cued and uncued Boston Naming Tests and the so-called Neurobehavioral Evaluation System2 Continuous Performance Test[22]) yielded statistically significant correlations *only if* the Faroe researcher considers maternal cord blood as an independent predictor (Budd-Jorgensen et al., 2003, Environmetrics, vol. 14, 105-120). The statistical correlation for the same 3 test scores dramatically turned insignificant or only marginally significant when the measured maternal hair mercury[23] is adopted as the independent variable instead.

Dr. Gary Myers, one of the main authors of the Seychelles Island Child Development Study, makes the point that even the 3 statistical associations found by the Faroe Island study are a lot less impressive than one is lead to think if one properly weighs in the statistical odds. In a July 29, 2003's testimony to the Senate Environment and Public Work Committee, Myers[24] noted:

> "Through 107 months (9 years) and over 57 primary endpoints, the [Seychelles Island] study has found only three statistical associations with prenatal MeHg exposure. One of these associations was adverse, one was beneficial and one was indeterminate. These results might be expected to occur by chance and do not support the hypothesis that adverse developmental effects result from prenatal MeHg exposure in the range commonly achieved by consuming large amounts of fish. The test results do show associations with factors known to affect child development such as maternal IQ and home environment so there is evidence that the tests are functioning well [i.e., the Seychelles Island Child Development Study shows evidence for a high degree of internal consistency]."

Myer concluded in his senate testimony:

> **"We do not believe that there is presently good scientific evidence that moderate fish consumption is harmful to the fetus. However, fish is an important source of protein in many countries and large numbers of mothers around the world rely on fish for proper nutrition. Good maternal nutrition is essential to the baby's health. Additionally, there is increasing evidence that the nutrients in fish are important for brain development and perhaps for cardiac and brain function in older individuals."** [Emphasis added]

In the context of positive or negative brain development from trace-mercury fish consumption, **real world data trumps modeling or alarmist assertions**. For example, in the latest data from the Trends in International Mathematics and Science Study, students (grade 4 and 8) in Asia continued to excel. Singapore, Hong Kong, Japan and Korea were among the top performers in over 50 countries participating.[25] The United States placed well below these countries. As EPA noted, Asians are among the largest fish consuming peoples in the world. If alarming neuropsychological and neurodevelopmental deficits from prenatal MeHg exposure through fish consumption (as interpreted by Dr. Grandjean in the Faroe children study) are correct, then these Asian students are the very populations that should be evidencing an epidemic in low IQ, instead of topping the curve on international standardized math and science tests.

Conclusions

MeHg has been in fish and in the diets of fish consumers (both animal and human) since either evolved upon the earth. The source of this natural and historical condition lies in the sciences of earth's geology and marine ecodynamics, not the fossil-fueled engines of the world's economies.

Champions of regulation-at-any-cost seem bereft of the concept that over-regulation or bad regulation can be terribly harmful. One can easily envision a mercury regulatory path that would lead toward harm to public health, and diminishment of public life and the general well-being of all Americans.

For instance, if EPA, against all science and present technology regulated mercury to zero, and still found no change in fish mercury levels, then what would it do? In the process, it likely would have already harmed vast industries related to sea-food and fossil-fuel power generation and collapsed or driven off-shore a host of other dependent industries and small businesses. This, in turn, would have a highly negative impact on pension and retirement income for retired persons and on home heating and cooling costs, particularly for minorities, the poor and elderly on fixed incomes. Thousands of children, deprived of essential prenatal fish nutrition, would have suffered pre-term delivery and a host of attendant complications, including *real* neurodevelopment retardation, vision and behavioral impairment. As these outcomes occur, who will be held accountable?

The more rational, safe and responsible approach would be for EPA to embrace the advances in scientific understanding since the NRC report (2000) was issued. The U.S. government needs to revisit its radically conservative and scientifically unsupportable RfD, retract improper fish advisories (Alaska and Wyoming refuse to issue them) and stem its complicity in frightening pregnant women away from a safe, traditional source of prenatal nutrition.

Mercury clears the human system in about 45 to 70 days. Guided by a realistic RfD, along with more sensible guidelines from FDA and the American College of Obstetricians and Gynecologists, women who become, or desire to become, pregnant can make more informed decisions on fish consumption. They can balance out their own levels of concerns and benefits. It is that simple to address an alarmism with no basis in thousands of years of human experience. No costly, impotent regulation is required.

ENDNOTES

[1] Please visit http://www.scienceandpolicy.org/ for mercury-related reports: (1) Fish, mercury and cardiac health, (2) How safe are we from the fish we eat?, (3) Analysis of the Sierra Club's alarmist claims about the health impacts of mercury, (4) Misuse of rainwater mercury data in National Wildlife Federation's Cycle of Harm report and (5) EPA's mercury MACT rulemaking not justified by science.

[2] Additional scientific references are available upon request to Center for Science and Public Policy at bferguson@ff.org and 202-454-5249.

[3] http://teacher.scholastic.com/researchtools/articlearchives/volcanoes

[4] http://newsdesk.inel.gov/press_releases/2003/10-21mercury_testing.htm

http://www.billingsgazette.com/index.php?display=rednews/2003/10/22/build/wyoming/25-mercury.inc

[5] 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).

[6] Even if such reductions would be effectively meaningless to public health (See sections L-Q).

[7] Again, EPA needs to explain how, under its modeling assumptions, levels of anthropogenic Hg emissions can be dramatically *increasing* world-wide at the same time Hg concentrations can be *declining* significantly in sensitive populations.

[8] U.S. Department of Health and Human Services, Food and Drug Administration, Center for Food Safety and Nutrition, Food Advisory Committee. Methylmercury Meetings July 23-4, 2002.

[9] Ibid.

[10] http://www.marchofdimes.com/prematurity/5413_11560.asp

[11] Ibid.

[12] Ibid.

[13] British Medical Journal, February 23, 2002. Volume 324, pp.447-450.

[14] See comments O, P and Q herein for a discussion of this claim

[15] In press, draft available upon request

[16] **Albert et al. (2002, N. Eng. J. Med., vol. 346, 1113-1118**

[17] **Leaf et al. (2003, Circulation, vol. 107, 2646-2652)**

[18] **Siscovick et al. (2003, Circulation, vol. 107, 2632-2634)**

[19] April 10, 2004, NY Times article by Jennifer Lee

[20] Comments of the Utility Regulatory Group to EPA (69 Fed. Reg. 12398 [March 16, 2004]), Docket ID No. OAR-2002-0056.

[21] 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.

[22] For clarification, we quote the description for this test listed on p. 419 of Grandjean et al. (1997, Neurotoxicology and Teratology, vol. 19, 417-428) "This test was modified so that the children saw a series of animal silhouettes flashed on the computer screen. The child was required to press a button every time a cat appeared over a 4-min interval. Scores were the total number of missed responses and the average reaction time during the last 3 min. This test measures vigilance/attention."

[23] Please consult comment #3 in CSPP's report "Analysis of the Sierra Club's alarmist claims about the health impacts of mercury" for a detailed discussion on MeHg exposure estimates using both the maternal cord blood mercury and maternal hair mercury.
http://ff.org/centers/csspp/pdf/sierra-03-25-04.pdf

[24] http://epw.senate.gov/108th/Myers_072903.htm

[25] http://timss.bc.edu/timss2003i/intl_reports.html