A Scientific Critique of the

Environmental Protection Agency's

"National Emission Standards for Hazardous Air Pollutants [NESHAP] from Coal- and Oil-fired Electric Utility Steam Generating Units and Standards of Performance for Fossil-Fuel-Fired Electric Utility, Industrial-Commercial-Institutional, and Small Industrial-Commercial-Institutional Steam Generating Units"

Proposed Rule (March 16, 2011)[1]

Focusing on the Mercury Emission Issues

By

Willie Soon, PhD

June 2011

Summary of Findings

The Environmental Protection Agency's (EPA's) newly proposed National Emission Standards for Hazardous Air Pollutants (NESHAP) failed to describe the scientific reality of natural processes and multi-factorial controls that govern the cycling of mercury (Hg) and the ultimate biomethylation and bioaccumulation processes for methylmercury (MeHg). As this report documents, this natural cycle has been taking place for at least the last 650,000 years.

According to a new United Nations Environment Programme (UNEP) report on mercury,[2] U.S. mercury emissions from all sources are indeed far lower than those of China and India. Indeed, an earlier EPA press release and webpage acknowledge that US emissions are only "roughly three percent of the global total"[3] and that from "1990 through 2005, [U.S.] emissions of mercury into the air decreased by 58 percent."[4]

The ultimate question that EPA and the public should therefore ask then is this:

What will we get by cutting our already very small U.S. mercury emissions from power plants and other man-made sources – especially since new estimates from peer-reviewed papers suggest that mercury emissions from U.S. forest fires alone release about 44 tons of mercury per year,[5] an amount roughly equivalent to the annual emissions from all power plants in the United States today?

The bottom-line remains that trace amounts of mercury (Hg) or the biochemically-active form of methylmercury (MeHg) in fish, either from lakes and streams or oceans, are essentially a natural manifestation that has no clearly controllable relationship vis à vis any anthropogenic emissions of mercury. More importantly, consuming reasonable amounts of fish, at reasonable frequency, is safe and should be a crucial component of a healthy dietary plan for every Americans.

The proposed rules will have little, if any, impacts on mercury concentrations in the environment at a very high monetary and societal cost.

EPA's proposed NESHAP provides no detectable beneficial outcomes in the control of mercury emissions (even accepting EPA's own risk-benefit analysis without a challenge). The new rules will result in a major economic impact, harm American public health by creating exaggerated and unfounded fears about eating fish that are beneficial in everyone's diet, and further degrade the essential role of science in informing public policy.

WS (Willie Soon's report) has carefully examined the latest EPA's proposed new emission rules focusing on mercury. It found the following specific problems and deficiencies:

(a) The EPA proposal is an extreme form of political advocacy, seeking to limit the already low levels of mercury emissions (relative to other anthropogenic and natural sources of mercury) from U.S. electric power plants. Because the agency neglects most other active mercury emission sources, the emission cuts EPA proposes for mercury in Electric Generating Units (EGUs) will be "all pain and no gain" for Americans' public health. This fact can be demonstrated even by using the highly exaggerated risk-benefit analyses reported in this EPA proposal.

(b) The EPA proposal failed to highlight that its risk-benefit calculation, claiming an overwhelming benefit of $5 to $13 in benefits per dollar spent on emission controls, was dominantly derived from the "co-benefits" of $PM_{2.5}$ control.[6] In dollar terms quantified by EPA, the emission control cost is said to be about $10.9 billion per year, while the Hg-emission-cut-related benefit was determined to be no more than $6 million; this is a far cry from the "slam dunk" declaration from EPA, environmental and public health alarmists, and the press that mercury reduction benefits will be significant.

(c) The EPA proposal neglects key scientific knowledge and many peer-reviewed papers that suggest there is no straightforward connection between mercury (Hg) emissions from power plants, or other man-made sources, to the mercury level in fish. There is little doubt that levels of the biologically active form of mercury, methylmercury (MeHg), that are ultimately accumulating in fish tissue depend primarily upon environmental factors, such as sunlight and organic matter, pH, water temperature, and amounts of sulfate, bacteria, and zooplankton present in the ecosystem. MeHg levels in fish do not depend simply on the amount of elemental Hg available for conversion. This is why a distinguished group of mercury science experts[7] concluded that a simple change in bacterial activity alone could "**cause an *increase* in fish mercury concentrations, even as atmospheric deposition [from industrial mercury emission sources] *decreases***." [Emphasis added]

(d) The EPA proposal fails specifically to recognize the fact that existing accurate measurements of atmospheric mercury concentrations worldwide (first published in 2003[8], with an updated publication in 2011[9]) have long ***shown the systematic decreases of atmospheric mercury in the periods 1990-1996 and then 1995-2009 are completely inconsistent with the expected increase in atmospheric mercury concentrations adopting the current inventories of anthropogenic mercury emissions.*** Crucially, this has led the EPA, in the proposed NESHAP, to ignore the important role played by mercury emissions and recycling related to natural sources.

(e) The EPA proposal continues to incorrectly promote the flawed Faroe Islands' children study as the EPA's standard of proof for the harm that exposure to MeHg causes to children and women. It ignores the far different conclusions reached by superior epidemiological results from the Seychelles Island Children Development Study (SCDS). The SCDS study did not confirm any harmful effect on children due to MeHg exposure from eating a variety of ocean-caught fish at levels that are more representative for American public health.[10] In sharp contrast, the Faroe Island study population is well known to be exposed to not only MeHg but also other contaminants like polychlorinated biphenyls (PCBs) and lead. More importantly, the Faroe Island population got its MeHg dosage through consumption of highly contaminated pilot whale meats and blubbers, as admitted by Dr. Pal Weihe, the Chief Physician of the Department of Occupational and Public Health of the Faroese Hospital System. Despite EPA's claims, the agency's proposed NESHAP rules actually document the scientific bias of the NRC's 2000 report, ***Toxicological Effects of Methylmercury***, in that EPA specifically did not consider the results from the Seychelles Children Study, which fail to confirm any adverse effect of MeHg exposure through consumption of ocean fish. The result is that EPA's Reference Dose (RfD) for MeHg exposure can be shown to be excessively exaggerated by at least a factor of 10.

(f) The EPA proposal is demonstrably biased in failing to distinguish between prenatal and postnatal exposure to MeHg, and to note that the Faroe Island study concluded as early as 2006 that "postnatal methylmercury exposure had no discernible effect."[11] This reality is consistent with Seychelles SCDS analysis, which found "several associations between postnatal MeHg biomarkers and children's developmental endpoints. However, as has been the case with prenatal MeHg exposure in the SCDS main cohort study, no consistent pattern of associations emerged to support a causal relationship."[12]

(g) The EPA proposal gave outdated information for 1999-2000 and 2001-2002 (and based its discussion of risk) regarding the crucial public health endpoint for MeHg exposure risk of American women of childbearing age (16-49) and children (1-5) based on the Center for Disease Control and Prevention's (CDC) National Health and Nutrition Examination Survey (NHANES). This WS report corrected EPA's failure by showing the NHANES results for 2003-2004, 2005-2006 and 2007-2008. ***The good news for American public health is that the high-end 95th percentile levels of blood mercury measured for both women and children have undergone systematic decreasing tendencies from 1999 through 2008, and the 2007-2008 values are significantly below the already exaggerated RfD "safe" level established by EPA.*** This reality raises the puzzling question: who or which group in particular are EPA's proposed NESHAP rules supposed to protect, when available NHANES monitoring efforts clearly demonstrate that the overwhelming majority of Americans are already safe from any risk attributable to MeHg exposure through fish consumption?

(h) The EPA proposal failed in reviewing scientific literature, and understanding how to link MeHg exposure to cardiovascular health for adults. This report shows that the two major studies used by EPA (to imply causal link from MeHg to negative cardiovascular health) are flawed in design and the results are simply not applicable to fish-eating adults in America, or to U.S. public health in general.

(i) Authors of the EPA proposal failed to report and fully account for the most important role of dietary selenium's protective effects against MeHg toxicity. The literature on the beneficial role of dietary selenium[13] (Se) against MeHg toxicity is widespread,[14] and it is well known that the binding affinity of Hg to Se is up to a million times higher than for sulfur – mercury's second-best binding partner.

(j) The EPA proposal neglects several new scientific results and reports that will nullify any concerns about harmful health impacts from eating lake-caught fish. In particular, EPA neglected to consider an important study (funded by the EPA itself) showing that 97.5% of freshwater fish analyzed for a Western U.S. survey have sufficient selenium to "potentially protect them and their consumers against Hg toxicity."

(k) The EPA proposal also appears to reflect an unscientific practice of citing non-peer-reviewed private letters and conference talk presentations as key sources in reaching its major and crucial decision in issuing its NESHAP, whereas only peer-reviewed studies should form the basis for such an important rulemaking.[15]

(l) The EPA proposal is designed to set the stage for creating a socio-political consensus for determining a dose-response relationship on MeHg affecting cardiovascular health[16] for adults, despite very weak factual epidemiological evidence, as documented in this report. Additional research by WS revealed that such a model will allow EPA to promote its claim that increasingly stringent mercury emission reductions from EGUs will result in very significant public health benefits,[17] regardless of whether a causal basis for the asserted connection actually exists.[18]

Historically, MeHg bioaccumulation and methylation occur as a result of factors other than levels of elemental Hg available, either from pre-existing mercury naturally found in the soil, water and air, or from much lower emissions from human activity. ***The scientific literature to date strongly and overwhelmingly suggests that meaningful management of mercury is likely impossible***, because ***even a total elimination of all industrial emissions***, especially those from U.S. coal-fired power plants, will almost certainly **not** be able to affect trace, or even high, levels of MeHg that have been found in fish tissue over century-long time periods. A more rational and informed framework for dealing with the relatively low risk of MeHg exposure through fish consumption is required. ***Thus far, it is clear that the EPA's proposed NESHAP may actually be counter-productive to the protection of American public health.***

Note about the report: The author, Willie Soon, is solely and fully responsible for the scientific content and any errors in this report. The report has been studied and reviewed by the following multidisciplinary experts and professionals (listed alphabetically) covering the full spectrum of physical, chemical, biological, geological, ecological, medical, epidemiological, and statistical sciences.

(1) Professor William M. Briggs, Independent Statistician, http://wmbriggs.com/, USA

(2) Professor Robert M. Carter, James Cook University, Australia

(3) Professor Zigbniew Jaworowski, Central Laboratory for Radiological Protection, Poland (retired)

(4) Professor Selvaraj Kandasamy, Research Center for Environmental Changes, Academia Sinica, Taiwan and Xiamen University, China

(5) Professor David R. Legates, University of Delaware, USA

(6) Professor and Doctor Donald W. Miller, University of Washington School of Medicine, USA

(7) Associate Professor Nils-Axel Mörner, Paleogeophysics & Geodynamics, Stockholm, Sweden

(8) Dr. Kary B. Mullis, http://www.karymullis.com/

(9) Jane M. Orient, president, Doctors for Disaster Preparedness, USA

(10) Professor Arthur B. Robinson, Oregon Institute of Science and Medicine, USA

(11) Professor Noah Robinson, Oregon Institute of Science and Medicine, USA

(12) Dr. Harrison H. Schmitt, Geologist and Apollo 17 Lunar Astronaut, USA

(13) Associate Professor Tom V. Segalstad, University of Oslo.

(14) Professor Mitchell Taylor, Lakehead University, Canada

(15) Professor Wyss Yim, Hong Kong City University, Hong Kong

Table of Acronyms

BNT:	Boston Naming Test
CAA:	Clean Air Act
CDC:	Center for Disease Control and Prevention
CHD:	Coronary Heart Disease
CVD:	Cardiovascular Disease
EPA:	Environmental Protection Agency
EGUs:	Electric Generating Units
Hg:	Elemental mercury
KIHD:	Kuopio Ischaemic Heart Disease Risk Factor Study
MDN:	Mercury Deposition Networks
MeHg:	Methylmercury
NESHAP:	National Emission Standards for Hazardous Air Pollutants
NHANES:	National Health and Nutrition Examination Survey
NRC:	National Research Council
PCBs:	Polychlorinated Biphenyls
RfD:	Reference Dose
SCDS:	Seychelles (Island) Children Development Study
Se:	Selenium
UNEP:	United Nations Environment Programme
WS:	Willie Soon's report

Scientific Report

A scientific reply to specific claims and statements in EPA's proposed NESHAP, available at http://www.epa.gov/airquality/powerplanttoxics/pdfs/proposal.pdf on March 16, 2011 and printed in the Federal Register at http://edocket.access.gpo.gov/2011/pdf/2011-7237.pdf on May 3, 2011.

In what follows, WS (Willie Soon's report) provides a detailed scientific reply and clarification to specific incorrect or misleading claims in the EPA regulatory proposal, using summary charts and peer-reviewed scientific literature. Clearly, even a single violation of accepted scientific practices by EPA would be unacceptable. However, because of the cumulative weight of EPA errors, WS recommends that the EPA NESHAP proposal should be considered largely invalid and should be openly challenged on the basis of its sub-standard science and application of scientific knowledge.

The primary sequence of errors demonstrated by this WS report covers:

(1) The failure of the EPA proposal to recognize the large natural variability of mercury recycling within the atmosphere, ocean, soil and biomass, which dwarf the emissions from U.S. electrical power plants.

(2) The failure of EPA's proposal to fully review and recognize the biological, chemical and physical pathways and factors for converting Hg to MeHg that are beyond any ability of the EPA to modify by merely limiting mercury emissions from electric generating units.

(3) The failure of EPA to recognize that MeHg levels in fish are often naturally high at times without any human emissions of mercury – and even zeroing out mercury emissions from U.S. power plants and ultimately eliminating related atmospheric mercury deposition from those plants – will not be able to realistically affect or lower the MeHg levels in either ocean or freshwater fish.

(4) The failure of the EPA proposal to factor in the most appropriate epidemiological study from the Seychelles Children Development Study concerning the risk of prenatal and postnatal MeHg exposures of the most sensitive populations of U.S. women of childbearing ages from 16 to 49, and children from ages 1 to 5. Proper consideration of the SCDS findings would cause EPA to conclude that the consumption of fish is mostly a nutritious and healthy prospect in America, rather than labeling fish a burdensome or potentially dangerous poison.

(5) The tendency of EPA to exaggerate fears of MeHg exposures, by pointing to very weak scientific evidence or by insisting on employing poorly designed studies that simply have no direct relevance to American public health. The two specific examples shown in this report relate to EPA's assertion that MeHg exposures are

related to and result in cardiovascular health problems for adults and attention-deficit hyperactivity disorders in children.

(6) The clear exaggerations and unrealistic nature of the quantitative risk-benefit analyses provided in this EPA NESHAP proposal (in order to fulfill Executive Order 13563). The NESHAP completely failed to demonstrate any health benefit from limiting mercury emissions, even if one generously assumed that the cost of mercury emission cuts actually is only 1% of the total price tag of $10.9 billion per year that EPA claims will be needed to implement the new NESHAP.

(7) The failure of this EPA document to present the most up-to-date scientific data and information that are readily available and extensively discussed in current scientific literature. Worse still, this EPA NESHAP proposal appears to selectively omit scientific information and data that contradict EPA's predetermined rulemaking and will invalidate any rationale for the proposed new emission rules.

EPA Claim 1: EPA acknowledges that although EGUs contribute significantly to the total amount of U.S. anthropogenic Hg emissions, other sources both here and abroad also contribute significantly to the global atmospheric burden and U.S. deposition of Hg. It is estimated that the U.S. contributes 5 percent to global anthropogenic Hg and 2 percent the total global Hg pool. However, as the U.S. Supreme Court has noted in decisions as recently as Massachusetts v. EPA, regarding the problem of climate change, it is not necessary to show that a problem will be entirely solved by the action being taken, nor that it is necessary to cure all ills before addressing those judged to be significant. 549 U.S. 497, 525 (2007). (pp. 17-18)

Reply to EPA Claim 1: In a November 10, 2009 press release,[19] EPA reveals the "surprising" drop of U.S. mercury emission by 58% since passage of the Clean Air Act Amendments in 1990. This is why it is doubly difficult to support the EPA's insistence on further drastic emission cuts.

To put the role of U.S. mercury emissions in context, WS offers **Figures 1 and 2** taken from the United Nations Environment Programme (UNEP)'s December 2008 report on "The Atmospheric Mercury Assessment: Sources, Emissions and Transport."[20] ***Figure 1 shows that actual mercury emissions from the United States (especially its power plants) are a distant or even insignificant third, compared to 2005 emission outputs from China[21] or India, even though blind statistics would label the U.S. as one of "the three largest countries" discharging mercury.*** Table 5 of Pacyna et al. (2006)[22] reported the top seven mercury emitters in 2000 were China, South Africa, India, Japan, Australia, the United States and Russia, with 604.7, 256.7, 149.9, 143.5, 123.5, 109.2 and 72.6 tons, respectively. This listing further confirms the relatively small amount of mercury being emitted by U.S. sources for the last 10 to 20 years.

Figure 1

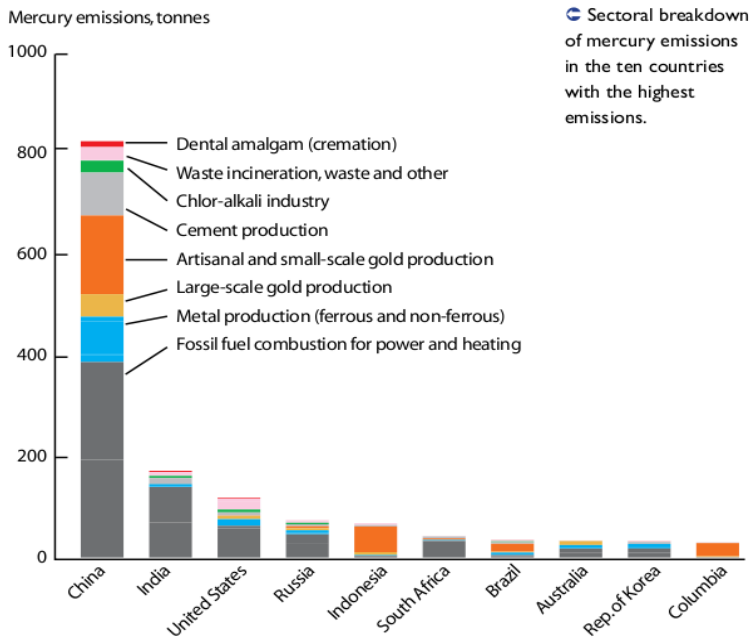
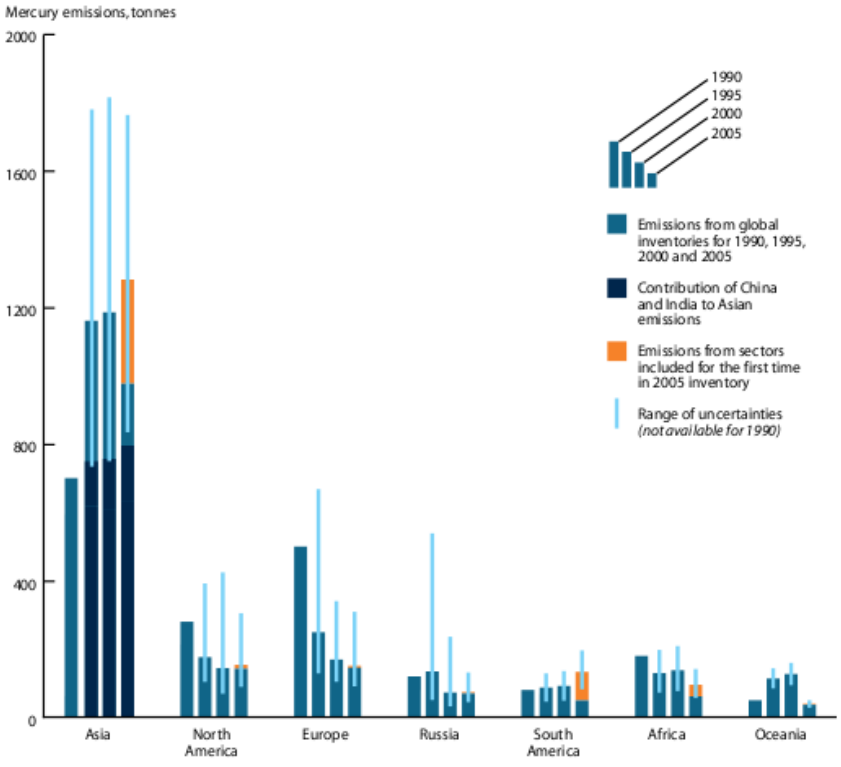


Figure 2



The data documented in **Figure 2** tell us that the mercury emissions history from North America, from 1990 to 2005, reflects significant and systematic improvements in terms of decreasing emissions, despite large increases in energy use for growing economic and industrial activities. In contrast, ***Figure 2 also shows that mercury emissions from Asia, which are dominated by China and India, are not only in large amounts but are also at increasingly larger rates of mercury per year.*** It is also apparent from **Figure 2** that mercury emissions from Europe, as a region, are also relatively larger than combined North American emissions (Canada, the USA and Mexico).

This EPA NESHAP proposal failed specifically in recognizing the relatively well-known discrepancies between the actual measured atmospheric mercury concentrations, in terms of the Total Gaseous Mercury (TGM) in nanogram of Hg per cubic meter of air (ng/m^3), and the expected atmospheric mercury concentrations from current best inventories of anthropogenic mercury emissions as pointed by Slemr et al. (2003)[23] long ago and recently updated in Slemr et al. (2011).[24] ***This scientific point is especially important since EPA's primary assumption for the dominant and specific role of U.S. mercury emissions from EGUs can be shown to be incorrect because of the relative importance of emissions and re-emissions of Hg involving natural sources.***

Slemr et al. (2003) in their pioneering paper pointed out that:

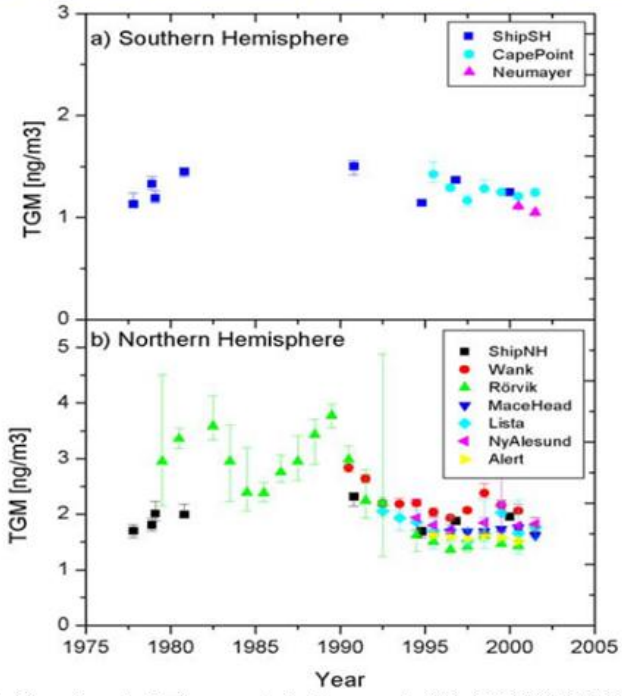
> "The inventories of global anthropogenic emissions of mercury for years from 1979/1980 to 1995 suggest a substantial reduction in the 1980s and almost constant emissions afterwards. ***In contrast to emission inventories, measurements of atmospheric mercury suggest a concentration increase in the 1980s and a decrease in the 1990s.*** Here we present a first attempt to reconstruct the worldwide trend of atmospheric mercury concentrations from direct measurements since the late 1970s. In combination, long term measurements at 6 sites in the northern, 2 sites in the southern hemispheres, during 8 ship cruises over the Atlantic Ocean (1977-2000) provide a consistent picture, suggesting that atmospheric mercury concentrations increased in the late 1970s to a peak in the 1980s, then decreased to a minimum at about 1996, and have been nearly constant since. [see **Figure 3** of this WS report] ***The observed trend is not consistent with published inventories of anthropogenic emissions and the assumed ratios of anthropogenic/natural emissions, and suggests the need to improve the mercury inventories and to re-evaluate the contribution of natural sources.***" [Emphasis added]

Figure 3 gives a summary of this important scientific measurements and monitoring efforts from Slemr et al. (2003). The authors concluded that:

> "Assuming natural emissions and re-emissions to remain constant, the global decrease of the TGM [Total Gaseous Mercury] concentrations of about 17% between 1990 and 1996 would imply a decrease in anthropogenic emissions by about 34% which is about 3-4 times larger than the 10% decrease suggested by estimates by Pacyna and Pacyna [2002] and Pirrone et al. [1996]."

Figure 3

Worldwide trend of atmospheric mercury since 1977

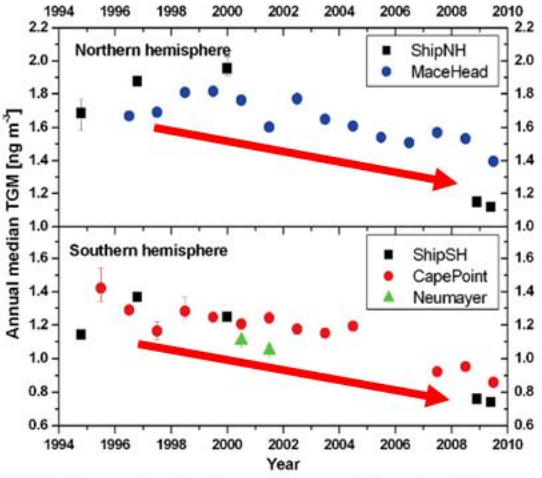


Slemr et al. (2003) Geophysical Research Letters, vol. 30, 2003GL016954

Figure 4

Worldwide trend of atmospheric mercury since 1995

Why is the atmospheric mercury data series decreased by 20% to 38% while the latest 2007 Panel on Source Attribution of Atmospheric Mercury (Lindberg et al. 2007) concluded that "there has been no discernible net change in the size of atmospheric pool of Hg in the Northern Hemisphere since the mid-1970s."?



Slemr et al. (2011) Atmospheric Chemistry and Physics Discussions, vol. 11, 2355-2375

The updated research and results documented in Slemr et al. (2011) offer additional scientific insights to the highly complex nature of mercury cycling and re-cycling in the Earth global atmosphere:

> "Concern about the adverse effects of mercury on human health and ecosystems has led to tightening emission controls since the mid 1980s. But the resulting mercury emissions reductions in many parts of the world are believed to be offset or even surpassed by the increasing emissions in rapidly industrializing countries. Consequently, concentrations of atmospheric mercury are expected to remain roughly constant [or even slightly increasing trend]. Here ***we show that worldwide atmospheric mercury concentrations have decreased by about 20 to 38% since 1996*** as indicated by long term monitoring at stations in the Southern and Northern Hemispheres combined with intermittent measurements of latitudinal distribution over the Atlantic Ocean. [see **Figure 4** of this WS report] ***The total reduction of the atmospheric mercury burden of this magnitude within 14 yrs is unprecedented among most of atmospheric trace gases and is at odds with the current mercury emission inventories indicating nearly constant emissions over the period.*** It suggests a major shift in the biogeochemical cycle of mercury including oceans and soil reservoirs. ***Decreasing reemissions from the legacy historical mercury emissions are the most likely explanation for this decline*** since the hypothesis of an accelerated oxidation rate of elemental mercury in the atmosphere is not supported by the observed trends of other trace gases." [Emphasis added]

Figure 4 presents the summary of the results from Slemr et al. (2011) where the authors also challenged the recent, but obviously out-dated now, consensus conclusion reached by the Panel on Source Attribution of Atmospheric Mercury by Lindberg et al. (2007)[25] where it was claimed that "***there has been no discernible net change in the size of atmospheric pool of Hg in the Northern Hemisphere since the mid-1970s.***" [Emphasis added] The scientific content and basis of the EPA NESHAP proposal would benefit significantly from adding in the results from both Slemr et al. (2003) and Slemr et al. (2010).

The interesting, and important, question for this **Reply to EPA Claim 1** is: how large or small are U.S. mercury emissions, compared to natural sources of mercury emissions and re-emissions?

A new study by scientists from the National Center for Atmospheric Research recently concluded that mercury emissions from fires, mainly biomass burning from forest fires in the Lower 48 U.S. States and Alaska amount to about 44 tons per year.[26] This is similar in magnitude to total mercury emissions from U.S. power plants. In addition to large Hg emissions from active volcanoes (with a median of about 700 tons per year according to Pyle and Mather 2003 and with a lower bound of 80 tons and an upper bound of 4000 tons per year),[27]several recent papers by a team of volcanologists and chemists suggest that just the persistent degassing from relatively quiescent volcanoes emits 75 to 100 tons

of Hg per year.[28] For another perspective, the 2008 UNEP mercury report gives 26 tons per year as the contribution to total atmospheric levels from cremating human remains around the world.[29]

What may be truly surprising to many is that several literature reviews revealed that the total natural contribution of mercury from all sources to the global environment may be in the ball park range of 35,000 tons per year while different authors offered a wide range of 25,000 and 150,000 tons per year.[30] This natural component stands in sharp contrast to estimated annual anthropogenic atmospheric contributions of around 1930 tons (with the range of 1230 tons to 2890 tons) during 2005.[31] Likely the most reliable current estimates of natural mercury emissions (58,000 tons/yr) were reported by Mark Richardson of Risklogic Scientific Service.[32] Mercury sources evaluated by Richardson included direct emissions from volcanoes, soils, soil particles, plants, marine waters, fires, freshwater, meteoritic dust and sea salt; all were substantially underestimated by previous "best-estimates" utilized by the EPA.

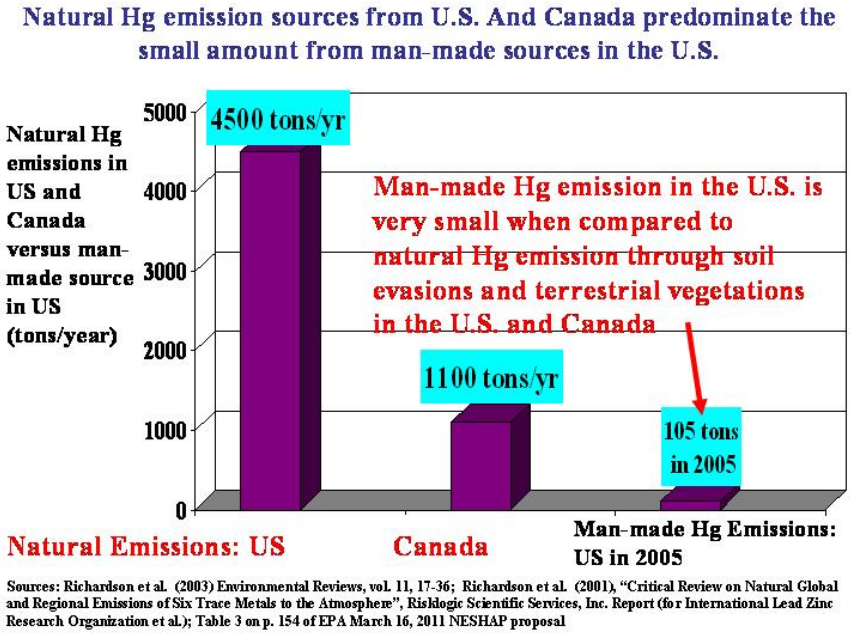
Richardson *et al*. (2003)[33] estimated the natural mercury emissions in Canada are 1100 tons/year and in the US about 4500 tons/year – versus the 105 tons that EPA estimated as the United States' anthropogenic contribution in 2005[34] (shown in **Figure 5**). **This raises major and *reasonable questions concerning how EPA can actually control mercury in the environment***. Momentarily setting aside issues of toxicology and epidemiology, even if 100% capture of man-made Hg emissions were technologically and economically feasible, would it make any difference in the persistent, historical levels of micro-trace MeHg present in fish affected by deposition from the vast North American air mass?

Figure 6 shows the new result from Ribeiro Guevara et al. (2010)[35] pointing out the naturally large contribution of mercury from volcanic events and forest fires that were detected in the pristine lakes of the Nahuel Huapi National Park situated in the Andes of Northern Patagonia. These authors commented that:

> "High Hg levels in the pristine lacustrine ecosystems of the Nahuel Huapi National Park, a protected zone situated in the Andes of Northern Patagonia, Argentina, have initiated further investigations on Hg cycling and source identification. … Observed background Hg concentrations, … ranged from 50 to 100 ng/g dry weight (DW), whereas the surficial layers reached 200 to 500 ng/g DW. In addition to this traditional pattern, ***two deep domains in both sequences showed dramatically increased Hg levels reaching 400 to 650 ng/g DW; the upper dated to the 18th to 19th centuries, and the lower around the 13th century. These concentrations are not only elevated in the present profiles but also many-fold above the background values determined in other fresh water sediments, as were also Hg fluxes, reaching 120 to 150 $\mu g/m^2/yr$ in Lake Toncek.*** No correlation was observed between Hg concentrations and the contents of organic matter, subfossil chironomids, biogenic silica, or the other elements determined. However, ***distinctly increased Hg concentrations were observed immediately above some tephra layers, suggesting a link to volcanic events. Extended fires might be another potential atmospheric source because the***

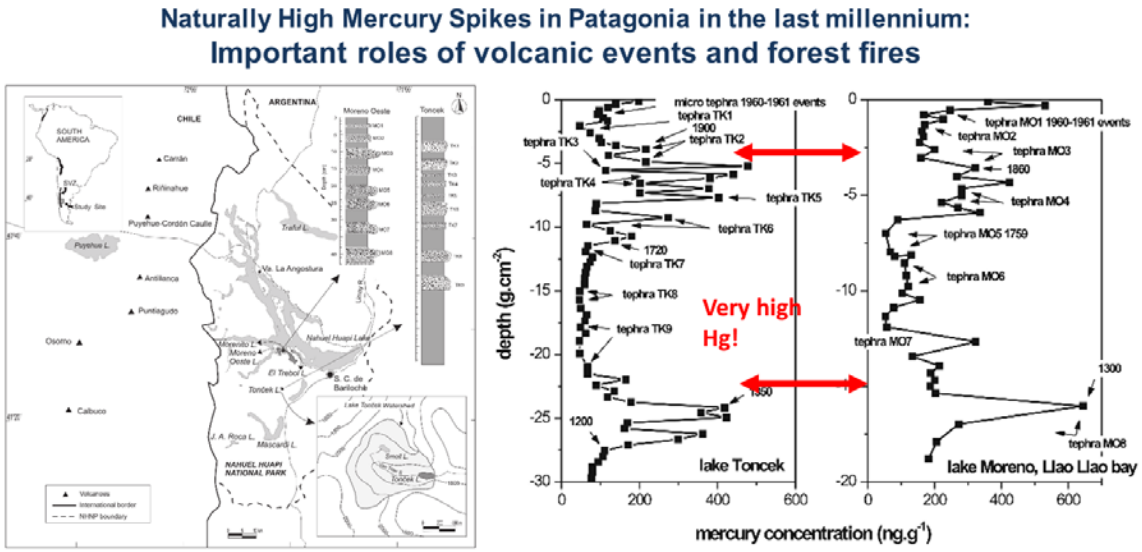
earlier Hg peaks coincide with the reported charcoal peaks, whereas the upper Hg peaks coincide with evidences of extended forest fires from tree-ring data and historical records." [Emphasis added]

Figure 5



Sources: Richardson et al. (2003) Environmental Reviews, vol. 11, 17-36; Richardson et al. (2001), "Critical Review on Natural Global and Regional Emissions of Six Trace Metals to the Atmosphere", Risklogic Scientific Services, Inc. Report (for International Lead Zinc Research Organization et al.); Table 3 on p. 154 of EPA March 16, 2011 NESHAP proposal

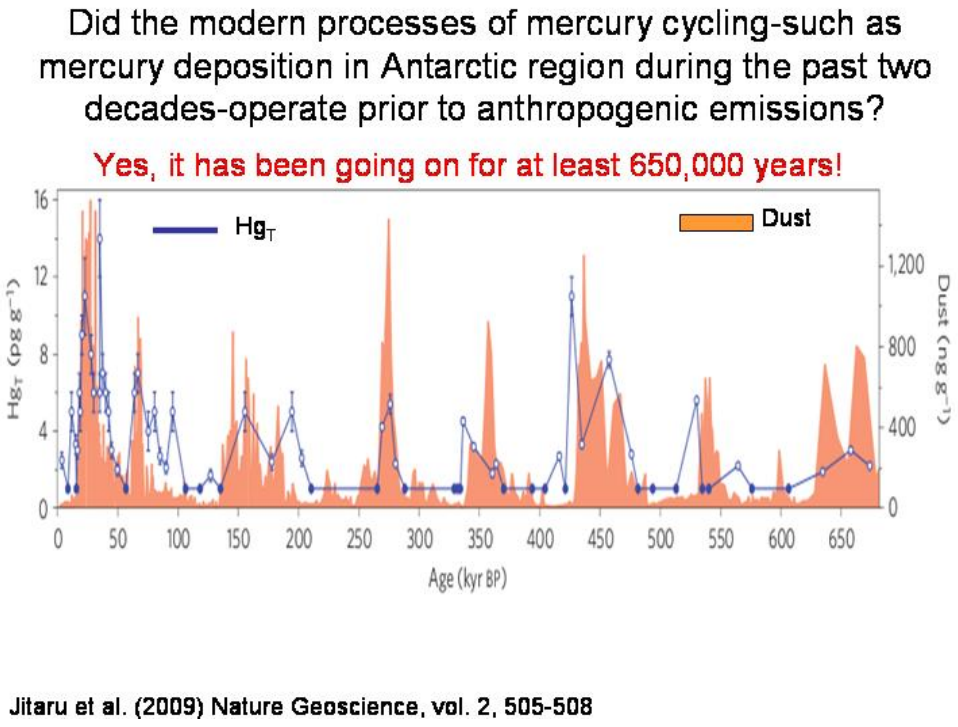
Figure 6



Ribeiro Guevara et al. (2010) Atmospheric Chemistry and Physics, vol. 10, 3443-3453

What is further lacking in EPA's approach – and the artificial elevation of the "significance" of mercury emissions from U.S. EGUs – is the historical and natural context of the essentially uncontrollable mercury recycling within the global atmosphere, ocean, soils, and various geological sources and sinks. **Figures 7 and 8** present the informative new results by Jitaru *et al*. (2009)[36] that successfully measured and estimated the range of natural variation of atmospheric mercury concentration and deposition flux for the past 650,000 years over Antarctica. The results clearly confirmed large natural changes in atmospheric mercury fluxes without any human sources. Even according to EPA's questionable NESHAP rules, there is serious doubt that any reduction in the minor U.S. EGU contribution would actually and positively affect the outcome by any measurable amount in either the environment or U.S. public health. ***Thus, the regulatory goal should be, not to cure "all ills," but to seek science-based commonsense management of exposure risk to eternally present and uncontrollable MeHg.***

Figure 7

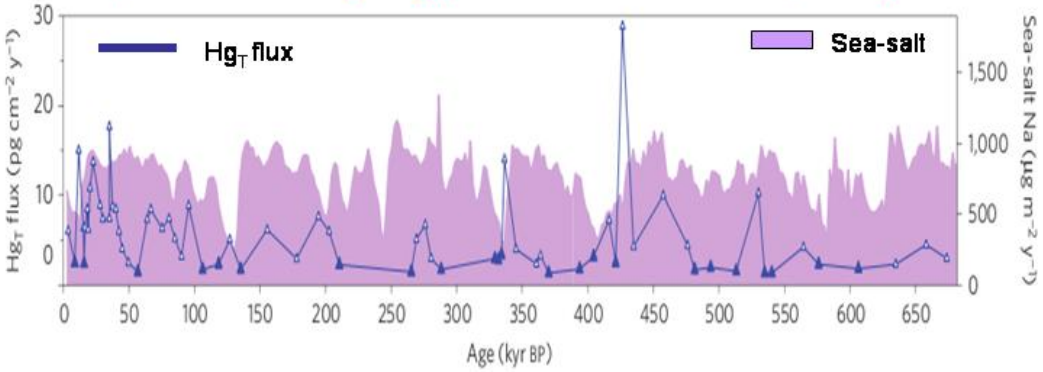


Jitaru et al. (2009) Nature Geoscience, vol. 2, 505-508

Figure 8

Did the modern processes of mercury cycling-such as mercury deposition in Antarctic region during the past two decades-operate prior to anthropogenic emissions?

Yes, it has been going on for at least 650,000 years!



Jitaru et al. (2009) Nature Geoscience, vol. 2, 505-508

EPA Claim 2: Consistent with the recently issued Executive Order (EO) 13563, "Improving Regulation and Regulatory Review," we have estimated the cost and benefits of the proposed rule. The estimated net benefits of our proposed rule at a 3 percent discount rate are $48 to 130 billion or $42 to $120 billion at a 7 percent discount rate. (p. 21)

SUMMARY OF THE MONETIZED BENEFITS, SOCIAL COSTS, AND NET BENEFITS FOR THE PROPOSED RULE IN 2016 (MILLIONS OF 2007$)[a]

	3% Discount Rate			7% Discount Rate		
Total Monetized Benefits[b]	$59,000	to	$140,000	$53,000	to	$130,000
Hg-related Benefits[c]	$4.1	to	$5.9	$0.45	to	$0.89
CO_2-related Benefits	$570			570		
$PM_{2.5}$-related Co-benefits[d]	$59,000 to $140,000			$53,000 to $130,000		
Total Social Costs[e]	$10,900			$10,900		
Net Benefits	$48,000	to	$130,000	$42,000	to	$130,000
Non-monetized Benefits	Visibility in Class I areas					
	Cardiovascular effects of Hg exposure					
	Other health effects of Hg exposure					
	Ecosystem effects					
	Commercial and non-freshwater fish consumption					

Reply to EPA Claim 2: Without challenging the risk-benefit analysis, EPA's summary table directly confirms the fact that the "Hg-related benefits" are truly miniscule and insignificant when compared to (a) the $PM_{2.5}$-related "co-benefits" and (b) the stated cost of $10.9 billion per year in implementing the proposed NESHAP rules. Even if one ***assumes*** that just 1% of the total cost (i.e., $109 million) will be needed to install and operate mercury emission control equipment and technologies, the maximum $6 million of "Hg-related benefits" are simply an insignificant gain for the very high cost of making the reductions.

However, WS also challenges the artificial reality and exaggerated atmospheric deposition scenarios that EPA used in arriving at this already highly optimistic "Hg-related benefits," under several of the EPA claims discussed below.

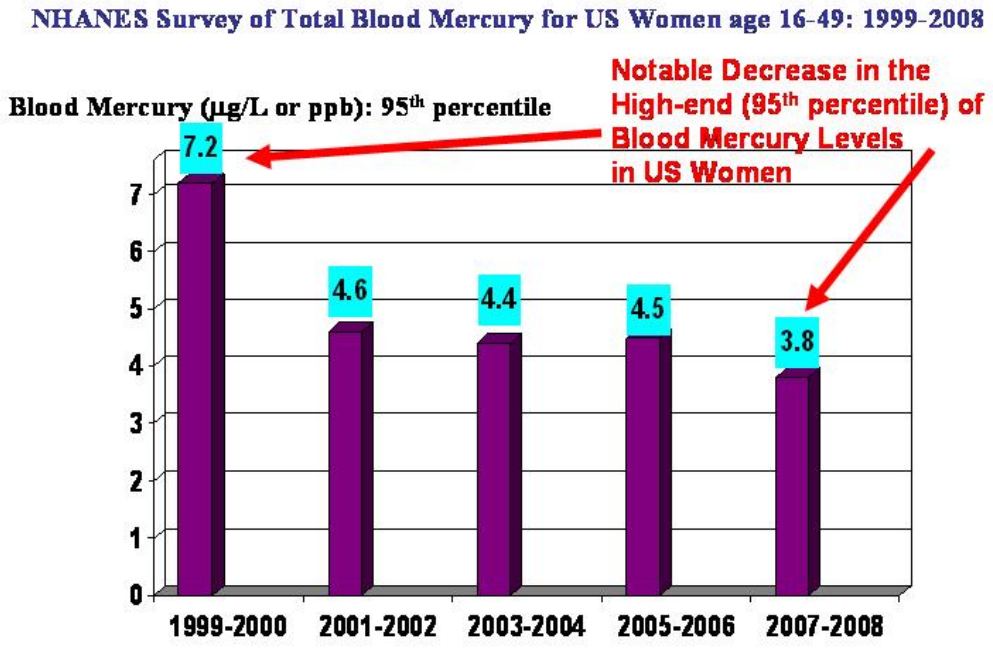
EPA Claim 3: Finally, the Agency had already delayed in completing the section 112(n)(1) studies. Additional delay would have been unreasonable because of the persistence of Hg in the environment and its tendency to bioaccumulate up the food chain, both aspects of Hg in the environment that make it critical to limit additional releases to the environment as quickly as possible. In addition, delay would have been unreasonable because EPA estimated at that time that about 7 percent of women of child-bearing age, one of the most at-risk populations, was exposed to Hg at levels exceeding the RfD, and EPA knew that as the level of exposure above the RfD increased, the level of risk and the extent and severity of adverse effects increased. Thus, EPA reasonably made the appropriate and necessary determination in 2000 to ensure that the largest unregulated domestic source of Hg would be required to install controls, thereby achieving an incremental reduction in the risk associated with a persistent, bioaccumulative HAP. (pp. 118-119)

Reply to EPA Claim 3: This strong sense of urgency in issuing and executing NESHAP rules and controls is mistaken. EPA's claim that 7 percent of women of child-bearing age have blood mercury above the agency's excessively stringent RfD was actually based on the CDC NHANES data set collected for 1999-2000 and ultimately published in Schober *et al*. (2003).[37]

Imagine now asking EPA to update all the NHANES blood mercury data monitored biyearly since 1999-2000. What would one see in the absence of the strict mercury emission controls that EPA is advocating for EGUs in the U.S.? Would the number of women and children in the U.S. with blood mercury exceeding EPA's RfD (of about 5.8 ppb in human blood) keep increasing, since there have been no direct and regulated controls on mercury emissions from EGUs?

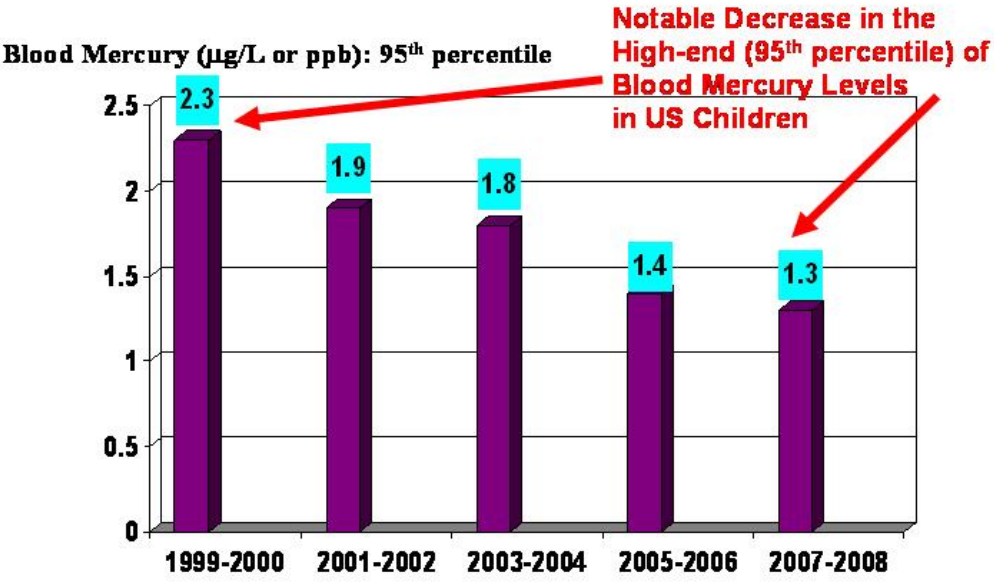
Figures 9 and 10 show the high-end 95th percentile values of blood mercury actually measured for women of childbearing age (16 to 49 years old) and young children (1 to 5 years old) for the NHANES results sampled biyearly from 1999-2000 through the latest dataset of 2007-2008. ***It should be immediately obvious to EPA and especially the agency's proposed NESHAP authors that rushing to judgment and implementing a new body of rules would be a mistake. During the past decade of available NHANES monitoring records, exposure risks for U.S. women and young children to MeHg in fish have apparently improved significantly, without any new strict Hg emission controls for EGUs.*** What is also especially relevant and important to note about the data presented in **Figures 9 and 10** is that they came from a summary created by EPA itself,[38] in addition to data collected by CDC and discussed in Caldwell *et al*. (2009).[39]

Figure 9



Data Source: CDC NHANES and EPA updates and Caldwell et al. (2009) International Journal of Hygiene and Environmental Health, vol. 212, 588-598

Figure 10



Data Source: CDC NHANES and EPA updates and Caldwell et al. (2009) International Journal of Hygiene and Environmental Health, vol. 212, 588-598

Another important new result comes from the Baltimore THREE (Tracking Health Related to Environmental Exposures) Study, in which Wells *et al*. (2011)[40] measured umbilical cord blood mercury and other chemicals for some 300 mothers between ages of 14 and 43 years. These authors found mean umbilical cord blood mercury level of 1.37 μg/L or 1.37 ppb, which is far below EPA's already stringent RfD of 5.8 ppb. Only 5 values (or about 1.7%) were above EPA's RfD. Finally, Wells et al. (2011) also reported that Asian-American infants had 87% higher cord blood mercury values than Caucasian infants mainly because higher rates of seafood consumption. African-American infants had 51% higher cord blood mercury values than Caucasian infants. It should be noted that there are only 5 cord blood mercury values in this Baltimore study above EPA's highly stringent RfD, so the exposure risk of these Asian-American and African-American infants is really not near dangerous levels. It is relevant to point out that this study is partly funded by EPA while one cannot find such relatively encouraging information in this EPA NESHAP proposal.

EPA Claim 4: Mercury is a persistent, bioaccumulative toxic metal that is emitted from EGUs in three forms: gaseous elemental Hg (Hg^0), oxidized Hg compounds (Hg^{+2}), and particle-bound Hg (Hg_P). Elemental Hg does not quickly deposit or chemically react in the atmosphere, resulting in residence times that are long enough to contribute to global scale deposition. Oxidized Hg and Hg_P deposit quickly from the atmosphere impacting local and regional areas in proximity to sources. (p. 139)

Reply to EPA Claim 4: It is clear that this particular claim in EPA's NESHAP proposal is neither accurate nor representative of the wider literature, which documented the fact that atmospheric deposition is simply not the primary mechanism or pathway for mercury cycling and recycling among its various reservoirs within the coupled air, land and water systems.

Critical findings by Edward Krug and Derek Winstanley of the Illinois State Water Survey clearly show that man-made mercury from atmospheric deposition is a very small contributor to the huge amount of natural mercury already contained in the soils of Illinois specifically and the nation generally.[41] After measuring mercury soil content, they estimated that it would take ***9,000 years*** at current atmospheric deposition rates to account for all the mercury present in just the top 380-cm of Illinois soils.

Similar analysis for a composite of all U.S. soils yielded an estimate of ***14,000 years*** at current atmospheric deposition rates to attribute U.S. soil mercury content to man-made "air pollution." Krug and Winstanley (2004) made their point crystal clear:

> "When widespread Hg pollution first became a popular concern, global anthropogenic Hg was compared to global soil Hg as part of a larger literature that criticized the common presumption that the principle source of Hg in the environment is anthropogenic. Regarding world soil Hg content, these early analyses reported that anthropogenic activities could have increased world soil Hg content by ***0.02 percent***. ***Despite this early seminal literature and a persistent stream of publications in following decades, the presumption that anthropogenic Hg is the principle source of Hg in the soils that mantle landscapes is still common*** **and** ***exerts a powerful effect on scientific and public perception*** of the role of anthropogenic atmospheric Hg deposition on the environment of Illinois and the USA. [In this work,] ***the hypothesis that most Hg in Illinois and the USA soils is of anthropogenic origin is rejected.***" [Emphasis added]

A study of metal concentrations, including Hg, in rural topsoil in South Carolina by Aelion et al. (2008)[42] where:

> "Using a Medicaid database, two areas were identified: one with no increased prevalence of mental retardation and developmental delay (MR/DD) (Strip 1) and one with significantly higher prevalence of MR/DD (Strip 2) in children compared to state-wide average. These areas

were mapped and surface soil samples were collected from 0-5 cm depths … Samples were analyzed for [metal concentrations]. ”

The results for Hg content in Strip 1 and 2 in the state of South Carolina yield a mean of 0.03 ppm and range of 0-0.12 ppm, and of 0.04 ppm and 0.025-0.051 ppm, respectively. Thus, no significant differences in Hg content in Strip 1 and 2 despite the higher prevalence of MR/DD for children in Strip 2. ***Such a result essentially tells us that it is extremely difficult to confirm or suggest any mental retardation and developmental delay in children from greater exposure to mercury within their local environment.***

In another important study of sources of mercury emission and dispersal and recycling of mercury in the urban city of Changchun at Northeast China, Fang *et al*. (2004)[43] found that ***“Only 11.6% of Hg emitted from coal combustion [was] deposited onto land surfaces in the urban district and the rest [of Hg] participated in the regional or global cycle.”*** Changchun is a metropolitan city of 158 km^2 with 2.92 million people; in 2001, the total coal consumed for the energy-use in the area was estimated to be about 7 million tons. It is clear that there is no mercury hotspot (see **Figure 11**) as implied in several of EPA’s discussion in this NESHAP proposal.

Figure 11

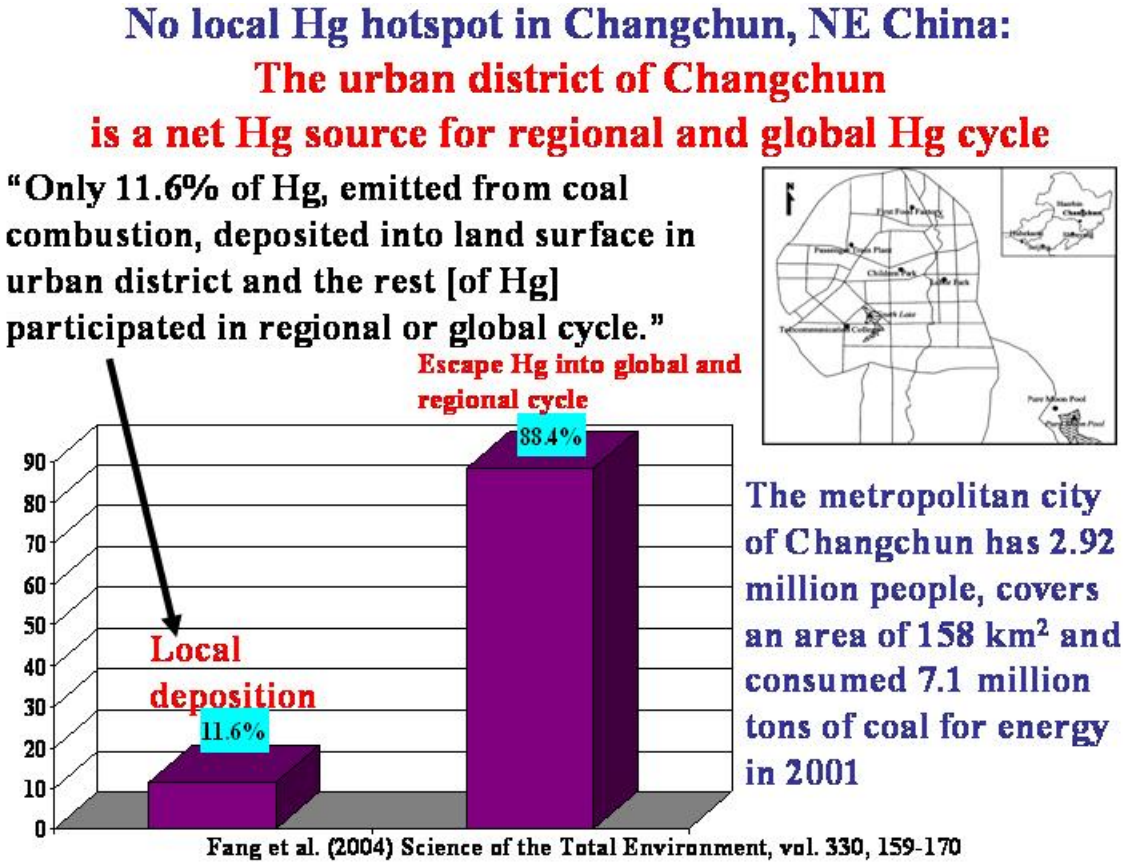


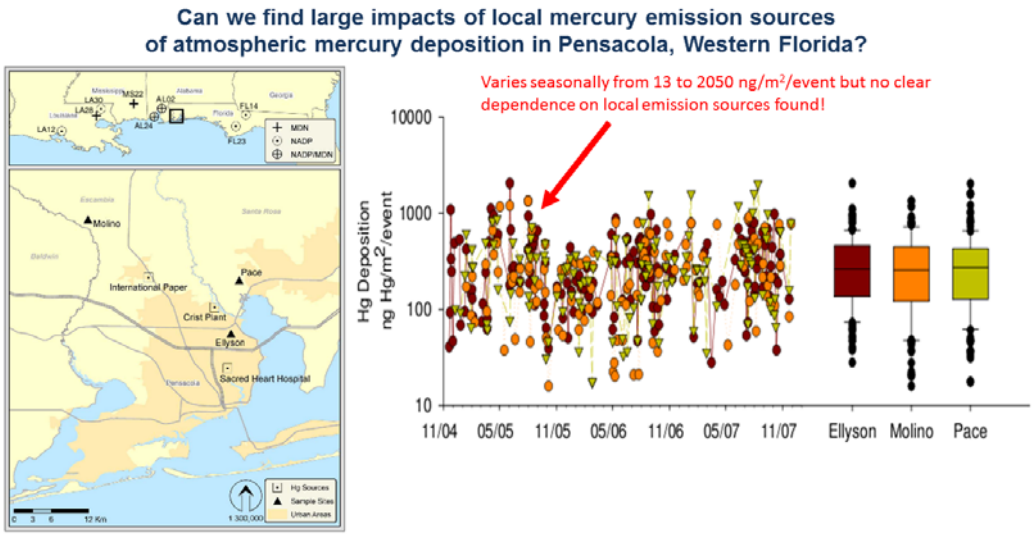
Figure 12 offers another reality check to the oft-assumed claims by EPA that local and regional mercury emission sources will almost automatically have large impacts on the local and regional deposition of atmospheric mercury. Not so according to the new study from Pensacola Bay, Florida published in Caffrey et al. (2010).[44] The results suggest large variability but no clear or direct connection to local mercury emission sources:

> "These results have shown that there were no significant differences in mercury deposition among our three sampling sites or between our sites and MDN sites in the Northern Gulf of Mexico between 2005 and 2007. The mercury deposition at Ellyson site, which is about 4.8 km away and generally downwind of the Crist coal fired power plant, was not significantly higher than at Molino site which furthest away from local mercury emission sources (24 km from Crist power plant and 14 km from International Paper). ***Our results contrast with studies in Michigan and Maryland which found higher mercury deposition in urban areas compared to rural areas and those finding higher concentrations of mercury in rainwater close to coal-fired utilities.***" [Emphasis added]

Instead of connections to local emission sources, Caffrey et al. (2010) pointed out that:

> "Sea salt aerosols are important in sequestering reactive gaseous and particulate mercury in the Gulf Coast. In this study, mercury deposition was significantly correlated with sodium deposition, however, the correlation coefficient was quite low (r=0.22, p=0.02). Sea salt aerosols have a significant impact on chemistry of rain water in Pensacola Bay area. … The distance of the sampling site to the Gulf of Mexico determines how significant an effect the sea salt aerosols have on rain water composition. Sites that are further from the Gulf have lower average annual sodium deposition than those near to it. … Fifty km was the distance where the line becomes asymptotic and the sea salt effect is lost."

Figure 12



Caffrey et al. (2010) Atmospheric Chemistry and Physics, vol. 10, 5425-5434

Figure 13 shows the results by Shotyk *et al*. (2005),[45] suggesting that atmospheric deposition of Hg at the Faroe Islands (estimated from concentration of Hg deduced in layers of a peat bog sedimentary core from Myrarnar, Faroes) actually dropped by more than 50% between 1954 (when deposition was calculated to be 34 μg/m^2/year) and 1998 (when deposition was deduced to be 16 μg/m^2/year). ***If such a drop in atmospheric deposition yields no noticeable effects on MeHg levels in pilot whale or cod in the area of the Faroe Islands during the last 30-50 years,[46] then what benefits can we expect to receive from EPA's new NESHAP rules, which would require mercury emission cuts of 91% from already low-emitting U.S.-based EGUs?***

Figure 13

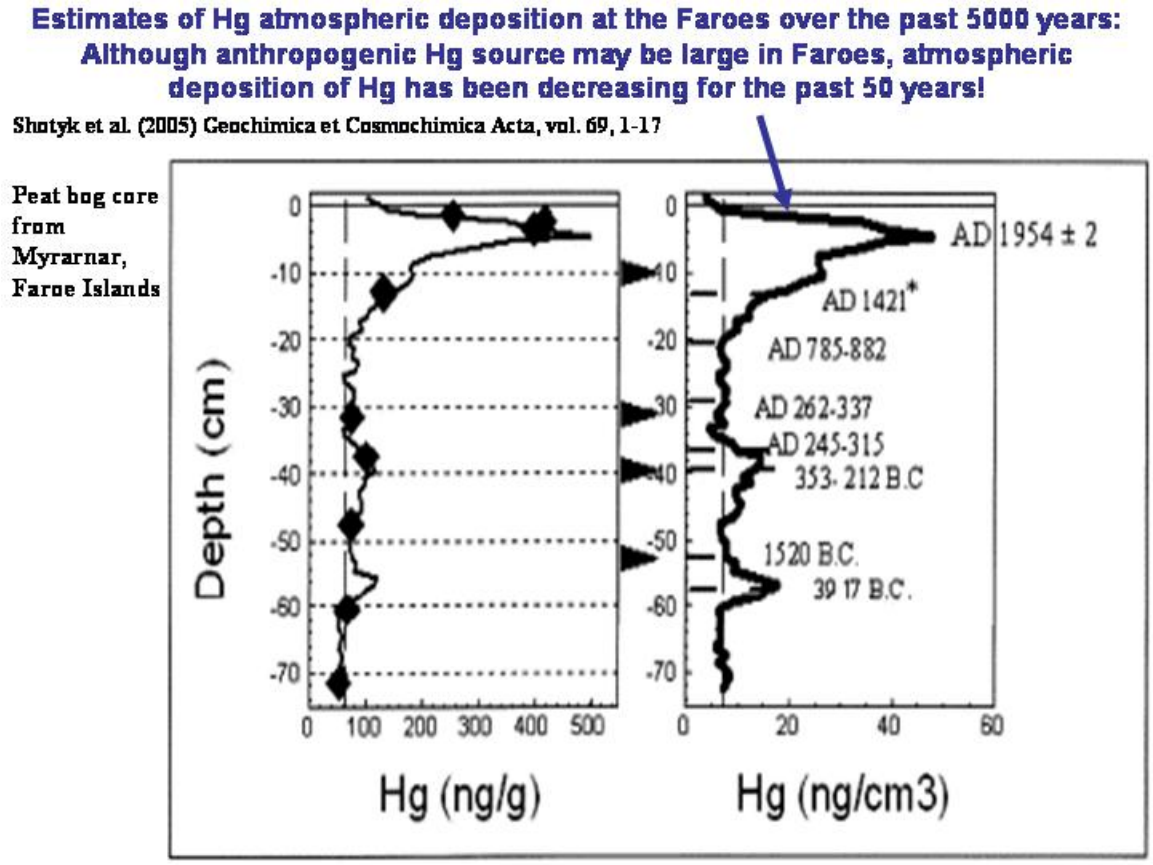
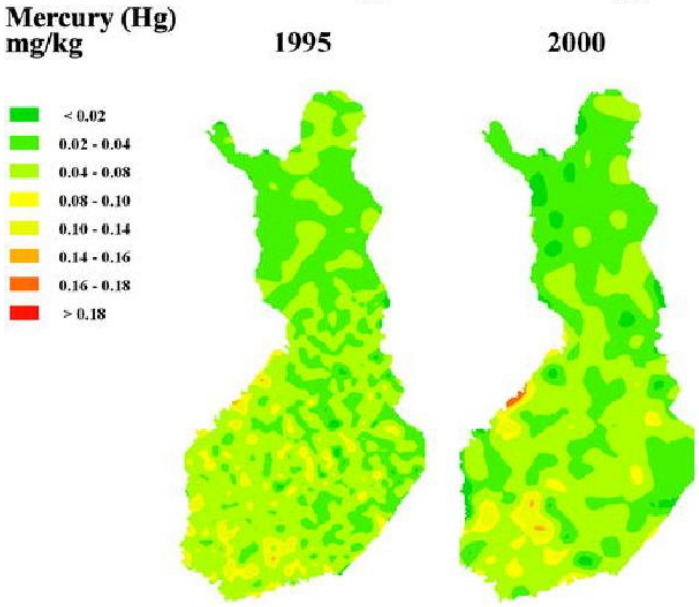


Figure 14 reinforces the point that large decreases in anthropogenic mercury emissions may occur without a corresponding systematic reduction in the actual mercury deposited from the atmosphere. The case in point is the novel work of Poikolainen *et al*. (2004),[47] ***showing no clear or systematic decrease in mercury deposited in Finland between 1995 and 2000, despite the significant 41% decrease in mercury emissions during the 1990s in Finland.*** (It is significant to note that Poikolainen et al. (2004) was also able to demonstrate a corresponding decrease in emission and deposition for other trace heavy metals like lead and cadmium.)

Figure 14

Large (41%) reduction in total emission of mercury in Finland during the 1990s but no clear decrease in mercury deposited (as measured in feather moss)



"Anthropogenic emissions and total deposition of Hg decreased during the 1990s throughout the whole Europe. Mercury emissions in Finland decreased during the 1990s from 1100 to 650 kg. However the decrease in emissions has not had any significant decreasing effect on Hg concentrations in mosses in the northern most parts of Finland. "

Poikolainen et al. (2004) Arctic, Antarctic, and Alpine Research, vol. 36, 292-297

In the new study of the mercury budget for a forested boreal catchment area in southeast Norway, Larssen *et al*. (2008)[48] concluded that:

> "The pool of Hg_{tot} stored in the catchment was about 8000 times larger than the annual Hg_{tot} output by stream water and about 2000 times larger than the input estimated from throughfall and litterfall. … ***Theoretically, this means that even if Hg depositions were to cease completely, sufficient Hg_{tot} is stored in the catchment to keep the current level of Hg output for several thousand of years.*** The estimated soil pool may be too large to be explained from historical atmospheric input only which suggests that there could be some mineral sources of Hg." [Emphasis added]

In any attempt to link industrial emissions of elemental mercury to mercury contents in our land and waters, it is important to note the somewhat "surprising" new results on "Seasonal variations in mercury concentrations in the coastal waters of Kalpakkam, southeast coast of India" by Sathpathy et al. (2008),[49] which was published in the August 10, 2008 issue of *Current Science*:

> "Studies were carried out in the coastal waters of Kalpakkam to monitor the seasonal variation in mercury (Hg) concentration. The Hg level

(dissolved + acid leachable) ranged from 3 to 50 ppb for surface and 1.5 to 47.9 ppb for bottom-water samples, yielding an annual average concentration of 20.42 ± 11.44 and 23.11 ± 13.06 ppb for surface and bottom waters respectively. Strong positive correlation of Hg with salinity and its relatively low concentration during monsoon and post-monsoon showed ***concentration of Hg in this coastal water was mainly of marine origin, indicating absence of any other external source of input. Relatively high Hg concentrations were encountered in bottom samples compared to the surface. Madras Atomic Power Station (MAPS) outfall discharge water did not showed elevated Hg level compared to ambient surroundings. The present observed values are significantly lower (30 times) than the earlier reported values from this coast."*** [Emphasis added]

Futhermore, Sathpathy et al. (2008)[50] documented important scientific confusion or possibly "foul-play" by one earlier study,[51] which rushed to link "industrial Hg" to natural levels of mercury contained in our land and waters:

"Although Hg values observed during the present study are marginally higher than those reported, the values reported by Selvaraj et al. [1999] were found to be significantly higher than ours (>30 times). The values reported in Selvaraj et al. ranged from 64 to 1374 pp in the surface and 0 to 1664 ppb in bottom-water samples during the pre-monsoon period, and 0 to 526 ppb in the surface and 0 to 321 ppb in the bottom-water samples during the post-monsoon period. Values in the present study ranged from 3 to 50 ppb for surface and 1.5 to 47.9 ppb for bottom-water samples. This showed that the highest value observed during the present study is about 30 times less than the earlier reported values. It is difficult to understand how the Hg concentration as reported by Selvaraj et al. was so high. The only simple and possibly convincing reason for such high values could be due to analytical error during extraction or during instrumental analysis or long duration between acidification and filtration leading to significant contribution of leachable fraction. ***Selvaraj et al. had attributed the higher concentration of Hg to anthropogenic from the coastal industries. But interestingly, there is no coastal industry located at the Kalpakkam coast, except MAPS [Madras Atomic Power Station], which does not use Hg for any purpose.*** Moreover, such a conclusion has been drawn without knowing the content of the discharge. ***The above conclusion could have been dialectical had the MAPS outfall water been analyzed for Hg.*** During the present study, the fourth location represented the outfall discharge from MAPS, and Hg values at this station throughout the year were comparable with the remaining stations. In fact, most of the values from this location were relatively low compared to the other stations. ***Thus, the discharge outfall from MAPS is no way different from the coastal water with respect to Hg content,*** as evident from the present study. Selveraj et al. have reported that to control biofouling and bio-corrosion in

> the cooling water system of MAPS, chlorine has been used intermittently- a distorted fact as only low-dose continuous chlorination has been in vogue at MAPS since 1988. Similarly, the source of chlorine used by MAPS, as mentioned by Selvaraj et al., was through the electrolytic chlorination process, which they had attributed as the cause of elevated levels of Hg observed at Kalpakkam coast. On the contrary, at MAPS, chlorination is done by injection of liquefied chlorine gas in the sea water. ***Selvaraj et al. have assumed MAPS as a pollution source. The results of the present study clearly show that the discharge from MAPS condenser does not contain elevated levels of Hg."*** [Emphasis added]

The recent review of the science of mercury deposition by Lindberg et al. (2007)[52] emphasized the lack of good understanding on the nature of natural and recycled Hg emissions:

> "Although we have a relative good understanding of primary anthropogenic emissions, this is not the case for natural and recycled (Hg [that was quickly] emitted after being deposited) Hg emissions. ***Assuming vigorous atmospheric oxidation of Hg implies that vigorous emissions from natural surfaces must be compensating. In this case, reducing or even eliminating anthropogenic emissions of Hg would have a smaller than expected effect on the total emission flux. Significant reductions in deposition would not occur until a commensurate fraction of the Hg now involved in the global-scale cycle is somehow sequestered (e.g., in geologic formations, much as it was before human industrialization).*** However, if atmospheric oxidation of Hg is slow, then more of the observed deposition flux must be coming from anthropogenic emissions of reactive Hg. In this case, reductions in anthropogenic emissions of Hg would lead to a more rapid decrease in total Hg deposition flux. The importance of resolving these issues for policy development is clear. Their associated uncertainties critically impact our ability to predict the outcome of any proposed emission-control strategies. " [Emphasis added]

EPA Claim 5: Methylmercury is formed by microbial action in the top layers of sediment and soils, after Hg has precipitated from the air and deposited into waterbodies or land. Once formed, MeHg is taken up by aquatic organisms and bioaccumulates up the aquatic food web. Larger predatory fish may have MeHg concentrations many times, typically on the order of one million times, that of the concentrations in the freshwater body in which they live. (pp. 139-140)

Reply to EPA Claim 5: EPA's current NESHAP proposal suggests that a decrease in elemental Hg emissions from U.S. power plants will somehow (i.e., mainly through unvalidated computer modeling) decrease the amount of MeHg accumulated in fish tissue. WS therefore asks: how does the bio-transformation of elemental (Hg) mercury into methylmercury (MeHg) actually work?

The biochemical transformation of Hg into MeHg, and its ultimate bioaccumulation up the food chain, is not easily predictable. Neither is the process simply dependent on manipulation of raw Hg inputs, regardless of source or magnitude. ***This important point has been clear in the scientific understanding because the world ocean is estimated to contain some 40 to 200 millions tons[53] of mercury and yet little of it has been converted into MeHg to be accumulated in fish resulting in the poisoning of other creatures.***

In the new 2009 study, "Spatial variability in mercury cycling and relevant biogeochemical controls in Florida Everglades,"[54] authors Liu *et al*. (2009) conclude that:

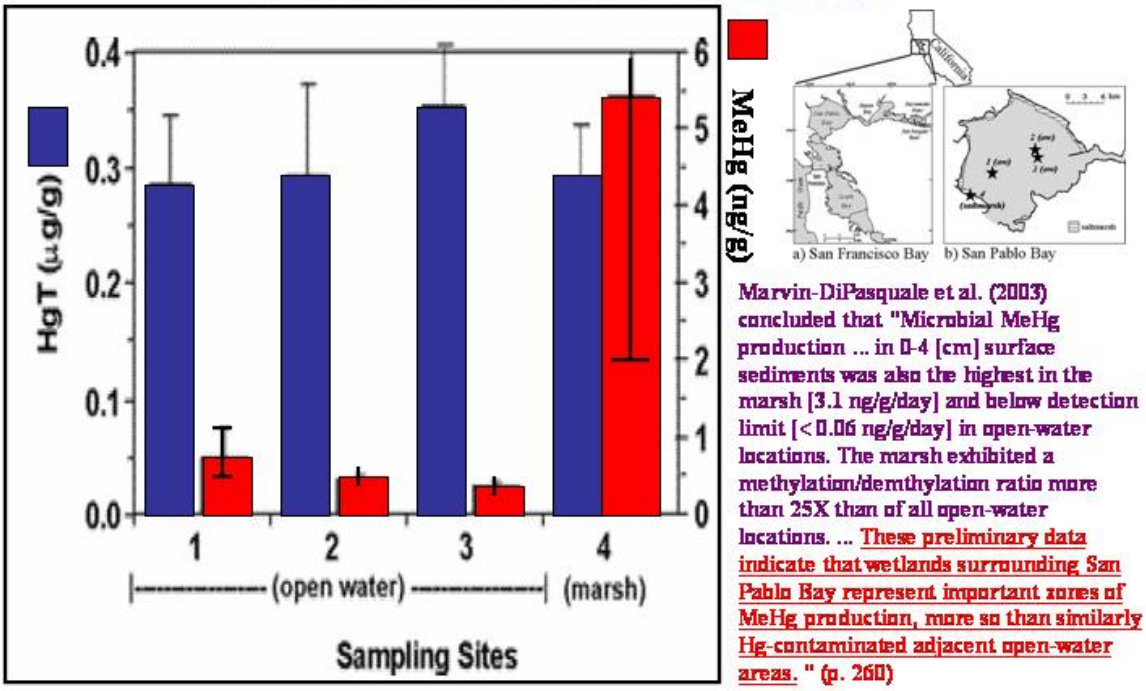
> "Multiple biogeochemical characteristics, such as surface water dissolved organic matter (DOC_{SW}), pH, chloride, and compositional properties of solid compartments (soil and floc), ***were identified to be important factors controlling THg [total mercury] distribution.*** … Higher mosquitofish THg and bioaccumulation factor were observed in the central and southern Everglades, partially in accordance with periphyton MeHg distribution, but not in the 'hot spot' areas of water, soil, or floc MeHg. The discrepancy in mercury bioaccumulation and mercury distribution in environmental compartments ***suggests that in addition to MeHg production, biogeochemical controls that make MeHg available to aquatic organisms, such as DOC_{SW} and compositional properties of soil and floc, are important in mercury bioaccumulation.***" [Emphasis added]

Figure 15 confirms the admission by the authors of EPA's NESHAP proposed rules that trace levels of MeHg in fish depend on the complex physical, chemical, and biological factors within each unique ecosystem. More importantly, it demonstrates 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 15**) 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 15**). 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".[55]

Figure 15

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)

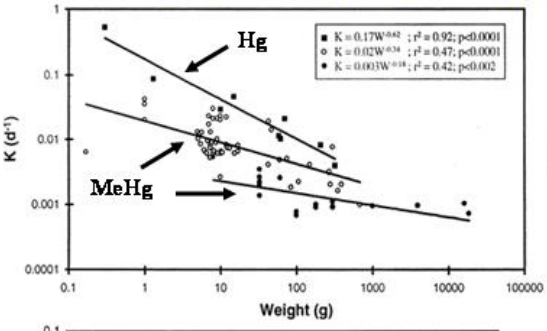
Reference: Marvin-DiPasquale et al. (2003) Environmental Geology, vol. 43, 260-267

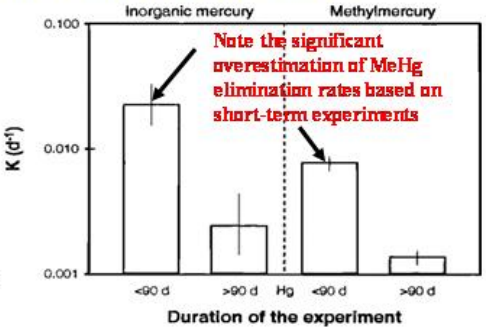
The San Francisco Bay findings add to the body of evidence showing that neither adding nor reducing Hg atmospheric deposition from any coal-fired power plant would measurably affect MeHg levels in San Francisco Bay ecosystems. To the contrary, ***MeHg levels are naturally self-limited*** by specific ecosystem dynamics: water quality variables like dissolved sulfate, algae and/or zooplankton population parameters, the availability of nutrients, amounts of sunlight, water and air temperatures, and other factors.

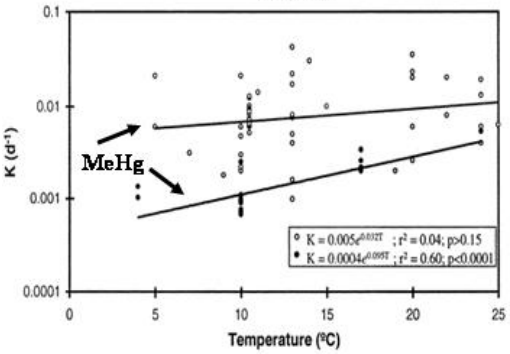
Figure 16 shows that an increase water temperature is likely to demethylate more MeHg,[56] as suggested by the increasing trend in the parameter K or demethylation rate as temperature increases, reflected in the chart at the bottom panel of **Figure 16**). **Figure 17** makes the additional point that the "temperature" effect just shown in **Figure 16** may be confused by the effects of sunlight and dissolved organic matter. Studies by Siciliano *et al*. (2005)[57] suggest that sufficient amounts of dissolved organic matter can render MeHg production capable of being abiotically enhanced by solar radiation. (Note the day-night cycle of MeHg levels, corresponding to the effects of the sunlight cycle, and the additional laboratory experiments confirming the role of sunlight in this important work.)

Figure 16

The elimination rate (K) of MeHg (and Hg) from fish as functions of body weight and water temperature







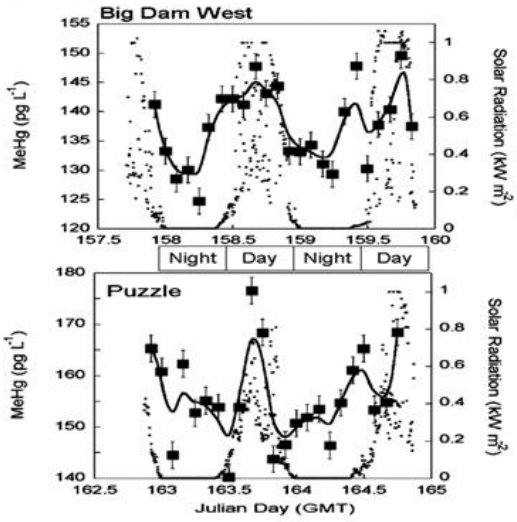
"Our analysis showed that short-term experiments (< 90 days) overestimated the elimination rate of mercury and that inorganic mercury was excreted 3-fold faster than MeHg. Both Hg and MeHg excretion were negatively correlated to body size, but only MeHg elimination was significantly correlated to water temperature. MeHg elimination tended to be overestimated by a factor of 2-6 by empirical models that were published prior to this study. A field test showed that our model adequately estimated the elimination rate of MeHg under natural conditions."

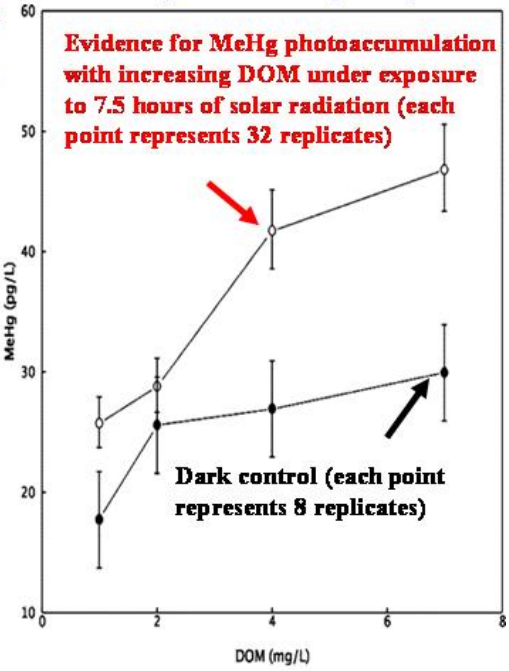
Trudel & Rasmussen (1997) *Environmental Science & Technology*, vol. 31, 1716-1722

Figure 17

Unexpected abiotic production of MeHg by solar radiation in freshwater lakes with helps from Dissolved Organic Matter (DOM)

"The significance of this process to freshwater lakes and the mechanism responsible for MeHg photoproduction is still unclear, but a correction in the conventional wisdom that MeHg is rapidly photodegraded is timely."

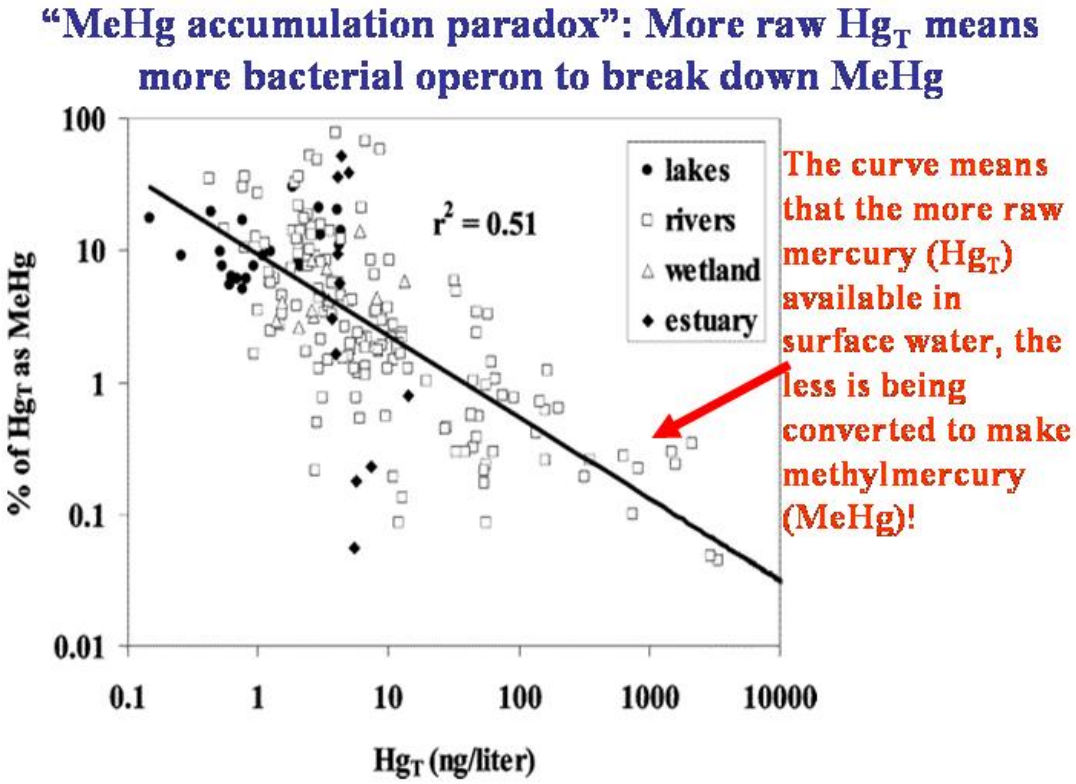




Siciliano et al. (2005) *Environmental Science & Technology*, vol. 39, 1071-1077

Figure 18 shows a very important observation concerning the accumulation of MeHg in various watershed systems. Schaefer *et al*. (2004)[58] discusses a particular "MeHg accumulation paradox" – the relative percentage of MeHg actually converted from available raw Hg actually *decreases* as the amount of available raw Hg *increases*. The authors of these equally 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 or *mer* operon) available to significantly break down the MeHg produced, and thus explaining the observed "MeHg accumulation paradox." ***Once again, the EPA's proposed NESHAP needs to fully account for the underlying science before requiring costly and ineffectual emission reductions.***

Figure 18



Schaefer et al. (2004) Environmental Science & Technology, vol. 38, 4304-4311

According to Mason *et al*. (2005),[59] the correlated factors of sulfate-organic matter-bacterial activity could **"possibly cause an *increase* in fish mercury concentration *even as atmospheric deposition decreases*"** [emphasis added]. The key point is that science-based observations reveal that both the production and destruction processes of MeHg ending up in fish *do not* depend exclusively on the amount of Hg available in a water system. Hence, key biological and chemical processes driving the methylation and demethylation – and the ultimate bioaccumulation of MeHg in fish tissue – *completely overwhelm* any insignificant contributions of Hg from U.S. power plant emissions, for example. This is because there already exist millions of tons of naturally occurring Hg in Earth's water, soil, sediment and atmosphere, ever-ready and ever-available for

conversion into MeHg. ***This explains why it is neither difficult nor surprising to find high, even extremely high, levels of MeHg in both fish and humans (see data and figure shown in this endnote)[60] in the past several centuries, at times when mercury emissions from power plants or other industrial applications were minimal or nonexistent.***

To further demonstrate the complexity and uncertainty of Hg to MeHg transformation pathways, WS proffers a ***partial*** listing from the literature of variable and interactive conversion and transformation factors:

(1) levels of MeHg are independent of raw Hg levels (Marvin-DiPasquale *et al*. 2003; Paller *et al*. 2004; Bonzongo & Lyons 2004),

(2) pH and sulfate (Bonzongo & Lyons 2004),

(3) leaf litter inputs and microbial growth (Balogh *et al*. 2003),

(4) roles of visible light (Seller *et al*. 1996), UVA (Lalonde *et al*. 2004), diurnal MeHg and solar radiation (Siciliano *et al*. 2005),

(5) experimental treatments with sulfate (Harmon *et al*. 2004),

(6) water temperature and fish body weight (Trudel and Rasmussen 1997),

(7) algal bloom-induced biodilution of MeHg in zooplankton Daphnia (Pickhardt *et al*. 2002),

(8) dependence of MeHg on species of zooplankton (Masson & Tremblay 2003),

(9) "MeHg accumulation paradox" (Schaefer *et al*. 2004),

(10) seasonal cycle of MeHg before and after control flooding (St. Louis *et al*. 2004), and

(11) 48 environmental variables, including land use, various catchment areas and lake characteristics, lake water chemistry and fish stocks (Soneston 2003).

The new study by Holloway et al. (2009),[61] which carefully examines various competing factors for governing the biomethylation and bioaccumulation processes for MeHg in a historic mercury mining area, further illustrates why the simplistic connection scenario of Hg-MeHg in this EPA NESHAP proposal is wrong:

> "The relationships between soil parent lithology, nutrient concentrations, microbial biomass and community structure were evaluated in soils from a small

> watershed impacted by historic Hg mining. Upland and wetland soils, stream sediments and tailings were collected and analyzed for nutrients (DOC, $SO_4^{=}$, NO_3^{-}), Hg, MeHg, and phospholipids fatty acids (PLFA). Stream sediment was derived from serpentinite, siltstone, volcanic rocks and mineralized serpentine with cinnabar, metacinnabar and other Hg phases. Soils from different parent materials had distinct PLFA biomass and community structures that are related to nutrient concentrations and toxicity effects of trace metals including Hg. ***The formation of MeHg appears to be most strongly linked to soil moisture, which in turn has a correlative relationship with PLFA biomass in wetland soils. The greatest concentrations of MeHg (> 0.5 ng/g MeHg) were measured in wetland soils and soil with a volcanic parent (9.5-37 μg/g Hg).*** Mercury methylation was associated with sulfate-reducing bacteria, including *Desulfobacter* sp. and *Desulfovibrio* sp., although these organisms are not exclusively responsible for Hg methylation. … ***Sulfate was not a limiting factor in Hg methylation as concentrations are elevated in all soil pore waters. Total Hg concentrations span three orders of magnitude in this historic Hg mining district, with the greater concentrations appearing to suppress the biological formation of MeHg.***" [Emphasis added]

As a more local illustration of mercury cycling complexity facing would-be regulators, consider that quantitative estimates of elemental mercury re-volatilization in the Great Lakes of about 2.3 to 13.7 tons per year. Estimated direct deposition of all species of mercury from the atmosphere is estimated at only 4.7 tons per year.[62] This suggests a perplexing management accounting in which natural re-volatilization output could easily exceeds total input from the atmosphere. ***This illustrates the futility of micro-management of Hg-MeHg bio-chemical states in the natural environment, be it in the U.S. or anywhere in the world.***

A full flavor of the scientific complexity, and the true level of scientific ignorance concerning the life cycles of MeHg, can be understood from the latest work by the distinguished scientist Francois Morel, Albert G. Blanke Professor of Geoscience at Princeton University, and students (Ekstrom and Morel 2008): **"Cobalt limitation of growth and mercury methylation in sulfate-reducing bacteria."**[63]

> "Sulfate-reducing bacteria (SRB) have been identified as the primary organisms responsible for monomethylmercury (MeHg) production in aquatic environments, but little is known of the physiology and biochemistry of mercury (Hg) methylation. … Here we explore the role of corrinoid-containing methyltransferases, which contain a cobalt [Co]-reactive center, in Hg methylation. … These results are consistent with mercury being methylated by different pathways in the two [SRB] strains: catalyzed by a B_{12}-containing methytransferase in *D. multivorans* and B_{12}-independent methyltransferases *in D. africanus*. If complete-oxidizing SRB like *D. multivorans* account for the bulk of MeHg production in coastal sediments as reported, the ambient Co concentration and speciation may control the rate of Hg methylation."

Poulain et al. (2007)[64] documented "the first evidence that microbes in the high Arctic possess and express diverse *merA* genes, which specify the reduction of ionic mercury [Hg(II)] to the volatile elemental form [Hg(0)]" and emphasized "the importance of microbial redox transformations in the biogeochemical cycling, and thus the toxicity and mobility, of mercury in polar regions."

Finally, Schaefer and Morel (2009)[65] recently discovered that:

> "mercury methylation by the bacterium *Geobacter sulfurreducens* is greatly enhanced in the presence of low concentration of the amino acid cysteine. … ***Our results suggest that mercury uptake and methylation by microbes are controlled more tightly by biological mechanisms than previously thought, and that the formation of specific mercury complexes in anoxic water modulates the efficiency of the microbial methylation of mercury.***" [Emphasis added]

Another important aspect on the natural cycling and speciation of mercury is the fact that methylated form of mercury (both as mono-methylmercury and dimethylmercury) can be found in high concentrations for example in deep water regions with relatively higher oxygen utilization (including Antarctic Intermediate Water and Antarctic Bottom Water and equatorial sub-thermocline regions). [66] ***Pongratz and Heumann (1999),[67]*** through experimentations on production of methylated mercury (and lead and cadmium) by marine macroalgae done in Pongratz and Heuman (1998),[68] ***estimated that the biogenic productions of dimethylmercury from Antarctic Ocean (Pacific part from 51°S to 71°S), Arctic Ocean (Greenland Sea from 64°N to 79°N) and Atlantic Ocean (North-South Profile from 38°N to 58°S) may amount to 210 tons/year, 240 tons/year and 1900 tons/year, respectively.*** This is a significant natural component of methylated mercury. In another experiment, Limper et al. (2008)[69] recently demonstrated that the gut of the Australian termite *Masteotermes darwiniensis* "possesses the capability to form methyl mercury."

Last but not the least, the key role of selenium (Se) in affecting the bioaccumulation of MeHg must also be incorporated in the EPA's modeling of the Hg-MeHg connection. Although the complex mechanisms and processes that are related to how Se itself is bioaccumulated and transferred among different trophic levels have been recently reviewed in Stewart et al. (2010)[70] and Jardine and Kidd (2011),[71] WS wish to highlight the specific mercury-related results reported in Belzile et al. (2006):[72]

> "***In freshwater environments, the bioaccumulation of Hg by biota was retarded by elevated concentrations of Se in water of experimental ecosystems and Se-treated lakes.*** Similarly, Hg concentrations in largemouth bass (*Micropterus salmoides*) increased significantly after the elimination of Se-rich discharges to a quarry pond. … In this study, we showed that selenium (Se) deposition from metal smelters in Sudbury, Ontario, greatly reduces the bioassimilation of mercury (Hg) by aquatic

> biota throughout the food web. Concentrations of total and methyl mercury in tissues of zooplankton, mayflies (*Stenonema femoratum*), amphipods (*Hyalella azteca*), and young-of-the-year perch (*Perca flavescens*) were positively correlated with the increasing distance from Sudbury smelters and showed weak or no correlation in most of the studied aquatic species, methyl mercury and total Se showed much clearer inverse trends. Similar to the results of our previous study of muscle tissues from adult perch and walleye (*Sander vetreus*), ***these findings suggest that Se plays an important role in limiting the whole-body assimilation of Hg at lower levels of the aquatic food chain. High Se concentration may force a preferential assimilation of the element over Hg through a competitive adsorption on binding sites. This may also restrict the solubility and availability of Hg to aquatic organisms or reduce the methylation of this metal in lakes.***" [Emphasis added]

WS further notes that other equally important, scientific aspects and discussion related to Se are given in endnote #13 and **Reply to EPA Claim 10** below.

EPA Claim 6: The NAS recommended that neurobehavioral deficits as measured in several different tests among these studies be used as the basis for the RfD. The NAS proposed that the Faroe Islands cohort was the most appropriate study for defining an RfD, and specifically selected children's performance on the Boston Naming Test (a neurobehavioral test) as the key endpoint. (p. 142)

Reply to EPA Claim 6: Apart from the few well-reported acute ***direct*** mercury poisoning cases in the past – such as occurred in Minamata, Japan[73] – there has been ***no convincing finding*** that regular consumption of fish with historically natural levels of mercury has caused "learning disabilities" in children. The best "evidence" presented for this claim is derived from a challenged study in the Faroe Islands, commonly used to suggest that prenatal exposure to trace levels of MeHg might cause subtle deficits in neuropsychological performance.

The U.S. Environmental Protection Agency's (EPA) dose level of concern is called a mercury reference dose (RfD). Understanding it is essential to sorting out the entire mercury debate and discussion which follows below.

An RfD is an *estimated* ("with uncertainty spanning perhaps an order of magnitude") amount of methylmercury that one can consume *every day*, *over a life time of 70 years* without appreciable risk of deleterious effects. The estimation of this EPA "safe" dose involves several judgments such as (a) the choice of the most *appropriate* No Observed Adverse Effect Level (NOAEL) or Benchmark Dose (BMD) of the critical effect, and (b) the choice of the *appropriate* uncertainty factors, based on a review of the entire database.[74]

Ultimately, EPA decided on a BMD of 58 ppb for blood mercury level and then further divided this BMD value by another "safety" factor of 10 to arrive at an RfD standard of 5.8 ppb in blood mercury level. EPA's rather extreme uncertainty factor of 10 exceeds those of other medical bodies and institutions.

Viewed alternatively, EPA's mercury RfD "safe" dose of ***5.8 ppb*** (parts per *billion*) when measured in human blood is equivalent to an intake of 0.1 (µg MeHg/kg-day) or about ***1.0 ppm*** (parts per *million*) when measured in human hair. For a context, EPA's mercury reference dose of 0.1 µg/kg/day is a factor of 2 to 4 lower (i.e., more stringent) than other estimates from human health organizations. The FDA dose was established at 0.4, the Agency for Toxic Substances and Disease Registry (ATSDR) at 0.3, and the newly revised FAO/WHO level at 0.21.[75]

Thus, WS concludes that ***EPA's current mercury RfD is the most restrictive in the world.***

That is to say, EPA appears to have set the stage for unnecessary alarm by (a) setting "low" estimates of safe levels, making things look worse than they are in reality, and (b) setting "high" estimates of exposure risks, again making things look even worse than

reality. The combination of the two can be highly confusing for the American public. Perhaps a more appropriate approach would be establishing a ***realistic* "safe" *range***, instead of a single number beset with confusion and wide latitude for statistical manipulation.

The Problematic Faroe Islands Study

An inappropriate study

EPA's mercury RfD is further problematic because it is based on inappropriate studies of Faroe Islands inhabitants who consume both fish (found to be generally low in mercury) and pilot whale meat and blubber (a unique practice no one in the U.S. pursues) containing multiple, confounding chemicals (PCBs, lead, cadmium, pesticides, persistent organic pollutants, DDT, etc.), of which mercury is only one.[76]

Adding to this chemical cocktail, according to a report released recently by the Danish government, significantly high levels of perfluorooctane sulfonate (PFOS), perfluorooctane sulfonamide (PFOSA), polychlorinated naphthalenes (PCNs), and polybrominated diphenyl ethers (PBDEs) were also found in pilot whales from the Faroe Islands.[77]

Regardless of the scientific debate (examined below) about whether the interpretation of the Faroese data constitutes sound or reliable evidence, it is clear that the study is simply ***inappropriate*** for EPA use in determining safe fish mercury exposure to Faroese children, let alone U.S. children.

Dr. Pal Weihe, co-researcher of the Faroe study and Chief Physician of the department of occupational and public health for the Faroese hospital system implicitly agreed with this assessment in a letter to the Boston Herald dated February 9, 2004:

> "In the Boston Herald, Friday, February 6, 2004, p. 20, the following was stated about a mercury study in the Faroe Islands conducted in cooperation with the Harvard University[78]: "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.***" [Emphasis added]

This poses the question, how can EPA or the National Research Council (NRC) seriously cling to the Faroe study as a basis for their RfD formulation, when the lead author of the study states that MeHg levels in Faroese children have nothing to do with MeHg in fish, but only in whale meat and blubber; which were highly contaminated with a mixture of known neurotoxins?

In **Reply to EPA Claim 8** below, WS reports on the latest result from the Faroe study in Yorifuji et al. (2010)[79] where the concomitant exposure effects of MeHg and lead on cognitive deficit in 7- and 14-year-old children were found. Here, WS wishes to note yet another notable conclusion from the Faroe study where co-exposure of Faroe adult population to MeHg and PCBs is now recognize to be a key factor for Parkinson disease (Petersen et al. 2008a[80]):

> "Parkinson's disease (PD) occurs in Faroes at a prevalence twice as high as expected. … This study aimed to investigate the association of [PD] with dietary exposure to polychlorinated biphenyls (PCBs) and methylmercury (MeHg) in a community with increased exposure levels. … Increased intake of whale meat and blubber in adult life was significantly associated with PD, thus suggesting a positive association between previous exposure to marine food contaminants and development of PD."

The same group of authors (Petersen et al. 2008b[81]) however concluded that prenatal exposure to MeHg alone ***does not*** affect the risk of Parkinson's disease and ***thus once again emphasizes the multi-co-pollutant nature of exposure risks from the unusual dietary intake by the Faroe Island population.***

There are additional serious issues of inappropriateness for the underlying epidemiological data from the Faroe Island children studies; not the least of which is the refusal of the Faroe Islands researchers and the Danish government to release their raw data for independent statistical analyses and verification. By continuing to rely on this black-boxed data, both EPA and the NRC violate EPA's own data quality guidelines.[82]

The Boston Naming Test problems

Nevertheless, EPA and this NESHAP proposed emission rules continue to rely heavily on the 2000 NRC Report, which concludes that the Boston Naming Test results of the Faroe Islands study are an appropriate basis for EPA's mercury RfD. However, following a lengthy trial, a San Francisco Superior Court found in May 2006 that:

> "[T]he NRC failed to cite a critical paper in which the Faroe Islands authors state that a new cohort was being formed in the Faroe Islands to study the role of PCBs. Following the publication of the NRC report, four papers have been published discussing the high levels of PCBs in the Faroe Islands.

> When investigators controlled for concurrent PCB exposure, ***there was no statistically significant correlation between methylmercury exposure and performance deficits on the Boston Naming Test.*** The authors of the Faroe Islands study recognized the impact of PCBs, rather than methylmercury, on the results of the Boston Naming Test, noting that 'especially for the Boston Naming Test, the PCB concentration appeared to be an important predictor' of the children's performance."[83] [Emphasis added]

Former EPA official and co-author of the mercury RfD, Dr. Deborah Rice, published a paper in 2003 in which she herself concluded that PCBs ***caused the reported performance deficiencies measured by the Boston Naming Test***.[84]

However, according to the Judge's findings, during the San Francisco trial, Rice inexplicably denied ever writing the paper. But when shown the article in court, Dr. Rice finally "admitted to reviewing and approving it, and that the article was published under her name."[85]

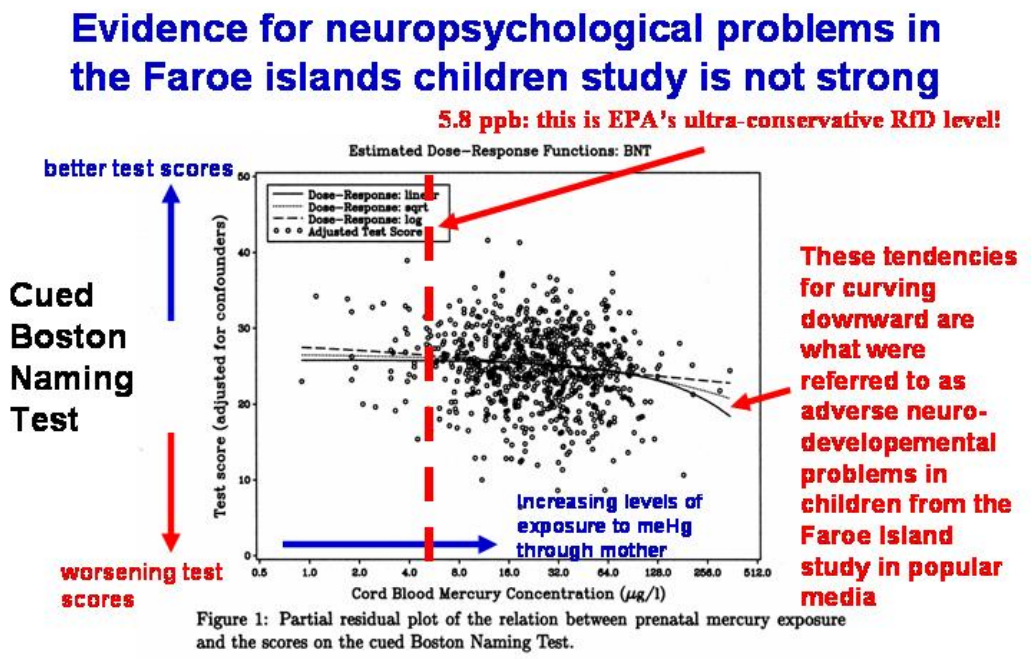
Finally, the authors of the Faroe study themselves admitted to PCB confounding:

> "Prenatal exposure to PCBs was examined by analysis of cord tissue from 435 children from a Faroese birth cohort... The association between cord PCB and cord-blood mercury (r=0.42) suggested possible confounding. While no PCB effects were apparent in children with low mercury exposure***, PCB-associated deficits within the highest tertile of mercury exposure indicated a possible interaction between the two neurotoxicants.*** The limited PCB-related neurotoxicity in this cohort appears to be affected by concomitant methylmercury exposure." [86] [Emphasis added]

Considering the actual data itself, **Figure 19** shows the rarely presented "smoking gun" evidence adopted by EPA and the NRC 2000 review to support claims of negative neuro-developmental impacts from prenatal MeHg exposure through marine fish consumption. The result was drawn from the Faroe Islands children study originally published by Grandjean *et al*.[87] and the endpoint test is the so-called cued Boston Naming Test.

Figure 19 clearly suggests a significant scatter in the test scores as the MeHg exposure level changes. In this curve fitting, there appears no strongly discernable trend even around 100 ppb, let alone at 5.8 ppb. In other words, EPA's adopted MeHg RfD of 5.8 ppb in blood shows a clear ***disconnect*** with the underlying data. ***If the EPA's "safe" MeHg level of 5.8 ppb is valid or correct, then large majority of the Faroese children in this study should have been declared to be mentally defective and unfit to function in society.*** Again, these data led Judge Robert Dondero (the presiding judge at the San Francisco Superior Court during the 2006 decision) to conclude, following expert testimony, that "The Boston Naming Test has no statistically significant relationship to methylmercury exposure."

Figure 19



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

It is worth repeating that this particular endpoint is among the *best* evidence relied upon by EPA and the NRC to suggest negative impacts with increasing MeHg exposure.

Figure 19 point*s to the distinction between actual "potential levels of harm" or concern for MeHg in prenatal exposure – and the ultra-precautionary level of safety set by EPA's RfD.* No equivalent epidemiological data has been produced demonstrating serious health concerns in adults from chronic exposure to methylmercury through dietary fish consumption. In fact, growing evidence exists for just the opposite.

Summary of Faroe Islands study problems

- Unique dietary habits of consuming pilot whale meat and blubber, making the Faroese an inappropriate subject group for an American RfD for MeHg exposure risk.
- Faroe populations are known to be exposed to the contamination of multiple chemicals from their unique diets and lifestyle factors, some of which are known neurotoxins, including PCBs and lead.
- Pilot whales are lower in mercury-binding selenium[88] that is abundant in ocean fish.
- Claimed relationships to "subtle deficits" are weak and do not fit the CDC definition of "developmental disabilities."

- Mercury concentrations in samples of pilot whale livers have been 5,000 times greater than the Japanese government's limit for mercury contamination of 0.4 ppm.
- Reports that concomitant exposure to MeHg and PCBs has synergistic toxicity.[89]
- PCBs are an important confounder that can lead to a false and misleading correlation between MeHg and childhood development.
- EPA and NRC (2000) ignored confounding chemical contamination in pilot whales – and rejected the Seychelles study, which found no adverse effects from prenatal and postnatal exposures resulting from heavy maternal fish consumption (see **Reply to EPA Comment 7** below).
- The NRC report, which endorsed reliance on the Faroe Islands Study, was published in 2000, before a series of articles focused on PCBs in the Faroes.
- Faroese researchers and the Danish government refuse to release study's raw data for independent analysis and scrutiny.
- The BNT end-point test relied on by EPA and the NRC for setting an RfD is so weak and unconvincing that after hearing testimony a California Court judge found it non-credible.
- Faroe researcher stated "children are *not* exposed to dangerous levels of MeHg by eating fish."
- Faroes fish are relatively low in MeHg so fish could not be a source of harms.
- Cord blood used in tests only detects levels of MeHg in last few weeks of pregnancy. Recent whale intake by test subjects could spike and skew test results. Association of cord blood Hg and brain mercury levels has not been reported.
- Some of the tests, such as finger tapping during a 15-second interval, have no clinical relevance for an individual.
- One of the reputedly strongest pieces of evidence for a detectable MeHg exposure claimed by the Faroe children study is the BAERS (brainstem auditory evoked responses) test at 14-years of age. But there is "no data to suggest that a change of 1/100,000th of a second would constitute a significant impairment in auditory processing and it would certainly not constitute a development disability."[90]
- Potential *post*-natal contamination due to children eating whale products from infancy until age 7, when tests were administered.
- Some mothers of test subjects smoked and/or consumed alcohol during pregnancy.
- Evaluations of children after 7 years of age is no longer double-blind.
- Diets and sources of exposure differ from Seychelles studies. Seychelles diet included fish, fruit and vegetables; Faroe diet included fish, whale meat and fat. (American diet is in no way similar to Faroese diet.)
- Additional deficiency findings of San Francisco Superior Court Judge in *California vs. Tri-Union Seafoods*: The Faroe Islands Study –
  * Has no exposed or reference groups,
  * Lacks a reliable control on exposure,
  * Suffers from incomplete follow-up,
  * Does not adequately identify or quantify biases and confounding factors,
  * Does not adequately separate prenatal from postnatal effects,

Again, it is critical to repeat that EPA defines its RfD for methylmercury as "an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be *without* an appreciable risk of deleterious effects during a lifetime."[91] Further, EPA did not account for the research showing fish are rich in selenium, which likely counteracts MeHg in fish (see the figure shown in this endnote).[92] Pilot whales, on the other hand, are selenium deficient (see discussion on the role of selenium in fish under the **Reply to EPA Claim 10**, below).

Finally, it is very relevant to note the following declaration about the potential conflict of interests and scientific objectivity of the chief scientific researcher of the Faroe Island children study, Dr. Philippe Grandjean in 3 of his research papers[93] starting in 2010:

> "***P.G. has provided paid expert testimony on mercury toxicology for the U.S. Department of Justice in a legal case concerning environmental pollution from coal-fired power plants.***" [Emphasis added]

This speaks to the issue of objective science and neutrality of scientists rather than a scientist acting as both judge and executioner deciding on a predetermined outcome.

EPA Claim 7: Results from all three studies were considered in defining the RfD, as published in the "2001 Water Quality for the Protection of Human Health: Methylmercury," and in the IRIS summary for MeHg: "Rather than choose a single measure for the RfD critical endpoint, EPA based this RfD for this assessment on several scores from the Faroes' measures, with supporting analyses from the New Zealand study, and the integrative analysis of all three studies." EPA defined the updated RfD of 0.0001 mg/kg-day in 2001. **Although derived from a more complete data set and with a somewhat different methodology, the current RfD is numerically the same as the previous (1995) RfD** (0.0001 mg/kg-day, or 0.1 µg/kg-day). (pp. 142-143) [Emphasis added]

Reply to EPA Claim 7: Anyone who read the **Reply to EPA Claim 6** above and realized how badly EPA distorted the science to reach its ***world's most stringent RfD*** for MeHg exposure risk would have to conclude that the remarkably ***exact agreement*** between the RfD derived by EPA during 1995 and during 2001 is mostly an artificial reality.

The Superior Seychelles Mercury Studies

An appropriate study

In sharp contrast to the Faroe Island study, the Seychelles Child Development Study (SCDS) "was specifically designed to test the validity of [the] hypothesis [of adverse neurodevelopment effects] in a well-nourished population exposed to MeHg *only* from high consumption of unpolluted [by *other* chemicals] ocean fish."[94] The Seychelles Island results are clearly superior for deriving RfD exposure to methylmercury for the U.S. population. This is because the Seychelles study is without toxic confounders and the Seychelles Island mothers consumed about ten times the amount of fish as U.S. mothers[95] – ocean fish containing MeHg concentrations.

This evaluation was confirmed in a post-NRC (2000) analysis. Dourson *et al*.[96] restated 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.***"

Dr. Dourson was a former EPA RfD/Reference Concentration Working Group co-chair.

It is also very important to dispel the running myth that NRC (2000) included or weighted the SCDS results into their recommendation of RfD. That is simply not true.

> **"**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[97] 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."*** [Emphasis added] **(p. 6 of NRC (2000) report, *Toxicological Effects of Methylmercury*)**

The SCDS authors from the University of Rochester School of Medicine continued their important scientific work and concluded in 2003 that:[98]

> "[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."*** [Emphasis added]

Table 2 of Myers et al. (2003) shows the result of the Boston Naming Test from the SCDS for Seychelles children at age 9 that ***cannot confirm*** the prenatal MeHg exposure risk (with $p = 0.79$). As recalled, Boston Naming Test was deemed to be solid conclusion from the Faroe children study (see **Figure 19** in Reply to EPA Claim 6). The EPA NESHAP proposal has yet to incorporate this relatively well-known result from SCDC that was published in 2003.

Constantine Lyketsos of the John Hopkins Hospital offered a professional overview on the implications of the Seychelles study, concluding that:

> "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***. ... 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.*** "[99] [Emphasis added]

In the latest examination and re-analysis by Davidson *et al*. (2008),[100] the SCDC researchers "continue to find no consistent adverse association between MeHg and visual motor coordination" for their Seychelles children at mean age 10.7 years. Huang *et al*.

(2007),[101] in applying ever-more-powerful statistical methods, ***is able to identify a key segregating role for the home environment factor***. For the 53% of the children in an average stimulating home environment group, improved motor proficiency and activity level were found with increasing prenatal MeHg exposure or maternal fish consumption. In contrast, for 7% of the children from below average stimulating home environments, motor proficiency decreased significantly with increased prenatal MeHg exposure – ***thus possibly shifting the negative childhood developmental factor from prenatal MeHg exposure to social conditioning from the home environment.***

This possibility is totally consistent with the new results by Fonseca *et al*. (2008),[102] contrasting two very poor and remote neighborhoods of Brazil with very large contrasts in fish-MeHg exposure risks. The authors conclude that:

> "The high dissimilarity of fish-MeHg exposure between Riparian and Agrarian children cannot explain comparable poor neurodevelopment performance, possibly slightly better academic environment in luna [Agrarian] could account for the better performance of Agrarian children. … ***Global strategies for reducing human exposures to MeHg by curtailing fish consumption are unrealistic options for riverine subsistence populations and are not justifiable to prevent low cognitive scores. … In these isolated communities there are stronger determinants of neurocognitive poor performance than MeHg exposure.***" [Emphasis added]

In describing the conditions of these poor children in Brazil and why they are not neurodevelopmentally healthy, Fonseca *et al*. (2008) explains:

> "According to UNICEF, 35% of the Brazilian population is comprised of school-age children with nearly 50% still living below the poverty line. ***These adverse conditions reflect directly and indirectly in physical and cognitive development of youngsters: reduced stature and first grade failure rates reach 70% in urban-slum communities. The children in our study groups are exposed to intestinal parasites, but the Puruzinho [riparian] sample were additionally exposed to endemic malaria which aggravates anemia because splenic hemolysis is secondary to protozoa-infected red cells.*** Another feature that might contribute to poor performance is the deficit in stimuli due to isolation, which is common to both groups but much more accentuated in the Riparians." [Emphasis added]

It is thus clear from the case of Brazilian children that poverty and diseases are their natural enemies – not any exposure to MeHg from eating fish.

Thurston *et al*. (2007),[103] in an attempt to assess the claim of negative effects of prenatal exposure to MeHg (and, as noted above, to cadmium and other pollutants in pilot whale meat) on blood pressure in Faroese children, could not find consistent signals in the

Seychelles children, whose mothers are mainly exposed to MeHg from ocean fish. The careful Thurston *et al*. (2007) study found:

> "no association between prenatal MeHg and BP [blood pressure] …in girls at either age [12 and 15 years] or in either sex at age 12 years. At age 15, diastolic BP in boys increased with increasing prenatal MeHg exposure, while systolic BP was unaffected."

The authors concluded that this one detected signal out of eight tests could be a chance finding with unclear biological significance.

Highlights of Seychelles children study

- Ocean fish uncontaminated by non-mercury compounds are sole source of exposure – no sea mammals in diet or fresh water fish in diets.
- Measured levels of PCBs were undetectable in the Seychelles.
- The ocean fish consumed have mercury levels similar to commercial fish sold in the U.S.
- The cohort had extensive evaluations at 6, 19, 29, 66, 107 months and 10.7 years of age.
- There have been no consistent adverse associations with mercury exposure present.
- Mothers in the study consumed fish 12 times per week.
- Prenatal exposure averaged 6.9 ppm (1-27 ppm) in hair mercury or about 40 ppb in blood mercury. (Note that EPA's RfD is 5.8 ppb for human blood levels.)
- The study has been conducted double blind for nearly 20 years, with no clinical investigators or anyone in the Seychelles knowing the mercury exposures.
- The study provided *no support* for an adverse association between child neurodevelopment and prenatal exposure to MeHg from maternal consumption of ocean fish at the levels being studied (5.8 – 156 ppb).[104]

Thus, at best, the Faroe Islands studies are useful for understanding a mixed chemical exposure, especially for PCB. The Seychelles Islands studies are very good at revealing and understanding exposures to MeHg. ***Furthermore, as illustrated in the figure under endnote #92, Kaneko and Ralston (2007)[105] showed that the quantitative Selenium Health Benefit Values for 4 out of 16 species of fish consumed by Seychelles population are above 400 compared to the negative values for pilot whales consumed by the Faroe Island population.*** Thus, as emphasized by Ralston et al. (2007)[106] and Ralston and Raymond (2010)[107], selenium content in fish could also be a very important and decisive factor for deciding MeHg exposure risks.

In the interest of American public health, based on more recent and robust science, ***EPA needs to reconsider its mercury RfD – and reset it upward.***

EPA Claim 8: Studies published since the current MeHg RfD was released include new analyses of children's neuropsychological effects from the existing Seychelles and Faroe Islands cohorts, including formation of a new cohort in the Faroe Islands study. There are also a number of new studies that were conducted in population-based cohorts in the U.S and other countries. **A comprehensive assessment of the new literature has not been completed by EPA. However, data published since 2001 are generally consistent with those of the earlier studies that were the basis of the RfD, demonstrating persistent effects in the Faroe Island cohort, and in some cases associations of effects with lower MeHg exposure concentrations than in the Faroes.** These new studies provide additional confidence that exposures above the RfD are contributing to risk of adverse effects, and that reductions in exposures above the RfD can lead to incremental reductions in risk. (pp. 144-145)[Emphasis added]

Reply to EPA Claim 8: First, if one clearly distinguishes the prenatal and postnatal exposure risk and outcomes, then this EPA Claim is truly suspicious and negligent, since it has been known at least since 2006, from the research team of the Faroe Island children study, that "***postnatal methylmercury exposure had no discernible effect.***"[108]

Second, to show one simple contradiction to the claim in **EPA Claim 8** above, one simply has to look at the latest paper from the Faroe children study group, "Prenatal exposure to lead and cognitive deficit in 7- and 14-year old children in the presence of concomitant exposure to similar molar concentration of methylmercury" by Yorifuji *et al*. (2010):[109]

> "This study examined the effects of prenatal lead exposure on cognitive deficits in the presence of a similar molar concentration of a neurotoxic co-pollutant (methylmercury) in 7- and 14-year-olds born in the Faroe Islands. The analyses of the total cohort and those of cohort members without interaction terms among lower co-pollutant category showed equivocal results. However, when the subjects were restricted to a lower co-pollutant category, and statistical interaction terms were entered within the category, adverse effects of prenatal lead exposure on cognitive functions in childhood were observed, especially on attention, learning and memory. ***When the total cohort was considered, lead exposure seemed to have beneficial effects on language (Boston Naming Test) and learning (in CVLT-C [or California Verbal Learning Test-Children's version]) at age 7 despite adjustments for mercury. Furthermore, even when we entered multiplicative terms between mercury and lead, positive associations did not change and a positive association between lead and long-term recall in CVLT-C was observed.*** In contrast, a negative effect on digit span in WISD-R at age 7 was noted. These findings showed that, in the presence of the co-pollutant which varied considerably (and measured with a certain degree of imprecision), adjustment for it or entering and interaction term resulted in equivocal findings. … The

present study indicates that adverse effects of exposure may be overlooked if the effects of a co-pollutant are ignored." [Emphasis added]

This new result and conclusion clearly has implications for the discussion of Boston Naming Test results in **EPA Claim 6** above – and EPA would have **to evaluate the co-interactions of exposures of the Faroe children to MeHg, lead and PCBs** much more carefully, before insisting that the Faroe study is appropriate or necessary for American children as well.

Third, several of the updated studies, especially those from the Seychelles SCDS, have been recognized as the most appropriate and relevant epidemiology study for MeHg exposure risk that is similar to the American public's, and even to the most sensitive groups of women of child-bearing age and young children one to five years old. These studies clearly concluded that ***MeHg exposure risk is a completely manageable risk, with no specific crisis that would impel EPA to set forth the NESHAP rules***.

Davidson *et al*. (2010)[110] of the SCDC group reported the first results for scholastic achievement scores in associations with both prenatal and postnatal MeHg exposures:

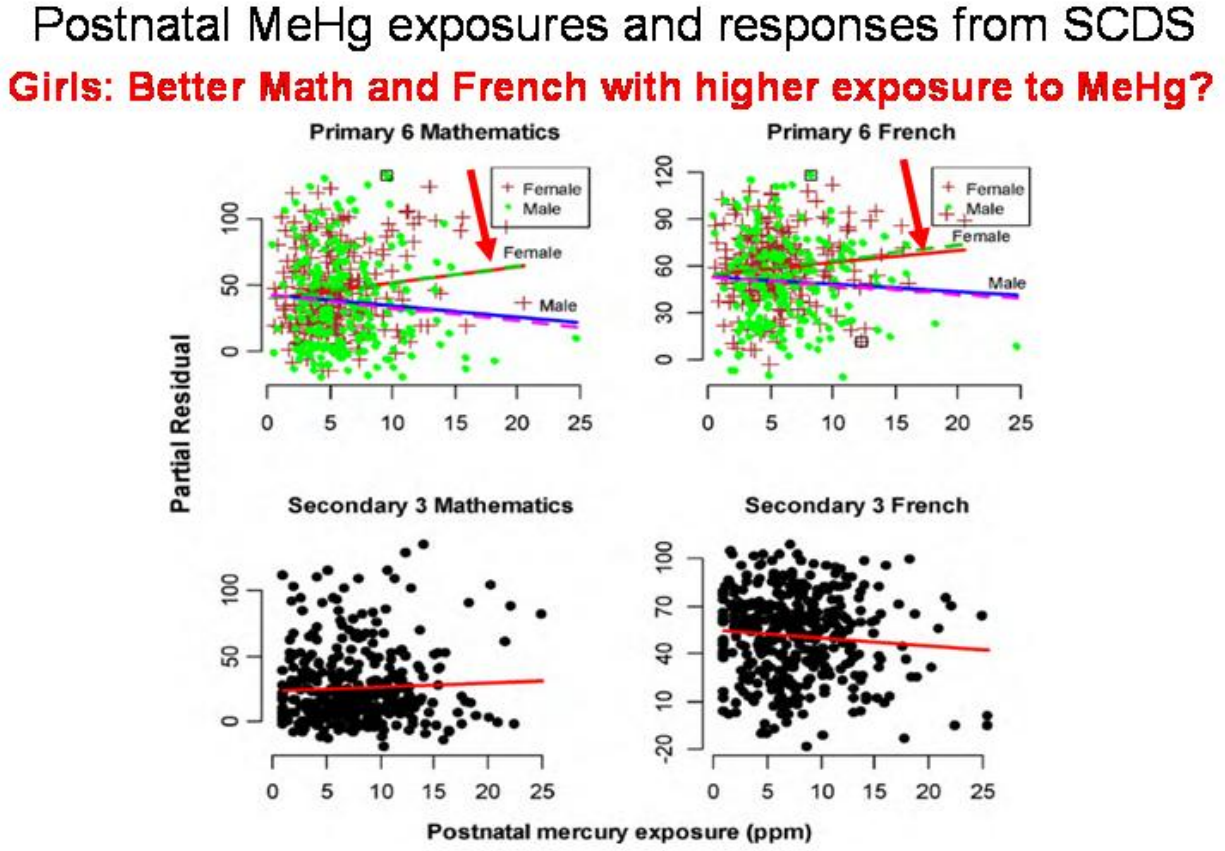
> "Our results for Primary 6 and Secondary 3 end-of-year examinations indicated no consistent pattern of association between prenatal or recent postnatal MeHg hair levels and any outcome, either at the end of primary school or in mid-high school. In all, we studied 12 end-of-year examination scores, six at the end of Primary 6 and six at the end of Secondary 3. We found one association between prenatal MeHg exposure and 11 out of 12 achievement scores, and no association between recent postnatal exposure and 11 out 12 examination scores. At the end of Primary 6, we found a significant negative association between prenatal exposure and achievement in French and another significant negative association between postnatal exposure and Social Studies achievement. The findings may not have statistical or biological meaningfulness, given the lack of association in these content areas at other time points, and may have resulted by chance alone. … ***These results are consistent with our earlier studies and support the interpretation that prenatal MeHg exposure, at dosages achieved by mothers consuming a diet high in fish, are not associated with adverse educational measures of scholastic achievement***." [Emphasis added]

In a simple and straightforward way, the SCDS scientists have systematically searched for any statistically significant effects of MeHg exposure on children cognitive and other essential functions and behaviors for close to two decades. And yet they are still unable to confirm any harmful effects of MeHg exposures through fish consumption. The review study of Myers *et al*. (2009)[111] clearly reached similar conclusions. It is important for EPA to include this gold-standard of epidemiological study for children's health in order for its proposed emission rules to be scientifically credible.

Figure 20 shows the summary result from Davidson *et al*. (2010), showing both the negative (for male) and positive (for female) associations between postnatal MeHg exposure levels with school test scores. The highlighted positive associations in **Figure 20** for female students also clearly made another important point: namely, beneficial effects from eating fish can outweigh the relatively tolerable MeHg exposure risks. The first result from the new Seychelles Child Development Nutrition Study as reported by Davidson *et al*. (2008)[112] and Strain *et al*. (2008)[113] clearly support this interpretation. In fact, Strain *et al*. (2008) specifically found that:

> "These data support the potential importance to child development of prenatal availability of Ω-3 LCPUFA [Long-Chain PolyUnsaturated Fatty Acids] present in fish and of LCPUFA in the overall diet. Furthermore, they indicate that the beneficial effects of LCUPUFA can obscure the determination of adverse effects of prenatal MeHg exposure in longitudinal observational studies."

Figure 20



Davidson et al. (2010) Neurotoxicology, vol. 31, 439-447

Finally, the latest study from the ALSPAC (Avon Longitudinal Study of Parents and Children) cohort reported by Hibbeln *et al*. (2007)[114] strongly emphasized that all the concerns, scares and fears on MeHg in fish is not totally innocent. Quite the opposite: they will have actual negative impacts on the most sensitive population of our American society:

> "Maternal seafood consumption of less than 340 g per week in pregnancy did not protect children from adverse outcomes; rather, we recorded beneficial effects on child development with maternal seafood intakes of more than 340 g per week, ***suggesting that advice to limit seafood consumption could actually be detrimental.*** These results show that risks from the loss of nutrients were greater than the risks of harm from exposure to trace contaminants in 340 g seafood eaten weekly." [Emphasis added]

This is why WS continues to urge EPA to re-consider American public health and stop unnecessarily scaring pregnant women and young children, especially those from low-income families, by issuing fish consumption limits and attention-grabbing bad news on how MeHg will supposedly affect children's mental health.

EPA Claim 9: Deposition of Hg for the continental U.S. was estimated using the Community Multiscale Air Quality model v4.7.1 (www.cmaq-model.org), applied at a 12 km grid resolution. The CMAQ modeling was used to estimate total annual Hg deposition from U.S. and non-U.S. anthropogenic and natural sources over each watershed. In addition, CMAQ simulations were conducted where U.S. EGU Hg emissions were set to zero to determine the contribution of U.S. EGU Hg emissions to total Hg deposition. U.S. EGU-related Hg deposition characterized at the watershed-level for 2005 and 2016 is summarized in Table 6 of this preamble for the complete set of 88,000 HUC12 watersheds. (pp. 184-185)

TABLE 6. COMPARISON OF TOTAL AND U.S. EGU-ATTRIBUTABLE HG DEPOSITION (μg/m^2) FOR THE 2005 AND 2016 SCENARIOS.*

Statistic	2005		2016 **	
	Total Hg Deposition	U.S. EGU-attributable Hg Deposition	Total Hg Deposition	U.S. EGU-attributable Hg Deposition
Mean	19.41	0.89	18.66	0.34
Median	17.25	0.24	16.59	0.15
75th percentile	23.69	1.07	22.83	0.46
90th percentile	30.78	2.38	29.90	0.85
95th percentile	36.85	3.60	35.16	1.18
99th percentile	58.32	7.77	56.23	2.41

* Statistics are based on CMAQ results interpolated to the watershed -level and are calculated using all ~88,000

Reply to EPA Claim 9: WS wishes to respond to **the highly exaggerated values of total atmospheric Hg deposition in 2005 from a mean of 19 μg/m^2 to the 99th percentile value of 58 μg/m^2.** Those values were assumed in this modeling exercise of EPA's NESHAP proposed rules, using available measurements (shown in **Figure 21 for 2005**) for the distinct geographical pattern and amount of atmospheric mercury deposition, based on monitoring efforts by the Mercury Deposition Networks (MDN) operated by the Illinois State Water Survey. Typical measured values are about 5 to 12 μg/m^2 over broad region of the central and northeast U.S. while the highest value in the southeast U.S. is around 22 μg/m^2—far below the 90th to 99th percentile values assumed by EPA for 2005. Updating the measured mercury deposition to 2009 (**Figure 22**), which includes wider spatial coverage, double confirmed WS's challenge to the high mercury deposition values assumed by EPA in its risk-benefit modeling.

Recently, Prestbo and Gay (2009)[115] reported the results from MDN for wet deposition in the U.S. and Canada from 1996-2005 and reached the following conclusions:

> "This study provides analysis and interpretation of MDN observations at 10 years (1996-2005) with an emphasis on investigating whether rigorous, statistically-significant temporal trends and spatial patterns were present and where they occurred. Wet deposition of mercury ranges from more than 25 μgm^{-2} yr south Florida to less than 3 μgm^{-2} yr in northern California. Volume-weighted total mercury concentrations are statistically different between defined regions overall (Southeast ≈ Midwest > Ohio River > Northeast), with the highest in Florida, Minnesota and several Southwest locations (10-16 ng/L). ***Total mercury wet-deposition is significantly different between defined regions (Southeast > Ohio River > Midwest > Northeast).*** Mercury deposition is strongly seasonal in eastern North America. ***The average mercury concentration is about two times higher in summer than in winter.*** Forty-eight sites with validated datasets of five years or more were tested for trends using the non-parametric seasonal Kendall trend test. ***Significant decreasing mercury wet-deposition concentration trends were found at about half of the sites, particularly across Pennsylvania and extending up through the Northeast.*** " [Emphasis added]

Figure 21

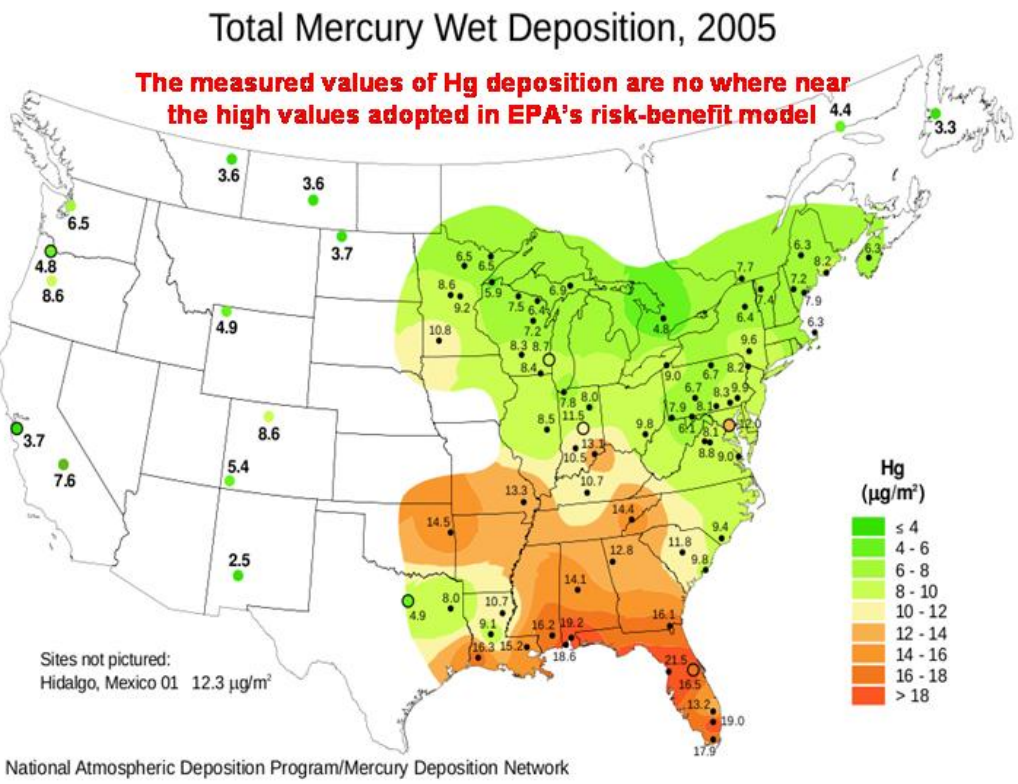
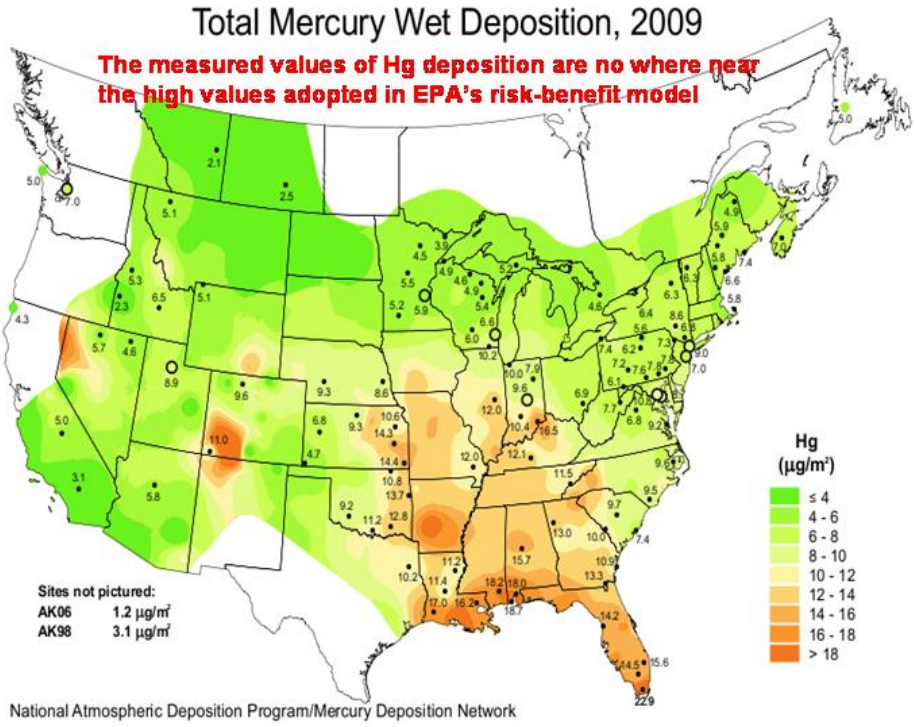


Figure 22



EPA Claim 10: Table 8 of this preamble compares total and U.S. EGU-attributable fish tissue MeHg concentrations for the 2005 and 2016 scenarios by watershed percentile. (p. 188)

TABLE 8. COMPARISON OF TOTAL AND U.S. EGU-ATTRIBUTABLE FISH TISSUE MEHG CONCENTRATIONS FOR 2005 AND 2016

Statistic	Fish tissue MeHg concentration (ppm)			
	2005		2016	
	Total	U.S. EGU-attributable	Total	U.S. EGU-attributable
Mean	0.31	0.024	0.29	0.008
50th Percentile	0.23	0.014	0.20	0.005
75th Percentile	0.39	0.032	0.36	0.011
90th Percentile	0.67	0.056	0.63	0.019
95th Percentile	0.91	0.079	0.87	0.026
99th Percentile	1.34	0.150	1.29	0.047

Reply to EPA Claim 10: The results tabulated in Table 8 of EPA's NESHAP proposal, summarizing a risk-benefit analysis performed by EPA, are truly remarkable – reflecting agency claims that it is able to track the amount of MeHg attributable to U.S. EGUs mercury emissions. The assertion is in addition to the NESHAP team's exaggerating the amount of atmospheric mercury deposition (noted in **EPA Claim 9** above). When one examines the analysis behind this **EPA Claim 10** – and in the context of the complex processes involved in mercury cycling and recycling, and the numerous biological, chemical and physical variables and factors involved in biomethylation and bioaccumulation – it becomes obvious that there is simply no scientific foundation for EPA's claims or this Table 8. All readers should realize that EPA's claim of being able to connect U.S. EGU mercury emissions to the tiny amounts of MeHg in fish tissues is nearly impossible and cannot be verified in the real world. This can only be done in a computer model that cannot be validated against the real-world data.

WS presents two examples of available fish mercury data, challenging the premise that anthropogenic industrial emissions and deposition of Hg to air and water has led to heightened levels of MeHg in fish over time, as incorrectly implied in EPA's proposed NESHAP emission rules.

Available evidence strongly suggests that: ***(1) MeHg has always been present in fish; (2) current MeHg levels in fish vary naturally over time; and (3) the natural production (and destruction) of MeHg is not limited by the amount of Hg available in aquatic systems.*** Hence, claims directly connecting fish mercury levels to minor[116] U.S. power plant Hg emissions confirm a serious misunderstanding of both real-world observations and scientific evidence.

WS first poses this question. Are the observed values of mercury, ranging from 23 ppb to 6605 ppb in fish tissues throughout a recent four-year study, according to the latest EPA fish report "The National Study of Chemical Residues in Lake Fish Tissue" (2009),[117] unusually high or exceptional?

Considering that the 95th percentile value for predator fish composites from this EPA report is only 833 ppb, one can safely assume that the maximum value is merely a one-time statistical outlier. **Figure 23** shows the full range of the mercury values for the standardized length of major predator fish, northern pike and walleye, from lakes in Minnesota, aggregated for the state as a whole and for the Northern Lakes and Forest Region from 1982 to 2006.[118] The values fluctuated between 20 ppb to 1800 ppb (i.e., inverting from the natural log Hg values of 3 and 7.5 on the vertical axis on **Figure 23**). These values are not drastically different from the fish mercury values presented in the 2009 EPA fish mercury report.

Figure 23

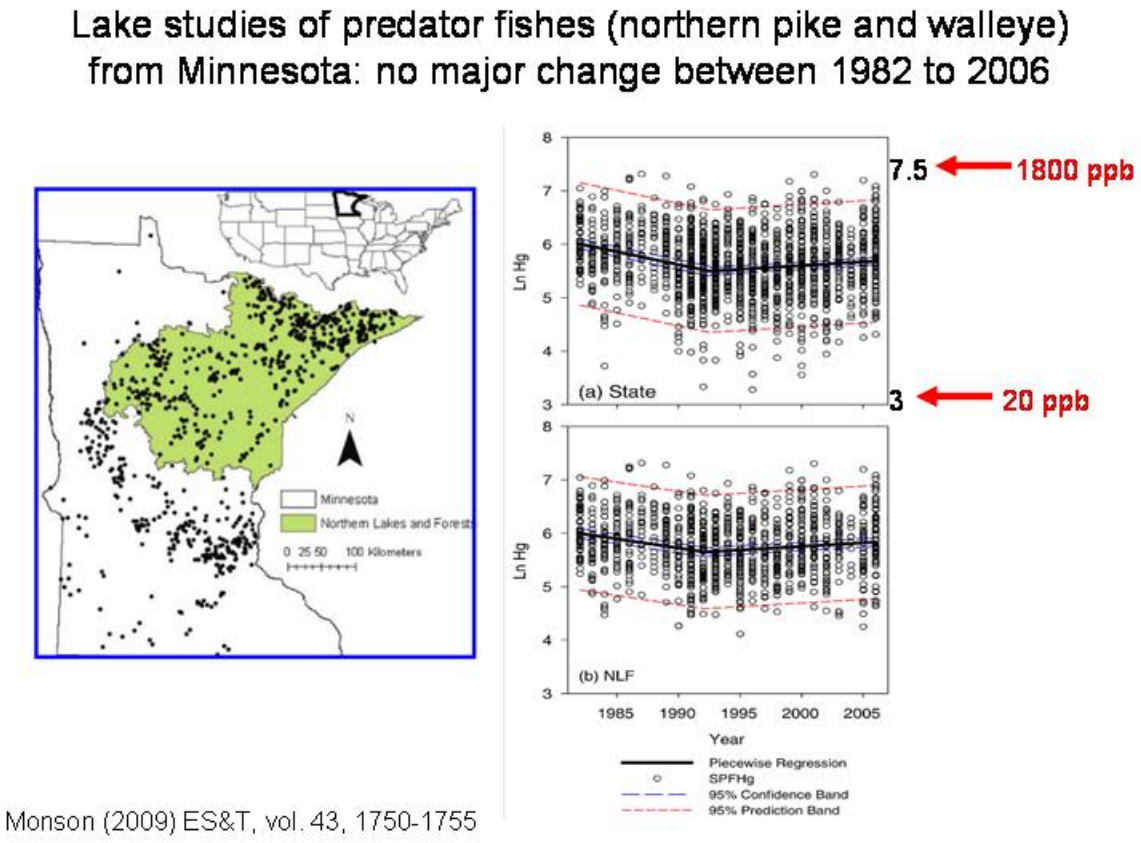


Figure 24 presents further evidence ***against*** EPA's theory that there has been an increasing trend in MeHg fish levels, by examining concentrations in the tissue of striped bass from the San Francisco Bay area over the period 1970-2000.[119] Those results show that, in any given year, there is at least one striped bass sample containing mercury values above the arbitrary 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 any power plant or industrial Hg emissions.***

Figure 24

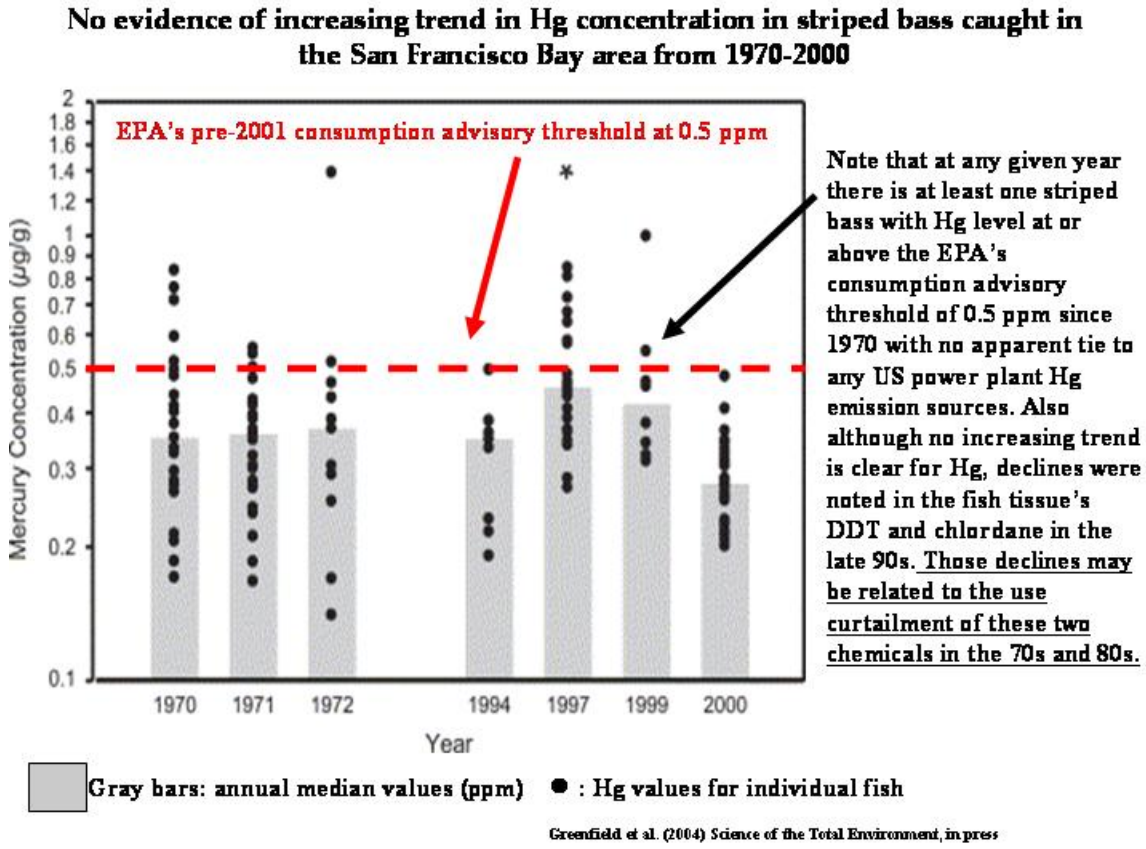


Figure 24 reveals another important finding. Even though no accumulation trend was noted for mercury in striped bass in the 1970-2000 intervals, significant declines in the late 1990s were noted for other contaminants like DDT and chlordane in San Francisco Bay fish tissues. The authors suggest that these 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 methylation and bioaccumulation of mercury in fish than supposed in EPA's current computer modeling efforts. That is, compared to other contaminants, the pathway and behavior of mercury transformation and accumulation in fish appears to differ significantly.

A similar tendency was also reported[120] for levels of contaminants in fish from the upper River Thames in Britain, according to zoologists from Oxford University and Cornell University. These authors concluded that, although the recent decrease in PCB contamination levels may be partly associated with industrial and human activities, ***it was difficult to find such associations for mercury.***

Figure 25 presents recently published fish mercury data sets for various sport fish species (yellow perch, walleye pike, smallmouth bass, northern pike, white sucker and carp)

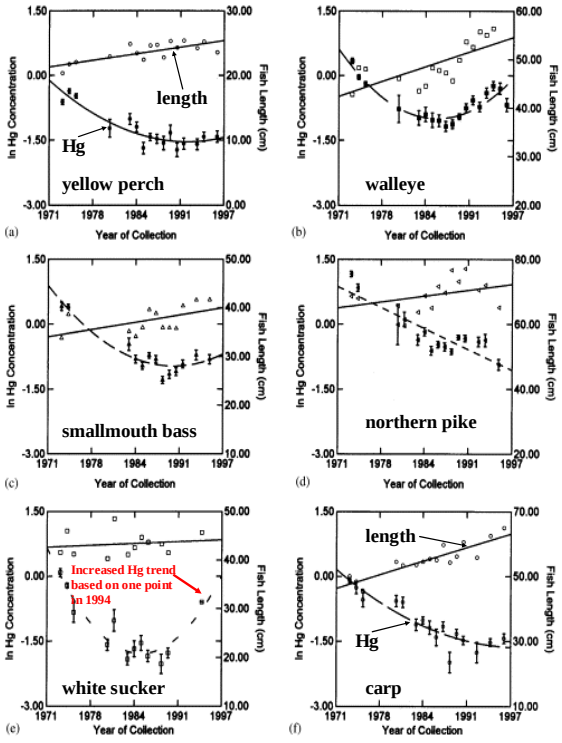
caught from 17 "areas of concern for mercury contamination" (and 10 other toxic substances) in the **Canadian Great Lakes** from 1971 to 1997. Once again, the results provide evidence that **historical changes in mercury concentrations *are not* simply to be expected from local industrial Hg emissions**. In fact, the author concluded that:[121]

> "Differences observed [among various areas of concern] ***did not*** consistently parallel expectations associated with the historical presence of chlor-alkali plants in the vicinities of some locations." [Emphasis added]

Equally important, 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."*** This fact illustrates how difficult it is to confirm various popular claims that trace amounts of MeHg cause serious childhood neurological health complications.

Figure 25

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

Figure 26 reports another important recent finding[122] that was neglected by the current EPA NESHAP proposed emission rules or even the 2009 EPA fish report, despite the fact that the study is partly funded by EPA:

"Newly mandated reductions in anthropogenic mercury emissions aim to reduce atmospheric mercury deposition and thus mercury concentrations in fish. However, factors other than mercury deposition are important for mercury bioaccumulation in fish. ***In the lakes of Isle Royale, U.S.A., reduced rates of sulfate deposition since the Clean Air Act of 1970 have caused mercury concentrations in fish to decline to levels that are safe for human consumption, even without a discernable decrease in mercury deposition.***" [Emphasis added]

EPA essentially failed to weigh-in the potential key role of decreasing sulfur deposition on the recent decrease of mercury content in the predatory fish, northern pike, caught between 2004-2006 (see **Figure 26**), relative to the high levels in 1995-1996 for eight lakes at Isle Royale. **Figure 27** simply documented the fact that ***such a decrease in fish mercury for 2004-2006 in those 7 of 8 lakes occurred despite continuing accumulation of Hg in the sediments and increasing atmospheric mercury deposition.***

Figure 26

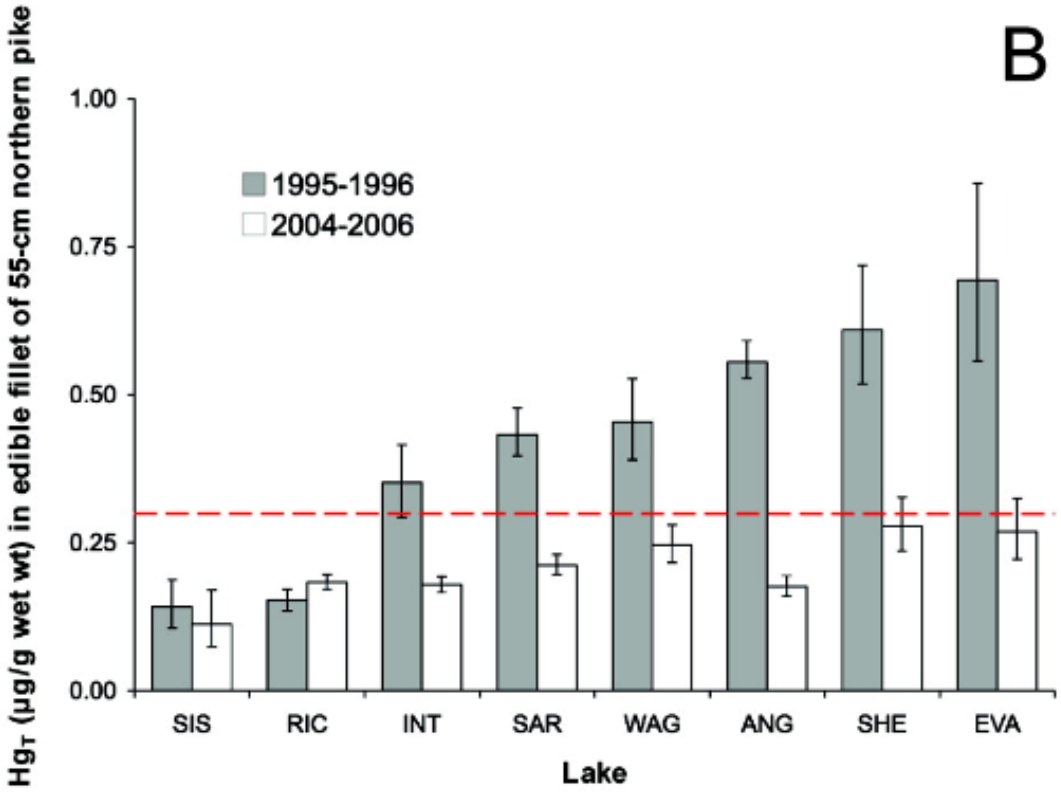
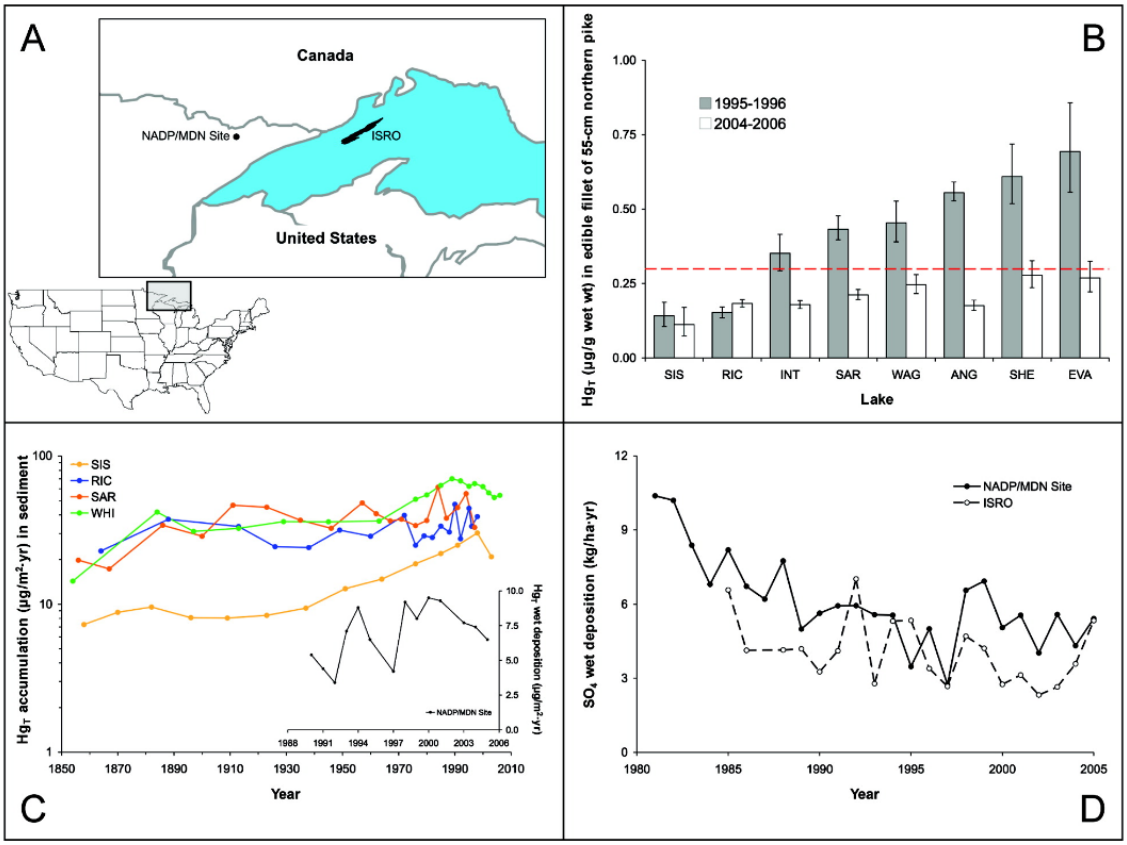


Figure 27



It is indisputable that fish is a nutritious, highly abundant resource for maintaining overall health for many, especially those within subsistence cultures. Fish is known to be rich in essential high-quality protein, omega-3 polyunsaturated fatty acids, sulfur-containing amino acids, vitamin E, selenium, lysine, iodine, copper, calcium, zinc, iron, manganese and other nutrients. Existing medical evidence from both clinical and epidemiological settings suggest that the trace levels of MeHg in our fish are not likely to suddenly overwhelm well-established nutritional benefits derived from consuming a variety of fish found in restaurants, grocery stores and most local waters.

This fact raises other important questions. How relevant is the 2009 EPA's new lake fish tissue report for the wider public that does not consume any fish caught from lakes or rivers? How do the beneficial effects of selenium (Se), which are so well-known for ocean fish, figure into this analysis? More importantly have the March 16, 20011's EPA NESHAP proposed new emission rules taken these key science questions and facts into account before reaching a decision?

Many studies have suggested that proportionally higher selenium levels in consumed ocean fish may counteract the toxicity of fish tissue MeHg. Swordfish for example, while

higher in MeHg, is a superior source for omega-3[123] fatty acids and selenium.[124] A survey of 11 commercial sea foods in New Jersey confirmed that selenium concentrations exceeded those of mercury; for some species the selenium-to-mercury ratio was as high as 23.[125] An analysis of 39 types of commonly consumed fish and shellfish in Modena, Italy by Plessi *et al*. (2001) also confirmed "a large excess of selenium in relation to mercury."[126]

Both fish selenium and amino acids are well-documented for helping to reduce toxicity, by inhibiting entry/transport of MeHg into brain cells. This critical finding may explain why Minamata-like poisonings have not been diagnosed for chronic MeHg-exposures through the large daily intake of fish not directly contaminated with multiple chemicals.[127]

Is there any evidence for relatively high concentration of Se in lake fish?

The study by Peterson *et al*. (2009),[128] which was said to be "funded wholly (or in part) by the U.S. Environmental Protection Agency, as part of the Environmental Monitoring and Assessment Program," provides just such an answer. Based on a new survey of stream fish of the Western U.S. (see **Figure 28**), Peterson *et al*. (2009) explained that:

> "The ability of selenium (Se) to moderate mercury (Hg) toxicity is well established in the literature. Mercury exposures that might otherwise produce toxic effects are counteracted by Se, particularly when Se:Hg molar ratios approach or exceed 1. We analyzed whole body Se and Hg concentrations in 468 fish representing 40 species from 137 sites across 12 western U.S. states. … 97.5% of the total fish sample contained more Se than Hg (molar ratio >1) leaving only 2.5% with Se:Hg ratios < 1. … ***Scientific literature on Se counteracting Hg toxicity and our finding that 97.5% of the freshwater fish in our survey have sufficient Se to potentially protect them and their consumers against Hg toxicity suggests that Se in fish tissue (Se:Hg molar ratio) must be considered when assessing the potential toxic effects of Hg.***" [Emphasis added]

Figure 29 confirms the profound result regarding 97.5% of the fish sample having Se:Hg ratios above 1, and thus potentially opening a deeper understanding of the Se protective mechanism. ***EPA's NESHAP proposal might have benefited from including the Peterson et al. (2009) paper and examining this protective mechanism for a comprehensive review and the ultimate protection of the American public health.***

Figure 28

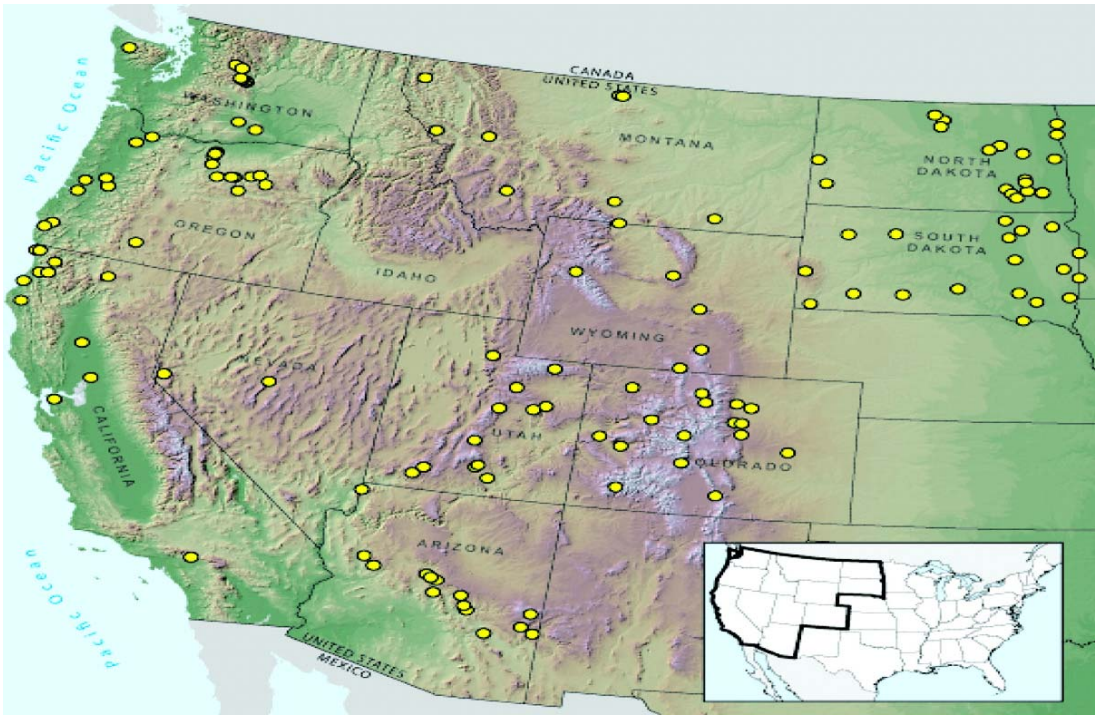
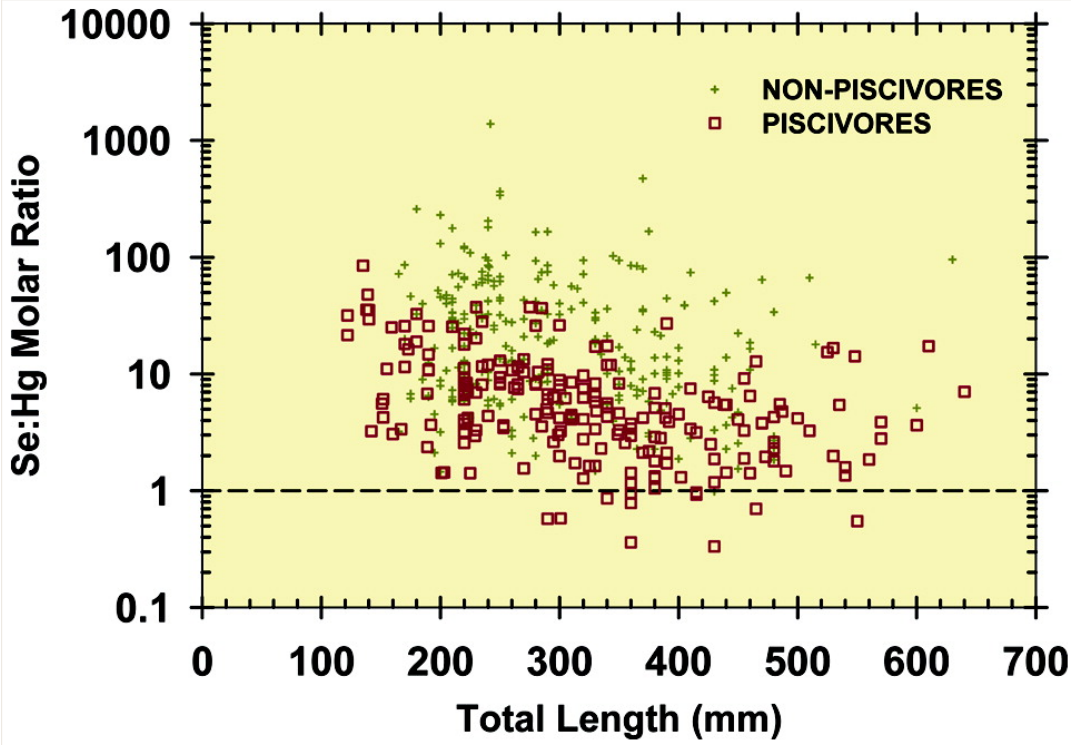


Figure 29



Ralston and Raymond (2010)[129] offers deeper medical explanations and mechanisms for the protective effects of Se against exposure risk to MeHg:

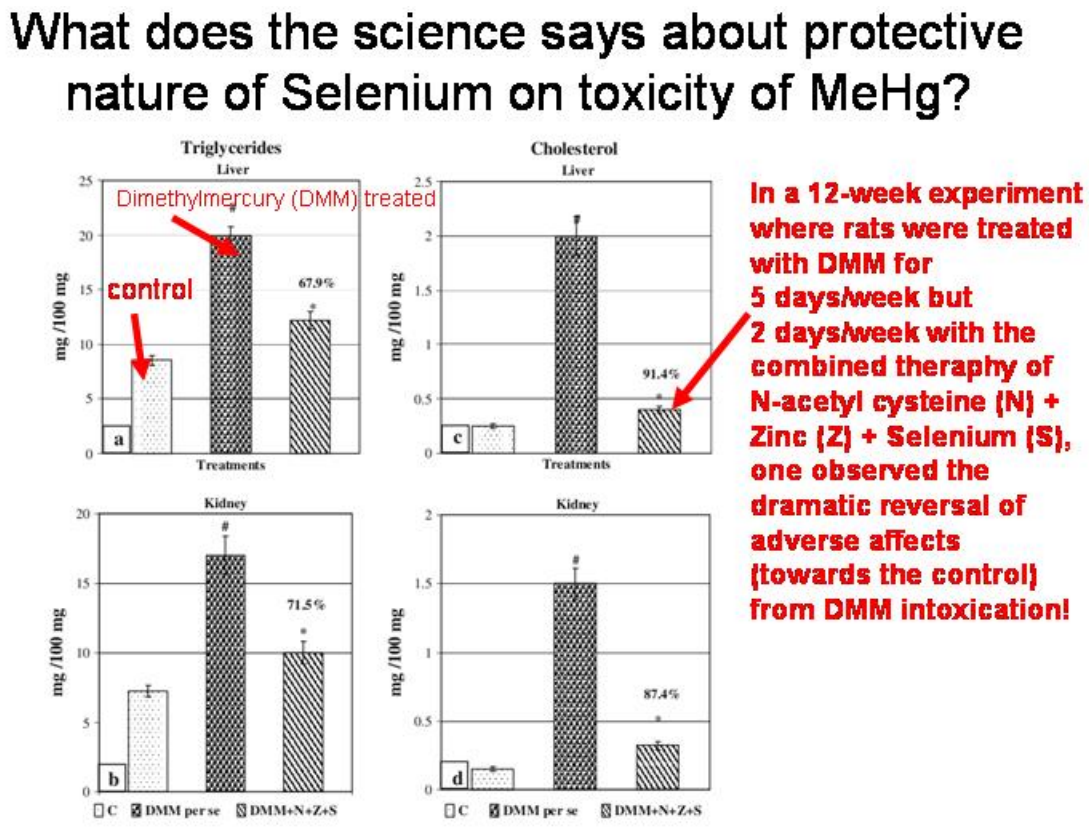
> Dietary selenium (Se) status is inversely related to vulnerability to methylmercury toxicity. Mercury exposures that are uniformly neurotoxic and lethal among animals fed low dietary Se are far less serious among those with normal Se intakes and are without observable consequences in those fed Se-enriched diets. Although these effects have been known since 1967, they have only lately become well understood. Recent studies have shown that Se-enriched diets not only prevent MeHg toxicity, but can also rapidly reverse some of its most severe symptoms. It is now understood that MeHg is a highly specific, irreversible inhibitor of Se-dependent enzymes (selenoenzymes). Selenoenzymes are required to prevent and reverse oxidative damage throughout the body, particularly in the brain and neuroendocrine tissues. Inhibition of selenoenzyme activities in these vulnerable tissues appears to be the proximal cause of the pathological effects known to accompany MeHg toxicity. Because Hg's binding affinities for Se are up to a million times higher than for sulfur, its second-best binding partner, MeHg inexorably sequesters Se, directly impairing selenoenzyme activities and their synthesis. ***This may explain why studies of maternal populations exposed to foods that contain Hg in molar excess of Se, such as shark or pilot whale meats, have found adverse child outcomes, but studies of populations exposed to MeHg by eating Se-rich ocean fish observe improved child IQs instead of harm.*** However, since the Se contents of freshwater fish are dependent on local soil Se status, fish with high MeHg from regions with poor Se availability may be cause for concern. Further studies of these relationships are needed to assist regulatory agencies in protecting and improving child health. [Emphasis added]

The powerful effects from selenium (Se), along with N-acetyl cysteine (NAC) and zinc (Zn), are recently documented in the biochemical and histopathological experiments reported in Joshi et al. (2011)[130]:

> "The present work was aimed to study the therapeutic potential of combined administration of N-acetyle cysteine, zinc, and selenium against dimethymercury (DMM)-intoxicated male rats for 12 weeks. … [DMM] is an organomercury. It is one of the strongest known neurotoxins that affects the immune system; it alters the genetic and enzyme systems and damages the nervous systems, including coordination and the senses of touch, taste and sight. It is adversely affects physiological, biochemical, and behavioral functions in humans and animals. … NAC, along with Zn and Se, dramatically reversed the alterations of all of the variables more toward control. The study results conclude that protective intervention of combined treatment of NAC, along with Zn and Se, is beneficial in attenuating DMM-induced systemic toxicity."

Figure 30 shows the significant increases in the levels of triglycerides and cholesterol in liver and kidney tissues of rats fed with DMM 5 days per week for the 12-week experiment above the control. However, when rats that were fed with same levels of DMM 5 days per week were also fed with 2 days per week of NAC, Zn and Se, the negative-health levels of triglycerides and cholesterol were dramatically improved toward the better level in the control experiments.

Figure 30



Joshi et al. (2011) Archives of Environmental Contamination and Toxicology, in press (accepted February 17, 2011)

WS concludes that with multiple lines of new evidence supporting the role Se, the science result effectively nullifies any concerns regarding the potential for serious health harm from consuming even the freshwater fish in the lower 48 U.S. states, contrary to the alleged health risks in EPA's September 2009 fish report and other literature.

EPA Claim 11: A recent case control study of Chinese children in Hong Kong (Cheuk and Wong, 2006) paired 59 normal controls with 52 children (younger than 18 years) diagnosed with attention deficit/hyperactivity disorder (ADHD). The authors reported a significant difference in blood Hg levels between cases and controls (geometric mean 18.2 nmol/L (95 percent confidence interval, CI, 15.4 - 21.5 nmol/L] vs. 11.6 nmol/L [95 percent CI 9.9 - 13.7 nmol/L], p < 0.001), which persisted after they adjusted for age, gender and parental occupational status (p less than 0.001). (p. 572)

Reply to EPA Claim 11: EPA's treatment of this paper by Cheuk and Wong (2006)[131] in the agency's NESHAP proposal fails in a very simple manner: it hides another important conclusion of the paper from unsuspecting audiences. EPA is clearly trying to insinuate that there is evidence for connecting blood Hg levels to the complex biological response of Attention Deficit Hyperactivity Disorder. Unfortunately, the reality is that this very small-sample (n=52) in a study of Hong Kong children makes it much more complicated and difficult to discern and confirm any blood Hg:ADHD link than EPA suggests in its report.

First, it was clear that the small sample of children is an important issue. Another study of blood mercury levels in Hong Kong children, published by Ip *et al*. (2004),[132] evaluated a significantly larger sample size of 137 children. Their mean blood mercury was 17.6 nmol/L, quite similar to the level found in the group of ADHD children in Cheuk and Wong (2006) – and about 10 times higher than found in American children. Yet, the prevalence of ADHD diagnosis for U.S. children is certainly not diminishingly small when compared to Hong Kong children if mercury levels were to be any important parameter for ADHD.

Second, it is important to note that the EPA proposal failed to account for this interesting and equally important conclusion reached by Cheuk and Wong (2006):

> "We found that children whose fathers were office or service workers had a higher risk of ADHD. This might indicate a socio-economic gradient in the risk of ADHD, or represent an indirect occupational exposure to noxious agents, and warrants further evaluation in future studies."

EPA Claim 12: Studies in two cohorts (the Kuopio Ischemic Heart Disease Risk Factor study, or KIHD study; and the European Community Multicenter Study on Antioxidants, Myocardial Infarction and Breast Cancer, or EURAMIC study), report statistically significant positive associations between MeHg exposure and AMI. … Although each of these AMI studies had strengths and limitations, the EURAMIC and KIHD studies appear to be most robust. Strengths of these two studies include their large sample sizes and control for key potential confounders(such as exposure to omega-3 fatty acid, which are related to decreases in cardiovascular effects). The KIHD study was well-designed and included a population-based recruitment and limited loss to follow-up. Additional strengths of the EURAMIC study include exposure data that were collected shortly after the AMI. In addition, recruitment of participants across nine countries likely resulted in a wide range of MeHg and fish fatty acid intakes. (pp. 575-576)

Reply to EPA Claim 12: This EPA claim clearly suggests there is evidence that trace amounts of "mercury" in fish could overwhelm the positive effects of Omega-3 fatty acids, causing cardiovascular disease (CVD), coronary heart disease (CHD) and even death in adults.

However, the so-called "evidence" appears to be based on two highly suspect studies.

The first is the Kuopio Ischaemic Heart Disease (KIHD) Risk Factor Study (Salonen *et al*. 1995, 2000) of a group of men in eastern Finland; it suggested a statistical relationship linking mercury with risk of CHD and CVD. The second is a case-control study of 684 men from eight European countries and Israel (Guallar et al. 2002); it concluded, "High mercury content may diminish the cardioprotective effect of fish intake."

However, other medical literature strongly suggests that the claimed role of methylmercury in negatively impacting human health, including the heart, is neither clear nor direct. Indeed, the asserted role of MeHg is often contradicted by published data. Instead, ***the available evidence suggests that numerous risk factors other than methylmercury from fish*** more likely explain most of the findings in Salonen *et al*. (1995, 2000) and Guallar *et al*. (2002). In addition, as with the troubled Faeroe Island data on neurodevelopment of children, there are reasonable concerns *that* ***the Finnish results are not directly applicable for the US population.***

At the same time, the American Heart Association has reviewed the benefits and risks of regular consumption of fish and fish oils. The review ***concludes that fish and fish oils help prevent cardiovascular disease including fatal and non-fatal heart attacks, strokes, sudden cardiac death, and coronary artery disease (angina).***

Finally, media-fed alarmism (and lack of critical analysis and reporting) over mercury may be causing a dangerous decline in the already inadequate levels of U.S. fish consumption. It is no wonder that public health professionals are beginning to express concern, including new worries that harm to public health may not be limited to

nutritional deficits,[133] but may extend to psychiatric effects of the inflated (mercury) fear itself (Ropeik, 2004),[134] promoted by many interest groups and the EPA.

Alan Stern of the New Jersey Department of Environmental Protection and a member of the NRC (2000) MeHg committee recently revealed in Stern (2005)[135] that:

> "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. ... ***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)." [Emphasis added]

Professor Thomas W. Clarkson from the University of Rochester, the world's leading authority on the human health effects of mercury and methylmercury, commented partly in response to claims like those in Stern (2005) that:[136]

> "***If these [cardiac-related] findings are confirmed, two long-held dogmas may have to be abandoned, namely, that methylmercury primarily affects the central nervous system and that the prenatal period is the most susceptible part of the life cycle.***"

In light of the very serious implications for both human health and the actual science of direct methylmercury toxicity, the following literature reviews and critical analyses are offered for a clarification of the strengths and weaknesses of the "new" claims linking MeHg and cardiovascular health.

First and foremost, the latest examination of two very large U.S. Cohorts with a total of 51,529 men (the Health Professionals Follow-up Study) and 121,170 women (the Nurses' Health Study) for risk of cardiovascular disease in response to mercury exposure by Mozaffarian et al. (2011)[137] brought important health conclusion on March 24, 2011:

"***We found no evidence of any clinically relevant adverse effects of mercury exposure on coronary heart disease, stroke or total cardiovascular disease in U.S. adults at the exposure levels seen in this study.***"

1. Claims for links of "mercury" to cardiovascular disease (CVD), coronary heart disease (CHD) and death

Salonen et al. (1995)[138], based on the Kuopio Ischaemic Heart Disease (KIHD) Risk Factor Study, was the first to suggest a strong statistical relationship linking non-fatty freshwater fish consumption, levels of urine and hair mercury, and risk of CHD and CVD for a group of men in eastern Finland. A subsequent study by the same group in Salonen *et al*. (2000)[139] reported a correlation between mercury accumulation and accelerated progression of carotid atherosclerosis. The most recent results from this University of Kuopio group are reported by Virtanen *et al*. (2002)[140]:

> "The [KIHD] study is an ongoing population-based study designed to investigate risk factors for CHD, atherosclerosis and related outcomes in [1833 to 2005] middle-aged men from eastern Finland, **a population with one of the highest recorded rates of CHD.**[141] [emphasis added] … The mean hair content of mercury was 1.9 ppm. The subjects were divided into quarters according to the mean hair mercury content (<0.66, 0.66-1.31, 1.32-2.50 and >2.50 ppm). The men in the highest quarter of hair mercury had almost **two times higher intake of fish** [original emphasis] than the men in the lower three quarters (68 vs. 38 grams/day …). During an average follow-up time of 12 years, 114 CVD deaths and 76 CHD deaths occurred among men free of CVD at baseline. In the Cox proportional hazards' model adjusted for age, examination years, serum HDL and LDL cholesterol and triglycerides, family history of ischaemic heart disease, systolic blood pressure, body mass index, serum selenium and intakes of saturated fatty acids, fiber, vitamin C and E and beta-carotene, **men in the highest quarter** [original emphasis] of hair mercury content (>2.5pm) had a **1.6-fold** (95% CI [confidence interval], 1.1-2.4) **risk of CVD death and 1.7-fold** (95% CI, 1.0-2.7) **risk of CHD death** [original emphasis] when compared with men in the lowest three quarters. "

But in "Epidemiology Faces Its Limits" (1995, *Science*, vol. 269, 164-169), science reporter Gary Taubes cautioned that most epidemiologists interviewed by *Science* magazine suggested that for a statistical association to be taken seriously, the relative risk ratio better be at least higher than 3.[142] Thus, ***it is clear that the relative risk ratios of 1.6 to 1.7 by the Finnish KIHD results of Salonen et al. (1995, 2000) are far below the statistical association standard that would be taken seriously by most epidemiologists.***

Next, Guallar *et al*. (2002)[143] based its conclusions on a case-control study of 684 men from eight European countries and Israel with myocardial infarction and 724 controls. It

reported that increasing toenail mercury levels from as low as 0.11 to 0.66 ppm (or about 0.34 to 2.03 ppm in equivalent hair mercury levels) "was directly associated" with a doubling of the risk of myocardial infarction **after adjusting for** age, DHA level, body-mass index, waist-to-hip ratio, smoking status, alcohol intake, HDL cholesterol level, diabetes, history of hypertension, family history of heart attack, blood levels of Vitamin E and beta-carotene and toenail selenium. These authors concluded that "high mercury content may diminish the cardioprotective effect of fish intake."

2. Scientific criticism of source papers by Salonen et al. (1995, 2000) and Guallar et al. (2002)

It is well recognized that **CVD and CHD have multiple risk factors**[144] like age, family history, stress, dietary habits, smoking, alcohol use, diabetes and socio-economic status **that are not easily resolved or separated**. In fact, the University of Kuopio group, while examining the KIHD database, reported many additional risk factors beyond the popularly promoted complications from mercury. These include:

(1) Hypertension[145],

(2) Mutation of hemochromatosis gene Cys282Tyr[146] (resulting in excess iron accumulation),

(3) Bingeing on vodka (a whole bottle or more in 1 session) or beer (greater than 6 beers at a time),[147]

(4) Vitamin C deficiency,[148]

(5) Low intakes of fruits, berries and vegetables,[149]

(6) Low folate,[150]

(7) Low intake of lycopene[151] (an antioxidant carotenoid mainly from tomatoes and tomato products), and

(8) Blood donation.[152]

In this context, the primary criticisms concerning the claimed association of mercury and CVD+CHD by Salonen *et al*. (1995, 2000) **are confirmed in the authors' 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***." [Emphasis added]

Additionally, both Salonen *et al*. (1995) and Virtanen *et al*. (2002) clearly identified and confirmed that the men with the highest hair mercury group in their sample have a relatively larger proportion of rural inhabitants consuming local nonfatty fish species caught from local lakes and a significantly higher level of saturated fatty acids intake and measured LDL cholesterol (i.e., low-density lipoprotein or so-called bad cholesterol). Statistics compiled by the "Seven Countries Study" (USA, Finland, The Netherlands, Italy, former Yugoslavia, Greece and Japan) in Menotti et al. (1999)[153] show that the **cohort from eastern Finland has the highest CHD 25-year death rate – 268/1000 deaths –** compared to 25/1000 CHD deaths for a cohort from Crete, Greece and 30/1000 CHD deaths for a cohort from Tanushimaru, Japan, where the consumption of animal fats is distinctly lower.

Coupling such information with the failure to account for stress – a suspected major risk factor[154] – in the KIHD study by Salonen *et al*. (1995), raises the significant question of ***whether this result for men from eastern Finland is at all relevant to assessing the potential CVD+CHD vulnerability of average Americans from consuming a variety of ocean fish.***

Stern (2005) identified another major weakness in the Finnish KIHD study. During the Salonen *et al*. (1995) study, ***up to nine years elapsed between the collection of hair and urine samples and the recording of a CVD, CHD and death event.*** (Most data collection was carried out between March 1984 and December 1989, but the single hair and urine mercury analysis[155] was conducted at a much later time, between May 1992 and August 1993 at the Department of Chemistry of the University of Kuopio. CVD, CHD and death events were reported until the end of 1992). An updated report of the KIHD mercury-related results in Virtanen *et al*. (2002) extends the problematic, long elapsed time to 16 years or so, thereby contributing to ***a serious exposure misclassification if there were any changes in the subjects' dietary habits or simply their amount of fish consumption during the intervening 16 years.***

Clarkson (2002) critically noted: "The highest recorded hair level of 15.7 ppm[156] was more than six standard deviations from the mean. A histogram of hair levels was not presented, but these statistics imply only a small percentage of the study group had high mercury levels. Such outlying and 'influential points' may play a major role in studies of this type. It would have been of interest to see if these correlations persisted when the very high mercury levels were excluded."[157]

Barbara V. Howard, both as chair of the American Heart Association (AHA) Nutrition Committee and president of MedStar Research Institute, cautioned:

> "These [mercury-related] results from Kuopio are intriguing, but preliminary, and should be viewed in the context of many other studies that have shown ***a clear cardiovascular benefit*** to consuming fish on a regular basis. It is important to note that this is an observational study, and the ***conclusions do not prove a direct relationship*** between the amount of mercury in hair and heart attacks. There may be factors such as the socio-

economic status of the men or other dietary factors that are hard to measure, that account for the higher risk."[158] [Emphasis added]

It is perhaps most telling that in an earlier experimental study of 62 healthy students from Kuopio, Finland, Agren *et al*. (1988) [159] had found that

> "***A moderate intake of fish-containing meals has some beneficial effects on plasma lipid and prostanoid metabolism, when coronary heart disease risk factors are considered***." [Emphasis added]

It is important to stress that the selected Kuopio students ate on average 3.7 times fish-containing meals per week for 15 weeks and that 87% of those fish meals consisted of locally caught freshwater fish (vendace, pike, perch and rainbow trout) and 13% of Baltic herring from brackish water. ***The largest decrease in CHD risk was found for those 21 students who both ate Finnish freshwater fish meals and restricted their lipid intake, when compared to two other groups***.[160] In other words, for the University of Kuopio students, restricting intake of bad, saturated fatty acids and consuming moderate amounts of even freshwater fish from local Finnish lakes appeared to provide beneficial health effects.

Having evaluated the problematic claims of the poorly designed KIHD study by Salonen *et al*. (1995, 2000), we now turn to the identified direct impact of mercury on myocardial infarction by Guallar *et al*. (2002).

In a rather sharp contrast to the results presented by Guallar *et al*. (2002), the same issue of the *New England Journal of Medicine* included a study by Yoshizawa *et al*. (2002).[161] ***Based on a 5-year follow-up of 33,737 U.S. male health professionals with no previous history of CVD or cancer,*** the Yoshizawa study concluded that ***there is no clear association between total mercury exposure*** (covering measured toe nail mercury levels from about 0 to 14.56 ppm) ***and risk of CVD***, after adjusting for age, smoking and other CVD risk factors.

In addition to the contradictory evidence offered by Yoshizawa *et al*. (2002) and the clear difficulty in singling out mercury as the predominant risk factor for CVD and CHD, Plante and Babo (2003)[162] raised the following criticisms in the *New England Journal of Medicine*:

> "We find it **difficult to reconcile** this [Guallar *et al*., 2002] finding with published data on the cardiovascular health of highly exposed populations. ***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. According to data from monitoring programs in Canada,***

> ***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 approximately 2 ppm, how can one explain the **absence** of significant effects at doses greater than 100 ppm? The authors [Guallar *et al*., 2002] raise the possibility of **modifying fish-intake recommendations** on the basis of their findings. ***In our opinion, this suggestion is ill founded and may do more harm than good, considering the nutritional value of fish."*** [Emphasis added]

3. Additional evidence questioning claimed mercury harm to heart-related health

It appears convincing from the above literature that the claimed role of methylmercury in negatively impacting the human heart is neither clear nor direct. However, it could reasonably be argued or concluded from the available evidence that ***numerous risk factors other than methylmercury from fish more likely explain most of the findings in Salonen et al. (1995, 2000) and Guallar et al. (2002).***

Moreover, as with the troubled Faeroe data concerning neurodevelopment of children or infants, there are reasonable concerns that ***the Finnish results are not directly applicable for the U.S. population.*** Thus, a relevant question is whether there is additional evidence either for or against the claim of a connection between methylmercury and heart-related health that speaks more directly and appropriately to the U.S. population (in addition to Yoshizawa *et al*. (2002) referenced above).

We look forward to reviewing additional results from the ongoing Center for Disease Control (CDC) US National Health and Nutrition Examination Survey (NHANES), such as the Vupputuri *et al*. (2005)[163] study in the journal *Environmental Research*. Related to heart health, these researchers report ***another failure to find support for a direct connection between mercury and blood pressure*** in the NHANES database:

> "***We found no significant association between total blood mercury and systolic and diastolic BP [blood pressure] among [the NHANES] study participants overall. … Our findings support the hypothesis that the intake of fish oils may counter the harmful effects of mercury on BP regulation.***"[164] [Emphasis added]

Vupputuri *et al*. point to support by the experimental study of omega-3 DHA fatty acids in Engler *et al*. (2003).[165] That analysis concluded, "It is possible that the harmful effects of mercury exposure on BP may be offset by the consumption of fish, which may reduce [i.e., rather than increase] BP and pulse pressure, as well as decrease arterial stiffness."

Similarly, Dorea *et al*. (2005)[166] examined data for hair mercury and blood pressure for the Munduruku and Kayabi Indians of Amazonia. They found that "Hg per se was not significantly related to blood pressure [for] all ages considered [from about 15 to 80 years old]. However, as a function of age, adult individuals of the Munduruku group had a tendency toward high pressure ... which was not shown for the Kayabi [group]." ***Thus, while age may be tentatively identified as a negative factor on systolic blood pressure for the Munduruku natives in the Amazon, mercury was not found to be a risk factor.***

Finally, the paper "Fish Intake and Risk of Incident Atrial Fibrillation" in the AHA's journal, Circulation, Mozaffarian *et al*. (2004)[167] conducted a 12-year follow-up for a cohort of 4,815 men and women over 65 in 4 US communities. It found that ***adults consuming tuna or other broiled or baked fish[168] 1 to 4 times per week had 28% lower risk of developing atrial fibrillation when compared to those who ate fish less than once per month. Those eating fish five times or more per week showed a 31% lower risk.*** Mozaffarian *et al*. (2004) concluded that "fish intake may influence risk of this common cardiac arrthymia" that affects more than 2 million individuals in the United States.

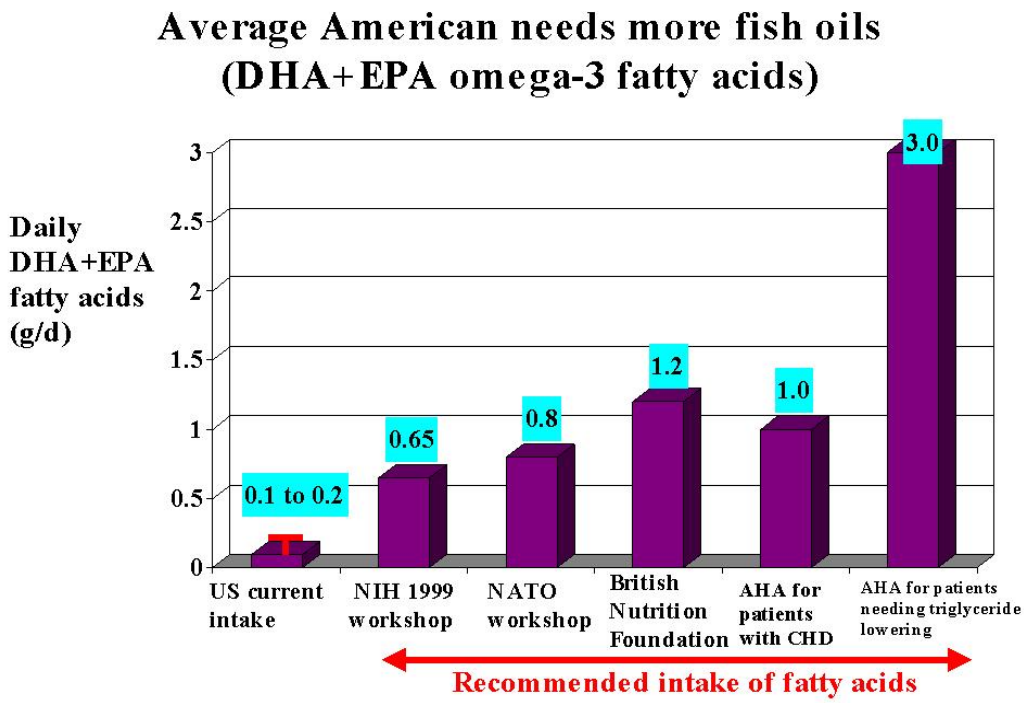
4. Americans need more, not less fish in their diets.

Figures 31 and 32 confirm that the U.S. population is susceptible to potential life-threatening diseases, because current levels of average EPA+DHA omega-3 polyunsaturated fatty acids consumption are **already 3 to 6 times lower** than recommendations by National Institute of Health (NIH) and AHA.

Dorea (2003) [169] and Dorea *et al*. (2004) analyzed the diet and health of native populations of the Amazonian rainforest. These papers point out the critical role that fish nutrition plays in human health:

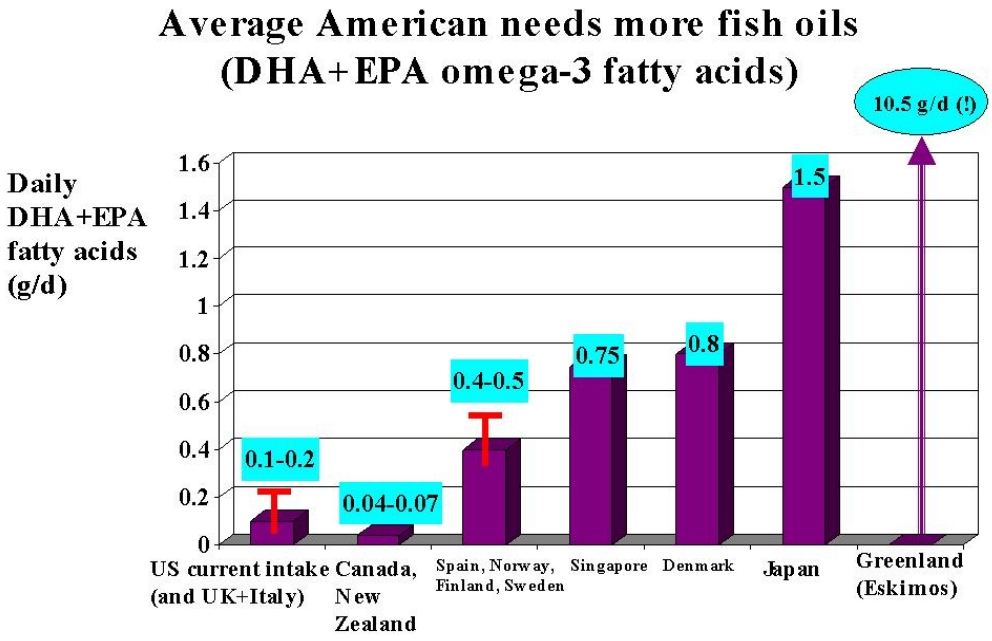
> "Fish is a nutritious and important dietary staple of the people of the Amazonian rain forest. It is an abundant natural resource that is rich in high-quality protein, lysine, iodine, sulfur-containing amino acids, copper, calcium, zinc, iron, manganese, selenium, and omega-3 polyunsaturated fatty acids, among others. In the Amazonian rain forest, fish supply much needed protein and provide a balance to starchy food-staples such yam, cassava, and plantain. ... In spite of substantial amounts of metallic Hg released due to gold-mining activity, ***there is no evidence*** that this Hg has impacted fish Hg in the head tributaries of Rio Tapajos. Fish consumption is the only source of MMHg [MonoMethylmercury] exposure for native people who do not have access to commodity foods. For these people, to reduce dietary MMHg means reducing the consumption of fish, a dietary staple and a source of many important nutrients. ***No evidence exists*** that shows that freshwater Amazonian fish cause neuropathies. As an abundant natural resource, fish has been consumed for generations in large amounts

Figure 31



Sources: Kris-Etherton et al. (2000, Am. J. Clin. Nutr., vol. 71, 179-188); Kris-Etherton et al. (2003, Arterioscler. Thromb. and Vasc. Biol., vol. 23, e20-e30); Din et al. (2004, Brit. Med. J., vol. 328, 30-35)

Figure 32



Sources: Kris-Etherton et al. (2000, Am. J. Clin. Nutr., vol. 71, 179-188); Iso et al. (2001, JAMA, vol. 285, 304-312); Terry et al. (2003, Am. J. Clin. Nutr., vol. 77, 532-543); Gago-Dominguez et al. (2003, Brit. J. Cancer, vol. 89, 1686-1692)

> by Amazonian people without any perceived problems. For these people, exposure to fish MMHg from the forest environment (non-industrial sources) is less of an issue than endemic infectious diseases such as malaria." [Emphasis added]

Ropeik (2004), in his evaluation of the consequences of exaggerated fear, finds that:[170]

> "[T]he cumulative load of modern threats may be creating an even greater risk that is largely overlooked: the risk that arises from misperceiving risks as being higher or lower than they actually are. As a result of some of the decisions we make when we are fearful, some of the choices we make when we are not fearful enough, and because of the ways our bodies react to chronically elevated levels of stress, the hazards of risk misperception may be more significant than any of the individual risks about which we fret. ... Most importantly, the costs of risk misperception, especially from fear and anxiety, must be included in cost-benefit analyses of risk management options."[171]

Again***, alarmism over mercury is causing a serious decline in U.S. consumption of fish, in the face of no convincing scientific evidence that dangerous threats exist for heart-related disease from the variety of fish available to Americans***. In contrast, there is huge potential for a public health crisis resulting from extreme precautions over hypothetical mercury health threats from fish consumption. This is so because the proven positive health benefits from fish are being compromised and de-emphasized. This negative health burden will fall disproportionately on poor and minority groups which depend on fish for vital daily nutrition, as well as on pregnant women who require vital nutrition to support themselves and healthy fetal development.

References and Notes

[1] The 946-page EPA's NESHAP proposal is available here: http://www.epa.gov/airquality/powerplanttoxics/pdfs/proposal.pdf. Published in Federal Register on May 3, 2011; available here: http://edocket.access.gpo.gov/2011/pdf/2011-7237.pdf.

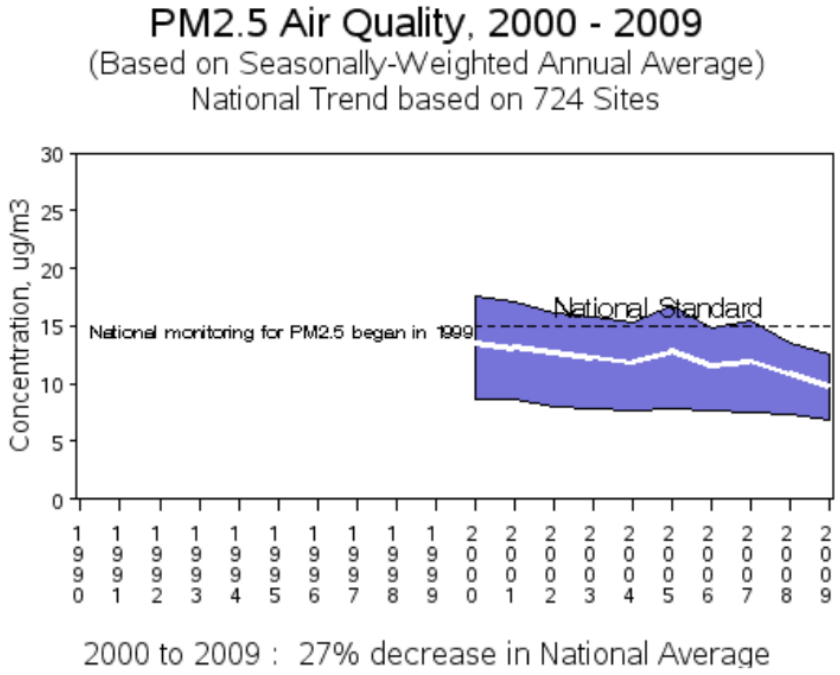
[2] UNEP's "The Global Atmospheric Mercury Assessment: Sources, Emissions and Transport" (2008; updated and corrected May 2009). Available from http://www.chem.unep.ch/Mercury/Atmospheric_Emissions/UNEP%20SUMMARY%20REPORT%20-%20CORRECTED%20May09%20%20final%20for%20WEB%202008.pdf.

[3] see http://www.epa.gov/mercury/control_emissions/global.htm.

[4] see EPA's Novermber 10 2009's press release http://yosemite.epa.gov/OPA/ADMPRESS.NSF/d0cf6618525a9efb85257359003fb69d/62b53c67bc92ef878525766a004b3456!OpenDocument

[5] Wiedinmyer and Friedli (2007) *Environmental Science & Technology*, vol. 41, 8092-8098.

[6] EPA's "co-benefits" claim should not go unchallenged. It is not entirely clear on what the EPA NESHAP proposal is achieving for the ever-more stringent PM2.5 control since as the real-world data is showing a steady decreasing tendency for the PM2.5 air quality standard from 2000 through 2009, with means below the National Standard, as shown below:



(source of the graph: http://junksciencecom.files.wordpress.com/2011/03/epa_s-clean-air-act-final.pdf)

[7] Mason et al. (2005) *Environmental Science & Technology*, vol. 39, A14-A22.

[8] Slemr et al. (2003) *Geophysical Research Letters*, vol. 30, 2003GL016954.

[9] Slemr et al. (2011) *Atmospheric Chemistry and Physics*, vol. 11, 4779-4787..

[10] The mercury exposure levels in Seychelles are about 10 times those of U.S. women and they eat fish with mercury similar to those of the U.S. commercial ocean fish.

[11] Debes et al. (2006) *Neurotoxicology and Teratology*, vol. 28, 536-547.

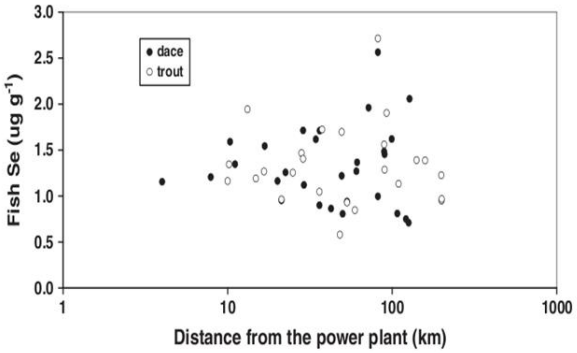
[12] Myers et al. (2009) *Neurotoxicology*, vol. 30, 338-349.

[13] WS notes that selenium, Se, is one of the other 83 toxic hazardous air pollutants considered in this EPA's NESHAP proposed rules. This EPA proposal estimated that total Se emission from EGUs for 2010 is about 258 tons (p. 171-172 of EPA document or p. 25006 in Federal Register) and the meaning of this emission control needs to be confronted with the reality that marine biogenic emission of Se is estimated to be roughly 5000-8000 tons and total Se recycled through the atmosphere is about 13000 to 19000 tons (see

Wen and Carignan, 2007, *Atmospheric Environment*, vol. 41, 7151-7165). Although not within the scope of this report, WS wish to report this relevant new paper by Jardine and Kidd (2011, *Science of the Total Environment*, vol. 409, 785-791) where the study could not find any relation between the concentration of Se in fish tissues and coal-fired power plant locations and emission outputs (see figure shown in this endnote):

> "Our data showed no relationship between the proximity to the coal-fired power plant and Se concentrations in local biota. **There was no relationship between Se concentrations and distance from the coal-fired power place for [blacknose] dace** ($r^2 < 0.01$, p=0.806, n=29), **[brook] trout** ($r^2 < 0.01$, p=0.910, n=25), **water strider** ($r^2 = 0.03$, p=0.307, n=41; not shown) **or predatory invertebrates** ($r^2 < 0.01$, p=0.977, n=14; not shown). **This may be due to the very low amount of Se emitted from coal burning relative to Hg and S [sulfur], as concentrations of Se are approximately 2000 times lower than S in coal.** This and other coal-fired power plants may also emit low levels of Se compared with that released from other point sources such as metal smelters. The Sudbury metal smelters have historically been identified as being one of the world's largest Se emission sources at approximately 2 tonnes per day, likely much larger than emitted from the power plant in the current study. … **[M]any fishes had lower [Se] concentrations than their invertebrate prey and trophic transfer was higher at sites with low invertebrate Se concentrations. Variability in Se concentrations in fishes was explained more by site of capture than microhabitat use within the site, suggesting among-site differences in geological sources of Se.** … Our studies suggest that, as predicted by kinetic models (Wang, 2002), trophic enrichment of Se is a rare phenomenon in these stream food webs. Furthermore, **the concentrations of Se are not a concern for fish and wildlife supported by these systems, and are consistent with previous, low Se measurements on the blood of fish-eating common loons in the region.** " [emphasis added]

No relation between Se concentration in fish tissues and distance from coal-fired power plants



Jardine and Kidd (2011) Science of the Total Environment, vol. 409, 785-791

[14] Ralston and Raymond (2010) *Toxicology*, vol. 278, 112-123.

[15] Although this is not the focus of this WS report, WS wishes to point out that:

(1) On pp. 272-273: although EPA claimed to base its proposed 91% Hg emission cuts on actual data, EPA specifically admitted (p. 273) that with the Active Carbon Injection (ACI) Control Technology, "EPA had no direct stack test results showing how effectively these ACI-equipped plants reduced their Hg."

(2) On p.399: similar statements by EPA, acknowledging that they have no measured or proven data to achieve the 91% Hg emission cuts.

(3) On p. 440: EPA specifically cited a private (opinion) letter from one David Foerter, executive director of the Institute of Clean Air Companies to former Senator Carper in footnote #172 for its claim that wet and dry scrubbers could be installed within 3 years time.

(4) On pp. 445-446: EPA cited another non-peer-reviewed PowerPoint presentation (as footnote #173) by one Paul M. Sotkiewicz of PJM Interconnection, in support of its claim that Hg control could be online by "third quarter of 2014".

[WS noted on April 7, 2011: Scott Segal, a partner at Bracewell & Giuliani and president of the Electric Reliability Coordinating Council, also independently raised this point in a t.v. interview on March 28, 2011 available here http://www.eenews.net/tv/2011/03/28/].

[16] Roman et al. (2011) *Environmental Health Perspectives*, in press, doi: 10.1289/ehp.1003012 (available online January 10, 2011).

[17] Rice et al. (2010) *Environmental Science & Technology*, vol. 44, 5216-5224.

[18] As most clearly shown in the March 24, 2011 publication of Mozaffarian et al. (2011) *New England Journal of Medicine*, vol. 364, 1116-1125.

[19] Available from http://yosemite.epa.gov/OPA/ADMPRESS.NSF/d0cf6618525a9efb85257359003fb69d/62b53c67bc92ef878525766a004b3456!OpenDocument.

[20] Available from http://www.chem.unep.ch/Mercury/Atmospheric_Emissions/UNEP%20SUMMARY%20REPORT%20-%20CORRECTED%20May09%20%20final%20for%20WEB%202008.pdf.
The main source of this UNEP is report is based on the new paper by Pacyna et al. (2010) *Atmospheric Environment*, vol. 44, 2487-2499.

[21] WS notes that the 390 tons of mercury emissions estimated from China's fossil-fueled combustion for electricity and heating in 2005 by Pacyna et al. (2010) is a bit larger than the 284 tons value from Tian et al. (2010, *Atmospheric Chemistry and Physics*, vol. 10, 11905-11919); however, **personal communication with Professor Jozef Pacyna suggested that Chinese officials tend to underestimate or undercount their own Hg emissions. Furthermore, the (independent) constraints from atmospheric mercury content and mercury transport measurements by Jaffe et al. (2005, *Atmospheric Environment,* vol. 39, 3029-3038) support the hypothesis of the underestimation of the mercury emissions from China that was obtained from emission inventory.**

[22] Pacyna et al. (2006) *Atmospheric Environment*, vol. 40, 4048-4063.

[23] Slemr et al. (2003) *Geophysical Research Letters*, vol. 30, 2003GL016954.

[24] Slemr et al. (2011) *Atmospheric Chemistry and Physics*, vol. 11, 4779-4787.

[25] Lindberg et al. (2007) *Ambio*, vol. 36, 19-32.

[26] Wiedinmyer and Friedli (2007) *Environmental Science & Technology*, vol. 41, 8092-8098.

[27] Pyle and Mather (2003) *Atmospheric Environment*, vol.37, 5115-5124; Mather and Pyle (2004) *Science of the Total Environment*, vol. 327, 323-329.

[28] Martin et al. (2011) *Chemical Geology*, vol. 283, 279-286; Bagnato et al. (2010) *Bulletin of Volcanology*, in press doi 10.1007/s00445-010-0419-y (published online October 27, 2010); Witt et al. (2008) *Journal of Geophysical Research*, vol. 113, 2007JB004501.

[29] p. 18 of the 2008's UNEP Mercury Report. Similar information can be found in Table 6 on p. 2494 of Pacyna et al. (2010, *Atmospheric Environment*, vol. 44, 2487-2499). For the U.S. alone; Cain et al. (2007, *Journal of Industrial Ecology*, vol. 11, 61-75) listed the pathway of emissions by "dental office/cremation/exhaled air" to be about 3 tons per year.

[30] Rasmussen (1994) *Environmental Science & Technology*, vol. 28, 2233-2241. Based on the careful research of Rasmussen (1994) and several other available estimates : (1) As discussed in the main text, Richardson et al. (2001, 2003) gave 58,000 tons, 4500 tons and 1100 tons for natural Hg emissions over global domain, USA and Canada, respectively and (2) Jaworowski et al. (1981, *Geochimica et Cosmochimica Acta*, vol. 45, 2185-2199) that 190,000 tons for natural Hg emission**, it is reasonable to suggest that total Hg emission from natural sources and archives must be at least 10,000 tons/year.** It can be noted that earlier publication by Nriagu and Pacyna (1988, *Nature*, vol . 333, 134-139) offered 6000 tons as the natural Hg emission. Two most recent estimates and geochemical modeling constraints by Pirrone et al. (2010, *Atmospheric Chemistry and Physics*, vol. 10, 5951-5964) and Holmes et al. (2010, *Atmospheric Chemistry and Physics*, vol. 10, 12037-12057) offered natural Hg emission of 5207 tons and 6250 tons, respectively, which are significantly larger than the estimated anthropogenic Hg emission amounts of 2320 tons and 2050 tons, respectively. The sensitivity study of atmospheric mercury cycling by Lohman et al. (2008, *Applied Geochemistry*, vol. 23, 454-466) using global chemical transport model also support the current scenario calling for an increasingly larger emission from natural sources or re-emission from previously deposited mercury. This new tendency emphasizing relatively larger emissions from natural sources differs slightly from most previous assumptions that called for roughly equal amount and role from natural and anthropogenic sources for Hg cycling and recycling in the atmosphere (see e.g.,

Mason and Sheu, 2002, *Global Biogeochemical Cycles*, vol. 16, doi:10.1029/2001GB001440). Lindberg et al. (2004, *RMZ-Materials and Geoenvironment*, vol. 51, 1172-1176) raised a very important point that "Recent advances in atmospheric mercury measurement technology, aircraft sampling, and application of near real-time flux techniques have dramatically expanded and improved the data on mercury fluxes and behavior in the atmospheric. It seems that these new approaches have most commonly led to the discovery of new sources of Hg emissions to the atmosphere, increases in earlier estimates of emissions or better quantification of emissions previously underestimated, and increased estimates of rates of previously deposited mercury. ***On the whole, we have seen many new sources quantified (e.g., wildfires), but few new sinks. If the global atmospheric pool is not increasing, current conceptualizations of the global cycle may be missing some important sinks.***" [emphasis added] Lindberg et al. (2004) estimated a throughfall dry deposition flux of reactive gaseous mercury and particulate mercury of about 500 tons/year and a large litterfall dry mercury deposition sink of 2400 to 6000 tons/year.

[31] Pacyna et al. (2010) *Atmospheric Environment*, vol. 44, 2487-2499.

[32] The reference to 58,000 tons of annual natural mercury emissions reflects better accounting for major geological sources, compared to previous study, and is based on the 2001 report "Critical Review on Natural Global and Regional Emissions of Six Trace Metals to the Atmosphere" by Richardson et al. That review is a technical report prepared for the International Lead Zinc Research Organization, International Copper Association, and the Nickel Producers Environmental Research Association.

[33] Richardson et al. (2003) *Environmental Reviews*, vol. 11, 17-36. See however the comment and criticism by Schroeder et al. (2005) *Journal of Geophysical Research*, vol. 110, 2004JD005699. It remains true that natural effluxes of Hg from various surfaces and reservoirs of Earth have been largely under accounted for.

[34] Table 3 on page 154 of EPA March 16, 2011 proposed NESHAP rules.

[35] Ribeiro Guevara et al. (2010) *Atmospheric Chemistry and Physics*, vol. 10, 3443-3453.

[36] Jitaru et al. (2009) *Nature Geoscience*, vol. 2, 505-508.

[37] Schober et al. (2003) *Journal of the American Medical Association*, vol. 289, 1667-1674.

[38] http://cfpub.epa.gov/eroe/index.cfm?fuseaction=detail.viewInd&lv=list.listByAlpha&r=224031&subtop=208.

[39] Caldwell et al. (2009) *International Journal of Hygiene and Environmental Health*, vol. 212, 588-598.

[40] Wells et al. (2011) *Environmental Research*, vol. 111, 411-417.

[41] Krug and Winstanley (2004) *Hydrology & Earth System Sciences*, vol. 8, 98-102.

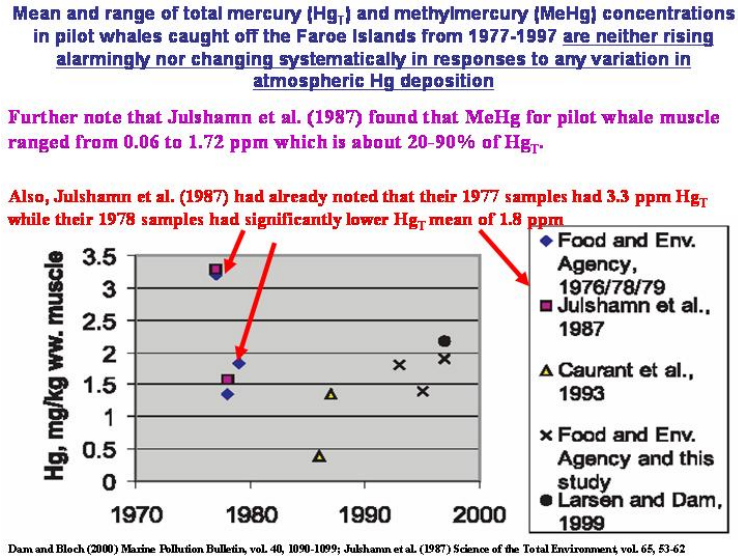
[42] Aelion et al. (2008) *Science of the Total Environment*, vol. 402, 149-156.

[43] Fang et al. (2004) *Science of the Total Environment*, vol. 330, 159-170.

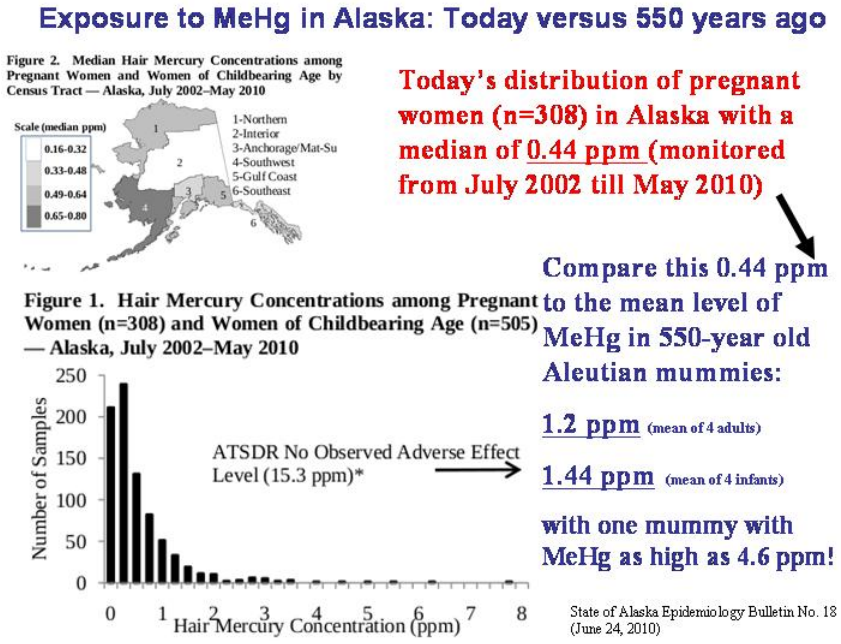
[44] Caffrey et al. (2010) *Atmospheric Chemistry and Physics*, vol. 10, 5425-5434.

[45] Shotyk et al. (2005) *Geochimica et Cosmochimica Acta*, vol. 69, 1-17.

[46] The mean and range of total mercury (Hg_T) and MeHg concentrations in pilot whale meat from the Faroe Islands over the 1977-1997 interval are shown to be neither rising alarmingly nor changing systematically in responses to any variation in atmospheric Hg deposition.



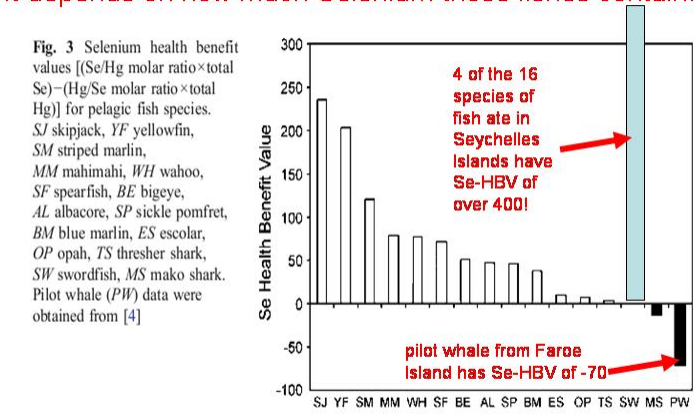
[47] Poikolainen et al. (2004) *Arctic, Antarctic, and Alpine Research*, vol. 36, 292-297. They applied the novel, biomonitoring technique of using mosses in tracing accumulation of both mercury and other trace metals deposited from the atmosphere.
[48] Larssen et al. (2008) *Science of the Total Environment*, vol. 404, 290-296.
[49] Sathpathy et al. (2008) *Current Science*, vol. 95, 374-381.
[50] from pp 379-380 of Sathpathy et al. (2008) *Current Science*, vol. 95, 374-381.
[51] Selvaraj et al. (1999) *Current Science*, vol. 77, 494-497.
[52] p. 29 of Lindberg et al. (2007) *Ambio*, vol. 36, 19-32.
[53] See Walsh et al. (1987) *Pure & Applied Chemistry*, vol. 59, 295-298; Camargo (1993) *Nature*, vol. 365, p. 302; CRC Handbook of Chemistry & Physics, 78th Edition (1997-1998) [available at http://www.speclab.com/elements/mercury.htm].
[54] Liu et al. (2009) *Environmental Science & Technology*, vol. 43, 4361-4366.
[55] p. 266 of Marvin-DiPasquale et al. (2003) *Environmental Geology*, vol. 43, 260-267.
[56] Trudel and Rasmussen (1997) *Environmental Science & Technology*, vol. 31, 1716-1722.
[57] Siciliano et al. (2005) *Environmental Science & Technology*, vol. 39, 1071-1077.
[58] Schaefer et al. (2004) *Environmental Science & Technology*, vol. 38, 4304-4311.
[59] Mason et al. (2005) *Environmental Science & Technology*, vol. 39, A14-A22.
[60] A comparison of MeHg levels in ancient and modern Alaskans (monitored from July 2002 till May 2010) found a higher mean level in 550-year old mummies.



[61] Holloway et al. (2009) *Chemical Geology*, vol. 267, 95-95.
[62] Shannon and Voldner (1995) *Atmospheric Environment*, vol. 29, 1649-1661.
[63] Ekstrom and Morel (2008) *Environmental Science & Technology*, vol. 42, 93-99.
[64] Poulain et al. (2007) *Applied and Environmental Microbiology*, vol. 73, 2230-2238.
[65] Schaefer and Morel (2009) *Nature Geoscience*, vol. 2, 123-126.
[66] Mason and Sullivan (1999) *Deep-Sea Research Part II*, vol. 46, 937-956.
[67] Pongratz and Heuman (1999) *Chemosphere*, vol. 39, 89-102.
[68] Pongratz and Heuman (1998) *Chemosphere*, vol. 36, 1935-1946.
[69] Limper et al. (2008) *Journal of Applied Entomology*, vol. 132, 168-176.
[70] Stewart et al. (2010) "Bioaccumulation and trophic transfer of selenium" book chapter in *Ecological Assessment of Selenium in the Aquatic Environment*, edited by Peter M. Chapman et al., Pensacola FL, CRC Press, pp. 93-139.
[71] Jardine and Kidd (2011) *Science of the Total Environment*, vol. 409, 785-791.

[72] Belzile et al. (2006) *Canadian Journal of Fisheries and Aquatic Sciences*, vol. 63, 1-10.
[73] Clarkson and Magos (2006) *Critical Reviews in Toxicology*, vol. 36, 609-662.
[74] Dourson, et al., *Toxicology Excellence for Risk Assessment*, ALEC meeting, December 3, 2000.
[75] Myers et al. (2005), *International Review of Research in Mental Retardation*, vol. 30, 141-169.
[76] Dourson et al. (2001) Neurotoxicology, vol. 22, 677-689.
[77] The Danish Ministry of Environment's National Environmental Research Institute's Technical Report (2004) No. 525, "Screening of 'new' contaminants in the marine environment of Greenland and The Faroe Islands".
[78] One might ask about Weihe's cited connection between the Faroe study and Harvard. The actual connection is merely that Phillippe Grandjean of Denmark, who led the Faroe Study, then moved to Boston to take an adjunct position at Harvard Medical School. Harvard had nothing to do with either the original study or the follow up analyses.
[79] Yorifuji et al. (2010) *Neurotoxicology & Teratology*, in press (Accepted September 16, 2010).
[80] Petersen et al. (2008a) *Neurotoxicology*, vol. 29, 584-590. WS like to note, however, that this study may have significant limitations especially when they used historical records of whale catches to determine the subject exposure levels. There is no evidence that this assumption is valid.
[81] Petersen et al. (2008b) *Neurotoxicology*, vol. 29, 591-595.
[82] Comments of the Utility Regulatory Group to EPA (69 Fed. Reg. 12398 [March 16, 2004]), Docket ID No. OAR-2002-0056.
[83] The State of California vs. Tri-Union Sea Foods. May 11, 2006. The court decision can be downloaded here: http://www.heartland.org/custom/semod_policybot/pdf/19103.pdf.
[84] Ibid. p 40.
[85] Ibid. p. 40, 41. The paper involving Deborah Rice (of EPA at the time of authorship) as a co-author is by Schantz, Widholm and Rice (2003) "Effects of PCB Exposure on Neuropsychological Function in Children", *Environmental Health Perspectives*, vol. 111, 357-376.
[86] Grandjean et al. (2001) *Neurotoxicology & Teratology*, vol. 23, 305-317
[87] Grandjean et al. (1997) *Neurotoxicology & Teratology*, vol. 19, 417-428
[88] Julshamn et al. (1987) *Science of the Total Environment*, vol. 65, 53-62. See also Kaneko and Ralston (2007) *Biological Trace Element Research*, vol. 119, 242-254.
[89] J. Bemis and R. Seegal, 1999, "Polychlorinated Biphenyls and Methylmercury Act Synergistically to Reduce Rat Brain opamine content in Vitro," *Environmental Health Perspectives*, vol. 107: 879–885.
[90] Myers et al. (2005) *International Review of Research in Mental Retardation*, vol. 30, 141-169.
[91] Methylmercury (MeHg). EPA's Integrated Risk Information System available at http://www.epa.gov/iris/subst/0073.htm.
[92] For example, see the objective comparison of the Selenium-Health Benefit Value derived by also Kaneko and Ralston (2007) *Biological Trace Element Research*, vol. 119, 242-254 shown here: Positive Se-HBV values mean the seafood is healthy and vice-versa.

Is the MeHg in ocean fish dangerous?
It depends on how much Selenium those fishes contain!



Kaneko and Ralston (2007) Biological Trace Element Research, vol. 119, 242-254

[93] Grandjean et al. (2010) *Environmental Health Perspectives*, vol. 118, 1137-1145; Julvez et al. (2010) *Neurotoxicology & Teratology*, vol. 32, 627-632; Yorifuji et al. (2010) *Neurotoxicology & Teratology*, in press (Accepted September 16, 2010).
[94] p. 1686 of Myers et al. (2003) *The Lancet*, vol. 361, 1686-1692.
[95] Clarkson TW, Cox C, Davidson PW, Myers GJ. (1998) "Mercury in Fish." Letter to the editor, *Science*. vol. 279, 459-460.
[96] Dourson et al. (2001) *Neurotoxicology*, vol. 22, 677-689
[97] It must be added that although NRC (2000) report emphasizes no "serious flaws" in the Seychelles, Faroe and New Zealand studies, it must be admitted that all studies indeed had not-so-inconsequential flaws. This is especially true in the New Zealand study. For example, Marsh (1994, p. 423) [in *Handbook of Clinical Neurology*, vol. 20, Intoxications of the Nervous System, Part I, edited by F. A. de Wolfe] pointed out that the New Zealand studies considered 3 populations of subjects (European, Maori and Pacific Islander children) which included the Maoris that were sociologically very diverse. Marsh (1994, p. 423) concluded that "This was a challenging study to execute and analyze, because the subjects were drawn from three distinct ethnic groups with social, linguistic, and scholastic differences which accounted for most of the differences in test results. It was the first fetal MeHg study that included sensitive psychological and educational tests in the evaluation of outcome. The data were adjusted for several co-variables, but others, such as maternal education and intelligence, were omitted. Studies of this type that seek to measure subtle threshold effects of MeHg have the difficult, and perhaps unattainable, goal of accounting for all other factors that affect child development."
[98] p. 1692 of Myers et al. (2003) *The Lancet*, vol. 361, 1686-1692.
[99] Lyketsos (2003) The *Lancet*, vol. 361, p. 1668.
[100] Davidson et al. (2008) *Neurotoxicology*, vol. 29, 453-459.
[101] Huang et al. (2007) *Neurotoxicology*, vol. 28, 1237-1244.
[102] Fonseca et al. (2008) *Neurotoxicology*, vol. 29, 1008-1015.
[103] Thurston et al. (2007) *Neurotoxicology*, vol. 28, 924-930.
[104] Myers et al. (2003, 2005).
[105] Kaneko and Ralston (2007) *Biological Trace Element Research*, vol. 119, 242-254.
[106] Ralston et al. (2007) *Biological Trace Element Research*, vol. 119, 255-268.
[107] Ralston and Raymond (2010) *Toxicology*, vol. 278, 112-123.
[108] Debes et al. (2006) *Neurotoxicology & Teratology*, vol. 28, 536-547.
[109] Yorifuji et al. (2010) *Neurotoxicology & Teratology*, in press (Accepted September 16, 2010).
[110] Davidson et al. (2010) *Neurotoxicology*, vol. 31, 439-447.
[111] Myers et al. (2009) *Neurotoxicology*, vol. 30, 338-349.
[112] Davidson et al. (2008) *Neurotoxicology*, vol. 29, 767-775.
[113] Strain et al. (2008) *Neurotoxicology*, vol. 29, 776-782.
[114] Hibbeln et al. (2007) *The Lancet*, vol. 369, 578-585.
[115] Prestbo and Gay (2009) *Atmospheric Environment*, vol. 43, 4223-4233.
[116] Best estimates are that Hg emissions from U.S. power plants account for less than 1% of total world annual emission budget. See in addition Pyle and Mather (2003, *Atmospheric Environment*, vol. 37, 5115-5124) for updated information on mercury emissions from volcanoes that were previously underestimated.
[117] The report is available here: http://water.epa.gov/scitech/swguidance/fishshellfish/techguidance/study/upload/2009_9_28_fish_study_data_finalreport.pdf.
[118] Monson (2009) *Environmental Science & Technology*, vol. 43, 1750-1755.
[119] Greenfield et al. (2005) *Science of the Total Environment*, vol. 336, 25-43.
[120] Yamaguchi et al. (2003) *Chemosphere*, vol. 50, 265-273.
[121] p. 341 of Weis (2004) *Environmental Research*, vol. 95, 341-350.
[122] Drevnick et al. (2007) *Environmental Science & Technology*, vol. 41, 7266-7272.
[123] Mahaffey (2004) *Environmental Research*, vol. 95, 414-428.
[124] Raymond and Ralston (2004) *Seychelles Medical and Dental Journal*, Special Issue, vol. 7, 72-77.
[125] Burger and Gochfeld (2005) *Environmental Research*, vol. 99, 403-412.
[126] Plessi et al. (2001) *Journal of Food Composition and Analysis*, vol. 14, 461-467.

[127] Myers, et al. (2005) "Developmental Disabilities Following Prenatal Exposure to Methyl Mercury from Maternal Fish Consumption: A Review of the Evidence," *International Review of Research in Mental Retardation*, vol. 30, 141-169.
[128] Peterson et al. (2009) *Environmental Science & Technology*, vol. 43, 3919-3925.
[129] Ralston and Raymond (2010) *Toxicology*, vol. 278, 112-123.
[130] Joshi et al. (2011) *Archives of Environmental and Contamination Toxicology*, in press (accepted February 17, 2011).
[131] Cheuk and Wong (2006) *Neuropediatrics*, vol. 37, 234-240.
[132] Ip et al. (2004) *Pediatrics International*, vol. 46, 715-721.
[133] This issue has been recently examined in the NRC (2007) report: "Seafood Choices-Balancing benefits and risks".
[134] Ropeik (2004) *European Molecular Biology Organization Reports*, vol. 5 (special issue), S56-S60.
[135] Stern (2005) *Environmental Research*, vol. 98, 133-142.
[136] Clarkson (2002) *Environmental Health Perspectives*, vol. 110 (supplement 1), 11-23.
[137] Mozaffarian et al. (2011) *New England Journal of Medicine*, vol. 364, 1116-1125.
[138] Salonen et al. (1995) *Circulation*, vol. 91, 645-655.
[139] Salonen et al. (2000) *Atherosclerosis*, vol. 148, 265-273.
[140] Virtanen et al. (2002) poster presentation in the April 23-26, 2002 American Heart Association, Asia Pacific Scientific Forum at Honolulu, Hawaii.
[141] The CHD mortality rate at eastern Finland is "10 times higher than in Crete in which the Mediterranean diet rich in plant foods and low in animal foods is consumed." (p. 199 of Rissanen et al., 2003, *Journal of Nutrition*, vol. 133, 199-204; see also Menotti et al., 1999, *European Journal of Epidemiology*, vol. 15, 507-515.)
[142] Taubes (1995, *Science,* vol. 269, 164-169) continued with the following important and revealing general discussion: "So what does it take to make a study worth taking seriously? ... **Sir Richard Doll** of **Oxford University**, who once co-authored a study erroneously suggesting that women who took the anti-hypertension medication reserpine had up to fourfold increase in the risk of breast cancer, suggests that **no single epidemiologic study is persuasive by itself unless the lower limit of its 95% confidence interval falls above a threefold increased risk.** Other researchers, such as **Harvard's [Dimitrios] Trichopoulos,** opt for a **fourfold risk increase as the lower limit.** Trichopoulos's ill-fated paper on coffee consumption and pancreatic cancer had reported a 2.5-fold increased risk. 'As a general rule of thumb,' says [Marcia] Angell of the *New England Journal* [*of Medicine, NEJM*], 'we are looking for a relative risk of three or more [before accepting a paper for publication] [*sic.*], particularly if it is biologically implausible or if it's a brand new finding.' **Robert Temple, director of drug evaluation at the Food and Drug Administration,** puts it bluntly: **'My basic rule is if the relative risk isn't at least three or four, forget it.'** But as **John Bailar, an epidemiologist at McGill University** and former statistical consultant for the NEJM, points out, there is no reliable way of identifying the dividing line. **'If you see a 10-fold relative risk and it's replicated and it's a good study with biological backup, like we have with cigarettes and lung cancer, you can draw a strong inference,' he says. 'If it's a 1.5 relative risk, and it's only one study and even a very good one, you scratch your chin and say maybe.'** Some epidemiologists say that an association with an increased risk of tens of percent might be believed if it shows up consistently in many different studies." [Emphasis added] **Therefore, in the best of scientific tradition and understanding of what constitutes a good epidemiologic evidence, WS finds that the University of Kuopio's KIHD study published by Salonen et al. (1995, 2000) has failed to pass the minimal standards outlined by some of the distinguished epidemiologists highlighted in Taube (1995).** Additional scientific criticisms of the KIHD results are presented in the main text.
[143] Guallar et al. (2002) *New England Journal of Medicine*, vol. 347, 1747-1754.
[144] See e.g., Bosma et al. (1998) *American Journal of Public Health*, vol. 88, 68-74 and Bonnet et al. (2005) *Atherosclerosis*, vol. 178, 339-344.
[145] Lakka et al. (1999) *Hypertension*, vol. 34, 51-56.
[146] Tuomainen et al. (1999) *Circulation*, vol. 100, 1274-1279.
[147] Kauhanen et al. (1999) *Arteriosclerosis, Thrombosis, and Vascular Biology*, vol. 19, 3001-3006.
[148] Nyyssonen et al. (1997) *British Medical Journal*, vol. 314, 634-638.
[149] Rissanen et al. (2003) *Journal of Nutrition*, vol. 133, 199-204.
[150] Voutilainen et al. (2000) *European Journal of Clinical Nutrition*, vol. 54, 424-428.

[151] Rissanen et al. (2003) *American Journal of Clinical Nutrition*, vol. 77, 133-138.
[152] Salonen et al. (1998) *American Journal of Epidemiology*, vol. 148, 445-451. See however a direct contradiction of this KIHD claim in the study of Ascherio et al. (2001, *Circulation*, vol. 103, 52-57) which found no significant associations of blood donation (via the lowering of body iron stores) and risk of CHD while examining the 4-year follow-up database for the 38,244 US male health professionals.
[153] Menotti et al. (1999) *European Journal of Epidemiology*, vol. 15, 507-515.
[154] See e.g., Bosma et al. (1998) *American Journal of Public Health*, vol. 88, 68-74 and Bonnet et al. (2005) *Atherosclerosis*, vol. 178, 339-344.
[155] It is important to note: although Salonen et al. (1995) pointed out that the KIHD men's hair mercury contents show a decreasing trend over the baseline examinations from 1984-1989, **there are no repeated measurements of hair mercury contents performed for the KIHD men**. (That study involved 6 annual mean values from 2.55, 2.47, 1.92, 1.51, 1.40, 1.72 ppm when those men were grouped annually according to the year of their respective examinations.) All the hair samples were collected in the original baseline examinations which were conducted in two batches: first for 1166 men between March 1984 and August 1986, and next for 1516 men between August 1986 and December 1989. **This clarification is important because even an expert and a NRC (2000) committee member like Alan Stern had apparently misunderstood the KIHD results in Salonen et al. (2000);** for example, in his review of the KIHD study he said that "Salonen et al. (2000) conducted a study ... Artery wall thickness was measured in 1014 men with an average age of 51.9 years at baseline. A second measurement was obtained 4 years later. Hair samples for measurement of Hg concentration and data on atherosclerotic risk factors were obtained during the follow-up period" (in Stern, 2005, *Environmental Research*, vol.98, 133-142). In fact, Salonen et al. (2000) made repeated measurements only for the two carotid intima-media thickness variables examined in order to quantify the 4-year progression of carotid atherosclerosis. The hair samples were collected during the baseline examinations conducted 1984-1989, as noted in Salonen et al. (2000).
[156] The highest value noted was reported in the KIHD study published by Salonen et al. (1995). However, we note the highest hair mercury content was raised to 23.3 ppm in Salonen et al. (2000) **with no apparent explanation or correction offered**.
[157] pp 13-14 of Clarkson (2002) *Environmental Health Perspectives*, vol. 110 (supplement 1), 11-23.
[158] from http://ww.scienceblog.com/community/older/2002/2002587.html (dated April 2002).
[159] Agren et al. (1988) *Lipids*, vol. 23, 924-929.
[160] The summary provided by the commercial fish oil website at http://www.oilofpisces.com/hearthealth.html (accessed November 10, 2004) further explained that: "A 15-week experiment involving 62 students was carried out to determine if a regular diet of freshwater fish affects coronary heart disease risk factors. The students were divided into three groups: a fish eating group who made no other changes to their diet; a fish eating group who also decreased their overall fat intake; and a control group (19 students) who ate a typical western diet. The special diet consisted of one fish meal a day (in addition to the regular diet) and provided about 0.25 g/day of eicosapentaenoic acid and 0.55 g/day of docosahexaenoic acid. Serum cholesterol was found to decrease in fish eaters who also decreased their lipid intake, but not in the other groups. Blood triglyceride levels decreased significantly in the fish eating groups, but not in the control group. Levels of apolipoproteins A1 and B were lowered in both fish eating groups, as was the formation of thromboxane B2 during incubation of whole blood. In the fish eating groups, the proportion of omega-3 fatty acids increased significantly in erythrocyte ghosts and platelets at the expense of omega-6 fatty acids. **The results of the study support the contention that moderate fish consumption has a protective effect against coronary heart disease**." [Emphasis added]
[161] Yoshizawa et al. (2002) *New England Journal of Medicine*, vol. 347, 1755-1760.
[162] Plante and Babo (2003) *New England Journal of Medicine*, vol. 348: 2151-2152.
[163] Vupputuri et al. (2005) *Environmental Research*, vol. 97, 195-200.
[164] Vupputuri and colleagues added that "However among those women who had not consumed fish in the previous 30 days, we observed elevated levels of systolic BP with increasing mercury exposure." It is important to point out that for those NHANES samples with low total blood mercury levels (i.e., say roughly from 0 to 4 ppb), it is known that the relative amount of methylmercury (the organic and biologically-active form of mercury associated with fish consumption) in blood declines dramatically, as shown in Figure 1 of Mahaffey et al., 2004, *Environmental Health Perspectives*, vol. 112, 562-570. This fact suggests that another source of mercury is the increasing concentration of inorganic mercury (such as those derived from dental amalgams) in blood at the low total blood mercury levels. Thus, the observed

tendency for elevated levels of systolic BP with increasing mercury exposure for tho specific group of 481 women that reported no fish consumption in the previous 30 days would require further clarification from the understanding of exposure risk to inorganic mercury.

[165] Engler et al. (2003) *Experimental Biology & Medicine*, vol. 228, 299-307.

[166] Dorea et al. (2005) *Environmental Research*, vol. 97, 209-219.

[167] Mozaffarian et al. (2004) *Circulation*, vol. 110, 368-373.

[168] In an earlier publication (Mozaffarian et al., 2003, *Circulation,* vol. 107, 1372-1377), Mozaffarian and colleagues pointed to the importance of types of fish meal consumed, finding that fried fish or fish sandwiches may not offer the same level of protection for the heart as broiled or baked fish.

[169] Dorea (2003) *Environmental Research*, vol. 92, 232-244.

[170] Ropeik (2004) *European Molecular Biology Organization Reports*, vol. 5 (special issue), S56-S60.

[171] Ropeik (2004) continues that "As argued for by Matthew Adler and others (2003), the effects of fear are harmful to health, no less than the physical harm from some toxic agent or pollutant, and these can and should be measured and economically quantified to help identify the most efficient approaches to improving public health."