MERCURY, CLIMATE AND THE FOOD WEB

A Critical Review of Claims and Assumptions in

Booth and Zeller (2005)

("Mercury, Food Webs, and Marine Mammals:
Implications of Diet and Climate Change for Human Health")

by

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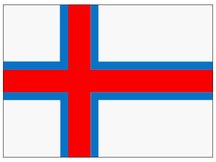

"The advantages of accepting a dogma or paradigm are only too clear. One no longer has to query the foundations of one's convictions, one enjoys the many advantages of belonging to a group that enjoys political power, one can participate in the benefits that the group provides, and one can delegate questions of responsibility and accountability to the leadership. In brief, the moment one accepts a dogma, one stops being an independent scientist."

-- Hendrik Tennekes, retired Director of Research, Royal Netherlands Meteorological Institute

"…[R]esults from ancient remains are consistent and provide evidence that humans have always been exposed to naturally occurring mercury through fish and marine mammals in their diets."

-- Scott M. Arnold, Ph.D., John Middaugh, M.D.
Section of Epidemiology
Alaska Division of Public Health

The great tragedy of science, the slaying of a beautiful theory by an ugly fact.

-- Thomas Henry Huxley (1825-1895)

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Unsupportable Claims and Assumptions in Booth and Zeller (2005)

Executive Summary

Writing in *Environmental Health Perspectives* (2005), Booth and Zeller [hereafter BZ05] embark on the highly ambitious task of applying ecosystem modeling to the difficult problem of tracing the flow of methylmercury (MeHg) - the biologically active, potentially toxic form of mercury - in the Faroe Island marine ecosystem as changing functions of both fish mortality (commercial catch rates) and climate. The paper further attempts to estimate weekly MeHg intake by the Faroese from consumption of mainly pilot whale meat and cod fish - two key sources of MeHg exposures in Faroese diets.

BZ05 displays the risk inherent in favoring computer modeling results over real world data. Such an exercise, increasingly common and problematic in climate science, often produces tenuous outcomes.

More specifically, Booth and Zeller, with their minimal "what if" modeling efforts, cobble together a grab-bag of speculative assertions, problematic statements, harm attributions and over-reaching conclusions:

> "Under present conditions and climate change scenarios, methyl mercury increased in the ecosystem, translating into increased human exposure over time. ... A large portion of the general human population exceed the TWI [Tolerable Weekly Intake] levels set by the World Health Organization (WHO; 1.6 µg/kg body weight [bw]), and they all exceed the reference dose (RfD) of 0.1 µg/kg bw/day set by the U.S. Environmental Protection Agency (EPA; equivalent to a TWI of 0.7 µg/kg bw). ... Methyl mercury will continue to be of global concern as long as there are ongoing anthropogenic inputs of mercury. Our ecosystem-scale simulations suggest that substantial reductions in mercury inputs (~50%) would be required to ensure safe exposure levels if people such as the Faroe Islanders wish to continue their cultural dietary traditions. Unfortunately, the United States in 2002 increased the release of mercury by 10% more than the previous year (U.S. EPA 2002), whereas China's emissions (~500 metric tons/year), driven primarily by coal combustion, rose by approximately 50 metric tons/year during the early 1990s (Pacyna and Pacyna 2002) and have been tracked across the Pacific Ocean to North America (UNEP 2004). Thus, anthropogenic

> pollution with mercury is a global problem that will continue to affect future generations in all regions of the world."

Again, these and other claims are largely groundless or highly doubtful because of questionable or erroneous assumptions contained in their ecosystem modeling exercises. For example, their underlying assumption that "ongoing anthropogenic inputs of mercury" are the driver for present MeHg levels in aquatic life suggests lack of familiarity with even the most basic literature.

In this review, we refrain from lengthy comments on the serious and contentious problems related to bright-line "safe" MeHg exposure guidelines offered by U.S. EPA or the World Health Organization (WHO). Additional CSPP papers critically examine the serious public health dangers resulting from the ultra-precautionary, seemingly arbitrary and scientifically unsubstantiated nature of the U.S. EPA's health advisories on exposure to historic, micro-trace levels of MeHg through fish consumption.[1]

The primary focus of this review challenges some of the modeling assumptions in BZ05 that had apparently escaped the interest or exceeded the expertise of the reviewers:

- The methylation and demethylation mechanisms for the transformation of inorganic mercury (Hg) to methylmercury (MeHg) are clearly more complex than the highly simplistic linear picture assumed in BZ05 (i.e., decreased emissions of Hg from coal-fired power plants leads to a decreased MeHg levels in pilot whale and cod in north Atlantic waters).

- A number of studies show that levels of MeHg in deep-sea fish undergo no appreciable change over time. This refutes BZ05's assumption that dramatic cuts in "anthropogenic" Hg emissions will result in dramatic reductions in marine MeHg tissue concentrations.

- The cooling temperature trends at depth for waters of the North Atlantic over the past 50 years (centered around 60ºN, near location of the Faroe Islands at 62ºN) calls into question the 0.4 to 1.0ºC per century warming in "seawater temperatures based on global climate-change scenarios" critically assumed in BZ05.

- Recent evidence shows that the atmospheric deposition of inorganic mercury (Hg) in the Faroese has been *decreasing*, rather than increasing, over the past 50 years -- while yielding no notable decrease of MeHg in either pilot whales or cod consumed in the Faroe Islands.

- The mean and range of total mercury (Hg_T) and MeHg concentrations in pilot whale from the Faroe Islands over 1977-1997 intervals show neither an alarming rise nor systematic changes in response to variations in atmospheric Hg deposition, as assumed and implied in BZ05.

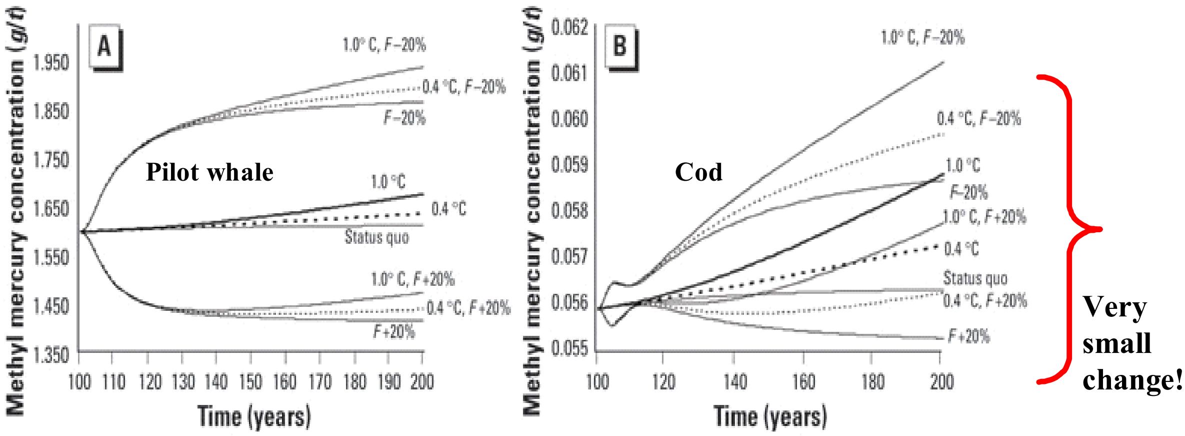
- The estimates of weekly intake of MeHg through consumption of cod and pilot whale in the model scenarios of BZ05 appear exaggerated because both the assumed (1) pilot whale intake of 12 g/person/day, and (2) a mean MeHg level of 1.6 ppm for pilot whale meat appear significantly inflated.
- Finally, serious endangerment to public health from exaggerated fears leading to unwarranted calls for restricting fish consumption (hinted in BZ05) are examined.

Preface Remarks

Even putting aside for the moment the following direct challenges to the remaining, highly questionable modeling assumptions in BZ05, their central forecasted changes in the MeHg 100-year accumulation levels for cod and pilot whale caught off the Faroe Islands are not even significant, let alone alarming (see **Fig. 1**). The modeled changes – and that's all they are – of MeHg in cod and pilot whale are at most 0.005 and 0.35 parts per million (ppm), respectively. The imagined cod changes are not likely even detectable, let alone proven to impact human health. Concerning cod consumption, the Chief Physician of the Faroe hospital system and co-author of the Faroese children study has publicly and emphatically stated that, "*Faroe Islands women do not eat mercury-tainted fish and fish consumption does not harm Faroese children. In the contrary, the fish consumption most likely is beneficial to their health.*"[2]

BZ05's prospective MeHg increases in pilot whale concentrations are not only equally

Figure 1



Effects of climate change and changing fishing mortality (*F*) on the methyl mercury concentrations in pilot whales (*Globicephala melas*; *A*) and cod (*Gadus morhua*; *B*). Note the significant difference in scale of the *y*-axis. Other species showed similar general trends. Increasing *F* decreased the load relative to the status quo scenario, whereas decreasing *F* had the opposite effect.

speculative but essentially meaningless to American and world populations due to Faroe-unique consumption practices. Additionally, Dr. Weihe further stated, "The Faroese authorities in 1998 recommended women who plan to become pregnant within months, pregnant women, and nursing women to abstain from eating pilot whale meat. The mercury concentrations in the blood of pregnant women have declined dramatically since and are now below the US-EPA limit."

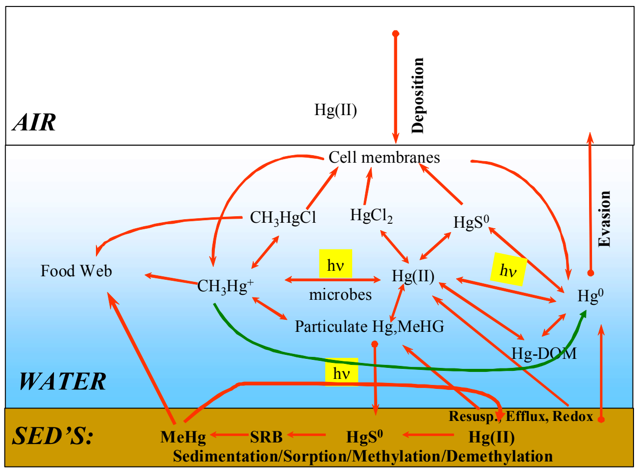
Acknowledging this, BZ05 then relates concerns for the general population of *whale* consumers. However, no epidemiological studies have raised concerns for the general Faroese population at any consumption levels. Other than direct Japanese poisoning events compounded with a cocktail of other toxic chemicals, there has never been a verified mercury poisoning resulting from consumption of marine products. Even studies by Grandjean et al. raising concerns of limited and "subtle" neurological effects on fetuses are weak, confounded with poor research design, non-transparent and non-reproducible.[3]

Clearly, mercury concentrations in pregnant women or the general population resulting from pilot whale consumption are a dominant function of the *social practices* and *consumption guidelines* of the Faroese, not U.S. or world emissions policies.

One has to wonder what's the scientific point or contribution of the paper.

Another awkward[4] conclusion drawn from BZ05, though not emphasized, is the suggestion that *increasing* fish mortality (F+20% in **Fig. 1**) promises to *reduce* the levels of MeHg ultimately accumulated in pilot whale hunted from the Faroe Islands. By contrast, *reducing* the numbers of fish taken commercially by 20% (F-20% in **Fig. 1**) would result in *higher* MeHg bio-accumulation in pilot whales from the Faroes.[5] In other words, heavier commercial fish catches in the North Atlantic emerges as an optional path to making pilot whales "safer" to eat. Again, BZ05 misses the implications of Dr. Weihe's declaration: Faroese mercury exposure is a dominant function of local practices and policies, irrespective of commercial fishing or whaling burdens.[6]

Figure 2 The Aquatic Mercury Cycle Conceptual Model



Adapted from (USGS) David Krabbenhoft's ppt presentation

Given all this alone, one has to wonder what's the *scientific* point or contribution of the paper.

Methylation of Mercury - Light versus Temperature and Other Factors

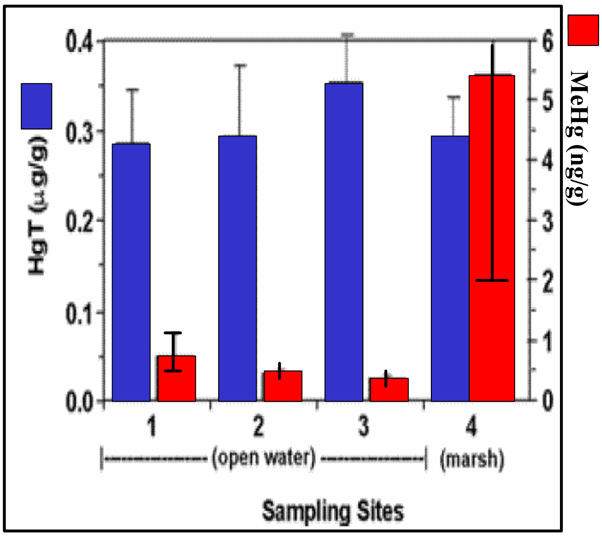
It appears that part of the reason BZ05 ignores the “increasing fish catch” and “personal responsibility” paths to “safer” Faroese whale binges is a political “twofer.” The world’s ills are often cast at the feet of anthropogenic-induced global warming and mercury “poisoning” brought on by coal-burning power plants. BZ05 “model” a combination of the two, claiming projected increases in North Atlantic water temperatures from global warming will drive increases in residual mercury concentration levels in marine life, eventually endangering health in the Faroe Islands.

To make this claim, BZ05 significantly simplify and downgrade the complex factors controlling both the methylation and demethylation of MeHg in the world’s watersheds. (**Fig. 2** only partly illustrates the extensive complexity of the aquatic mercury cycle.) The highly simplistic assumption that increasing water temperatures alone can cause more MeHg production and accumulation in marine life is likely wrong, being largely confounded by additional co-factors, including oceanic temperatures at variant depths.

For example, the research findings of Marvin-DiPasquale et al.[7] **(Fig. 3)** suggest that trace levels of MeHg in fish depend on highly complex physical, chemical, and biological factors within each unique ecosystem. More importantly, their findings evidence that despite the relatively constant level of total inorganic mercury available in *all four* (3 open water and 1 salt-marshland) sampling sites in San Pablo Bay**,** California (the four blue bars in **Fig. 3**), the production and concentration levels of MeHg were significantly enhanced only at the biologically active and organically rich marsh wetland site (the tallest red bar marked “marsh” in **Fig. 3**).

Figure 3

Methylmercury (MeHg) production does not depend on the amount of elemental mercury (Hg) available.



Microbial MeHg production in marsh wetlands is 25-50 times more than in open-water locations around San Pablo Bay area.

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

The authors stated:

> “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 that of all open-water locations...These preliminary data indicate that wetlands surrounding San

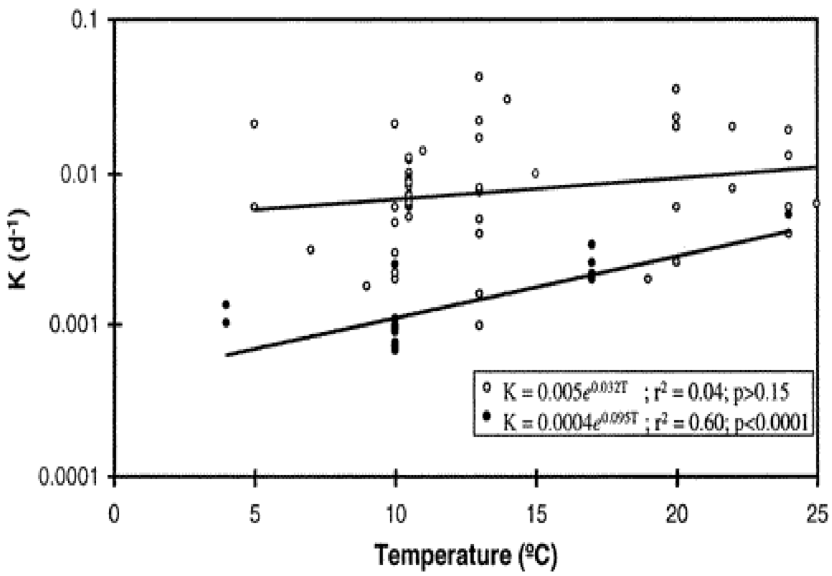
> Pablo Bay represent important zones of MeHg production, more so than similarly Hg-contaminated adjacent open-water areas. " (p. 260)

The authors further concluded that "sediment geochemistry (redox, sulfide, pH, organic content, etc.) is a much more important control on MeHg production than is the absolute total mercury concentration" (p. 266 of Marvin-DiPasquale et al., 2003, Environmental Geology, vol. 43, 260-267).

The San Pablo Bay findings add to the body of evidence suggesting that neither a warming of the Arctic ocean waters nor adding or reducing Hg atmospheric deposition from coal-fired power plants (as suggested in BA05) would measurably affect MeHg levels in north Atlantic open water ecosystems or in marine life near the Faroe Islands. To the contrary, *MeHg levels appear naturally self-limited* by specific ecosystem dynamics, water quality variables like dissolved sulfate, parameters like the population of algae and/or zooplankton, availability of nutrients and/or sunlight and so on.

Figure 4

The elimination rate (K) of MeHg (and Hg) from fish as a function of water temperature



Additional driving, dependent variables and factors affecting chemical and physical transformations of Hg into MeHg, including the following:

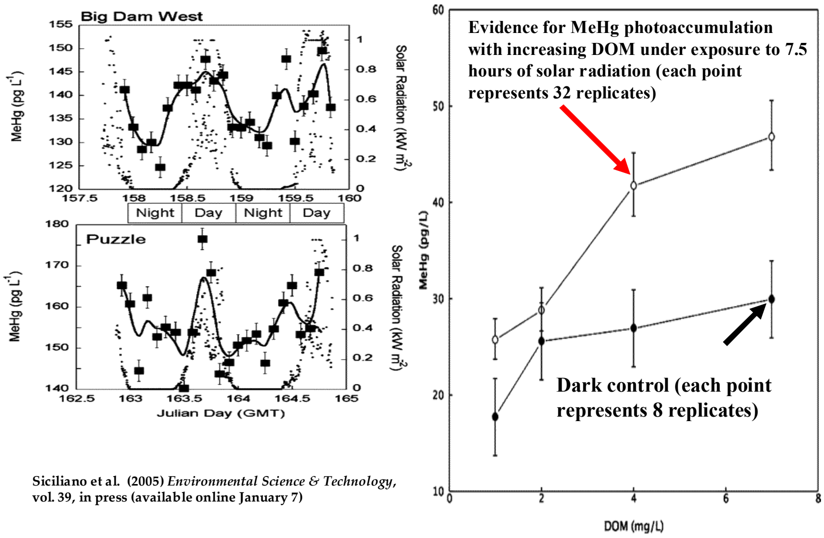
- levels of MeHg are independent of raw Hg levels (Marvin-DiPasquale et al. 2003; Paller et al. 2004; Bonzongo & Lyons 2004)
- pH and sulfate (Bonzongo & Lyons 2004)
- leaf litter inputs and microbial growth (Balogh et al. 2003)
- roles of visible light (Seller et al. 1996), UVA (Lalonde et al. 2004), diurnal MeHg and solar radiation (Siciliano et al. 2005)
- experimental treatments with sulfate (Harmon et al. 2004)
- sulfate, organic matter, and bacterial activity (Mason et al. 2005)
- water temperature and fish body weight (Trudel and Rasmussen 1997)
- algal bloom-induced biodilution of MeHg in zooplankton Daphnia (Pickhardt et al. 2002)
- dependence of MeHg on species of zooplankton (Masson & Tremblay 2003)

- seasonal cycle of MeHg before and after control flooding (St. Louis et al. 2004)
- 48 environmental variables including land use, various catchment areas and lake characteristics, lake water chemistry and fish stocks (Soneston 2003)

Also, directly contrary to the assumption in BZ05, increasing water temperature is likely to *demethylate* MeHg[8] (suggested by the increasing trend in the parameter K or demethylation rate as temperature increases in **Fig. 4**).

Figure 5

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



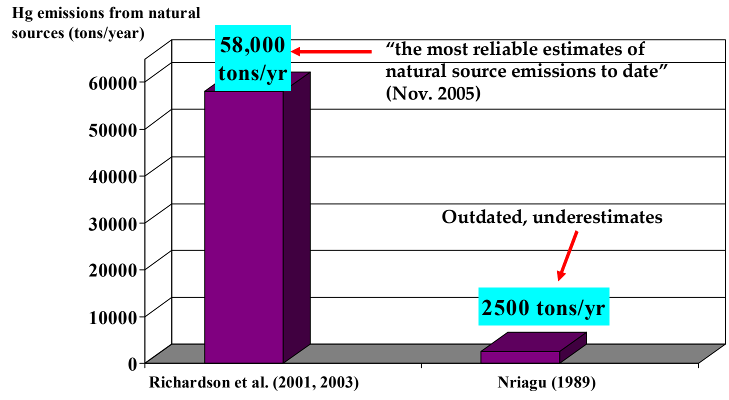
Siciliano et al. (2005) *Environmental Science & Technology*, vol. 39, in press (available online January 7)

The "temperature" effect in BZ05 may also be confused with effects of sunlight and dissolved organic matter on MeHg production. Recent research has shown that with sufficient amounts of dissolved organic matter, MeHg production is capable of being abiotically enhanced by solar radiation.[9] (**Fig. 5**). Note the day-night cycle of MeHg levels corresponding to effects of sunlight.

Returning to the question of Hg deposition and availability regulating MeHg conversion rates, Mason et al. (2005)[10] report that 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]. Once again, the key point here for BZ05 is that science-based observations reveal that *both* the production and destruction processes of MeHg ending up in marine life *do not* depend exclusively on the amount of Hg available in a water system.

Figure 6

Natural Hg Emissions Have Been Severely Underestimated



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.)

To repeat, this scientifically supports the notion (contrary to BZ05) that multiple key biological and chemical processes driving the methylation and demethylation – and the ultimate bioaccumulation of MeHg in fish tissue – *completely overwhelm* relatively insignificant contributions of elemental Hg from U.S. power plant emissions (about 40 tons in 2003).

Additionally, there exist millions of tons of naturally occurring Hg in Earth's waters, soils and sediments, ever-available for conversion into MeHg. As one example, recent research estimates that just *natural* Hg *atmospheric* contributions into the world reservoir amounts to 58,000 tons per year[11] (**Fig. 6**), compared to about 40 tons/yr from U.S. power plants or even the proximately 2,700 tons/yr *total* world wide from man-made emissions. This statistic alone renders quixotic BZ05's call for a whale-remedial, 50% reduction in man-made emissions.

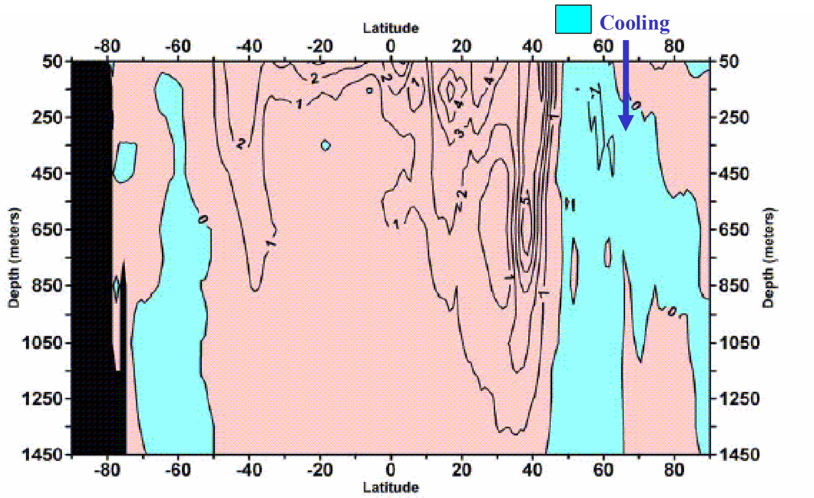
Given the dominant natural sources of Hg, it is neither difficult nor surprising to find "high" levels of MeHg in both fish and humans[12] in past centuries, at a time lacking modern, man-made emissions[13].

Ocean Temperature Trends in the North Atlantic

AAtempting to quantify effects of speculative future global warming trends on the accumulation of MeHg in pilot whales and cod, BZ05 cites the Intergovernmental Panel on Climate Change (IPCC, 2001) scenarios of 0.4 to 1.0ºC warming per century.[14] They did not specify where such warming near the Faroe Islands would occur – at the surface, ocean depth or both. However, in a poster presentation at the Fourth World Fisheries Congress session on May 4, 2004, the authors assumed in their modeling exercises more dramatic scenarios of 2 and 4ºC warming of the "sea surface temperature". These assumptions are questionable given the water temperature trends of the North Atlantic during the atmospheric warming already claimed for the later half of the 20th century.

Figure 7

Large heat loss or cooling trend over the North Atlantic Ocean centered at *60ºN* (near Faroe) from 1955-2003 -- yet Booth & Zeller (2005) adopted a warming scenario of 0.4-1.0ºC in the next century



Levitus et al. (2005) Geophysical Research Letters, vol. 32, L02604; Levitus et al. (1994) Science, vol. 266, 96-99

Levitus et al. (2005)[15] found that large regions centered on the 60ºN section of the North Atlantic Ocean underwent significant *heat loss* during the 1955-2003 periods. This *cooling* extended downward hundreds of meters below the surface (**Fig. 7**).

Levitus et al. commented:

> "It is well known that the subarctic gyre of the North Atlantic has been cooling during recent decades. Levitus [1994, 1995] documented a linear cooling trend of about 0.13ºC at 125 m depth based on Ocean Weather Station 'C' data during 1948-1995 with quasi-decadal oscillations of about 2ºC range [an important feature clearly not accounted for by BZ05's modeling effort]. Dickson et al. [2002] documented the cooling and freshening of the deep waters of the Labrador Sea since the early-1960s which has resulted in the cooling of the deep waters of the entire subarctic gyre of the North Atlantic."

Atmospheric Deposition

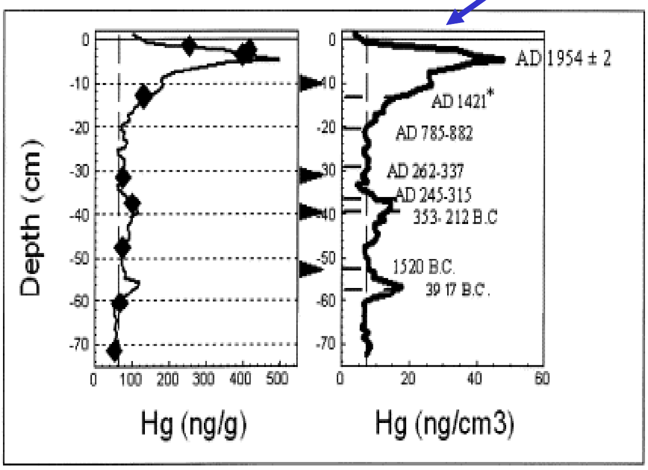
BZ05 assumptions regarding atmospheric emissions and loading are highly suspect. Broad-brush policy prescriptions such as, "[B]ase inflow rates of mercury input into the environment would need to be reduced by approximately 50% to ensure levels of intake below the WHO TWI levels, given the current levels of whale consumption [by the Faroe Islanders]" tend to ignored both the complex science of methylation and demethylation of MeHg (discussed above) and well documented empirical difficulties linking patterns and tendencies of atmospheric deposition of Hg with those of anthropogenic emission sources.

Shotyk et al. [16] recently examined Hg concentrations in layered peat bog sediments at Myrarner, Faroes (**Fig. 8**). They reported that mercury atmospheric deposition rates had actually *declined* by about 50% between 1954 (at the deduced rate of 34 µg/m^2/year) and 1998 (16 µg/m^2/year).

Figure 8

Estimates of Hg atmospheric deposition at the Faroes over the past 5000 years. During a period of increasing anthropogenic Hg emissions, atmospheric deposition rates of Hg have been decreasing for the past 50 years.

Peat bog core from Myrarnar, Faroe Islands



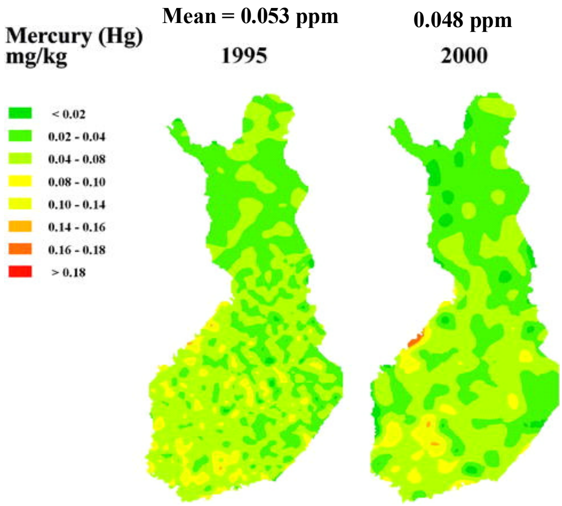
If such a persistent depositional decline over half a century yielded no noticeable response-drop of MeHg levels in pilot whale or cod in the area of the Faroe Islands, how could BZ05 rationally expect "substantial reductions in mercury inputs" by an additional 50% to do so in the future?

The point that large decreases in anthropogenic emissions of mercury may occur without a corresponding systematic reduction in the actual mercury deposited from the atmosphere seems reinforced (**Fig. 9**) by the work of Poikolainen et al. (2004)[17]. They found no clear or systematic decrease in mercury deposited in Finland between 1995 and

2000, despite a significant 41% decline in man-made mercury emissions during the 1990s in Finland. (It is significant to note that Poikolainen et al. *were* able to demonstrate corresponding decreases in emissions and deposition for other trace heavy metals like lead and cadmium.)

They reported, "Anthropogenic emissions and total deposition of Hg decreased during the 1990s throughout the whole of 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. "

Figure 9 **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)**



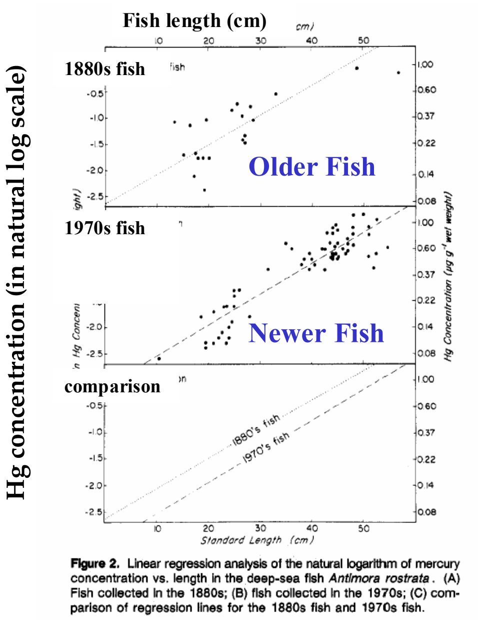
Poikolainen et al. (2004) Arctic, Antarctic, and Alpine Research, vol. 36, 292-297

Accumulation of Mercury in Marine life and Aquatic Systems

Apart from the virtual world of their creative model simulations, BZ05 offer no empirical evidence correlating fish tissue mercury levels to either warmer oceans or increased presence of Hg in the environment, either from smaller man-made or overwhelming large natural sources. However, there is ample real-world evidence to the contrary.

For example, Barber et al. [18] reported that although one can find a clear increase of mercury concentration in the tissue of the deep sea fish (blue hake) caught from western Atlantic waters as the size of the fish increases, one can hardly see any significant changes in the fish tissue mercury-size relation for fish samples caught in 1880s when compared to similar, modern samples caught in the 1970s (**Fig.**

Figure 10 **No evidence of increasing trend or any change in Hg of deep-sea fish (blue hake) over nearly a century**



"This result supports the idea that the relatively high concentration of mercury found in marine fish that inhabit the surface and deep waters of the open ocean result from natural processes, not 20th century industrial pollution."

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

10). This research clearly suggests that mercury concentration in marine life is *not likely to be changed or modified* by any altered amounts of inorganic Hg sources (either anthropogenic or natural). This is why it is *factually misleading* for BZ05 to propose that a 50% reduction in man-made mercury emissions is adequate to make pilot whale "safe" for Faroese consumption.

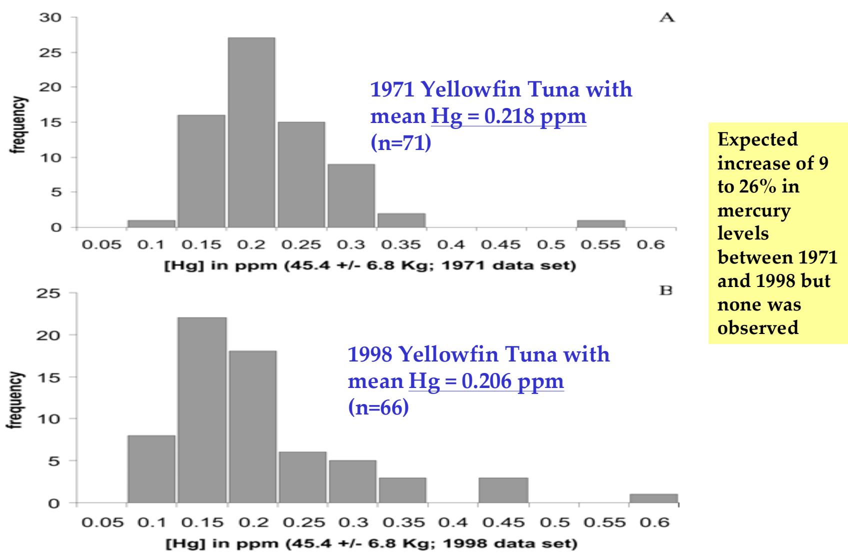
In contradiction of BZ05, Barber et al. report:

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

Similar results have been reported from the Pacific Ocean (**Fig. 11**). Princeton researchers[19] found no increase in the mercury levels of Yellowfin tuna caught in 1998 relative to a similar cohort caught in 1971. The theoretical expectation (similar in BZ05) was that the methylmercury concentration "should have increased by 9 to 26%" over the interval "if methylation occurred in the mixed layer or in the thermocline [of the Pacific oceans]." The theory was *not* proven.

Figure 11

No increase **in mercury levels for Yellowfin tuna caught in 1998 relative to a similar sample caught in 1971**



Kraepiel et al., 2003, *Environmental Science & Technology*, vol. 37, 5551-5558

We further note that Chinese researchers[20] have estimated that China's mercury emissions from coal combustion are increasing at the rate of 5% per year (available data from 1978 through 1995), which is consistent with the theoretical expectation of an increase in the amount of methylmercury in the waters of the Pacific Ocean *if* the Hg-to-

MeHg conversion process is sensitive to industrial emissions. To the contrary, Kraepiel et al. (2003) clearly concluded that *"[s]uch an increase is statistically inconsistent with the constant mercury concentrations measured in tuna. We conclude tentatively that mercury methylation in the oceans occurs in deep waters or in sediments*." [Emphasis added] They further stated:

> "Our findings that the concentration of mercury in tuna...has not changed over a period of time during which anthropogenic mercury inputs...have increased supports the idea that the source of methylmercury in tuna is not in surface waters. [This] provided *prima facie evidence that this concentration is not responding to anthropogenic emissions irrespective of the mechanisms by which mercury is methylated in the oceans and accumulated in tuna*" [or, we might add, in cod or pilot whales – CSPP].[21] [Emphasis added]

Similar findings have been reported by Greenfield et al.[22] and Yamaguchi et al.[23]

This is why it is unlikely that relatively small man-made sources of mercury emissions can either overwhelm or directly alter the natural cycling of mercury in the environment and biosphere. In other words, these and similar findings pose the question, if man did not put the mercury in the fish, how can he get it out with the 50% reductions [or *any* quantity reductions] offered in BZ05? Can one regulate or legislate an overturning of such large and persistent natural system dynamics?

Such real-world findings reinforce our concerns that BZ05's expectation and assumption of a direct, linear correspondence between changing levels of anthropogenic mercury emissions and MeHg levels in ocean fish are simplistic, misleading and seriously inappropriate for policy formulation.[24]

MeHg and Consumption of Pilot Whale in the Faroe Islands

Generally, in BZ05 the putative threat posed by a small increase in methyl mercury levels in cod and pilot whales assumes that Faroe Islanders *100 years from now* will still have the same dietary consumption patterns of cod and whale meat as today and will not change even in the face of an increased threat despite undoubtedly much clearer understanding of it then than we now have. This assumption is contradicted by the significant and immediate reduction in whale consumption by pregnant Faroese women in response to a perceived threat at current mercury levels with far less clear understanding than will likely be available 100 years from now.

More importantly, available literature calls into question BZ05 assumptions regarding mercury levels in pilot whales consumed in the Faroe Islands.

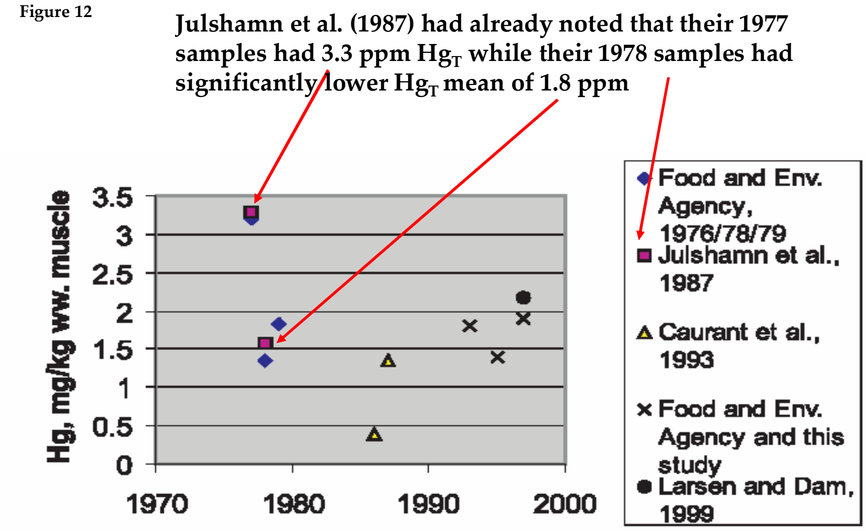
The pioneering research work by Julshamn et al. (1987)[25] (**Fig. 12**) outlined a few somewhat surprising findings about the available measurements of total mercury (Hg_T)

and methylmercury (MeHg) concentrations in tissue of pilot whale caught off the Faroe Islands from 1977-1997:

(1) The mean and range of Hg_T and MeHg values are neither rising alarmingly nor changing systematically in response to variation in atmospheric Hg deposition inputs (i.e., as erroneously assumed in BZ05 while formulating implications from their ecosystem modeling exercises).

(2) Large variability of mercury concentrations in pilot whale muscle from year to year is not surprising as the local catches may depend on sub-populations of the migrating whales.

Figure 12



Dam and Bloch (2000) Marine Pollution Bulletin, vol. 40, 1090-1099; Julshamn et al. (1987) Science of the Total Environment, vol. 65, 53-62

(3) Julshamn et al. (1987)[26] found through direct measurements[27] that MeHg for pilot whale ranged from 0.06 to 1.72 ppm - about 20-90% of total Hg (Hg_T). The arithmetic average of 18 MeHg measurements was 0.77 ppm (see discussion for **Fig. 13** for the implications). These levels significantly differ from the mean value of 1.6 ppm adopted in BZ05. The lowest and highest values of 1.41 and 1.92 ppm reported in BZ05 appears to come from Caurant et al. (1996)[28], based on July 1986, October and November 1987 catches. BZ05 failed to caution its readers that the highest MeHg value of 1.92 ppm quoted in Caurant are actually based on the sample of only two measurements which are clearly not statistically robust or meaningful.

Thus, there exist two significant problems in the estimates of the weekly intake levels of MeHg for the Faroese adult population (**Fig. 13**):

First, BZ05's assumed mean MeHg level of 1.6 ppm for the pilot whale meat (applied to their estimates of weekly MeHg consumer intake of 12 g/person/day) may be exaggerated compared to the 0.77 ppm mean derived from measurements of Julshamn et al. (1987) previously discussed.

The impact of this unchecked assumption can be rather dramatically shown by lowering the weekly MeHg intake to the level of about 92 µg rather than the ~162 µg estimated in BZ05. At this lower level, assuming mean pilot whale MeHg values to be 0.77 ppm, it then becomes clear that most of the general adult population in the Faroe Island would be

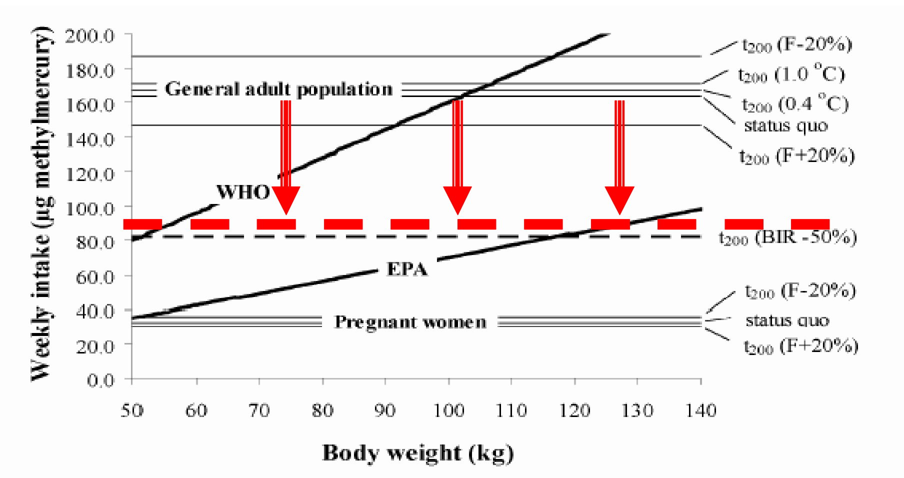
below the World Health Organization's "safe" intake level at most adult body weight categories (downward red arrows in **Fig. 13**). This adjustment paints BZ05's major conclusions and claims to be both doubtful and contentious in nature.

Next, it should be noted that the assumed daily intake of 12 g of pilot whale by Faroese adults is based on an old survey conducted for the 1981-1982 consumption pattern and habit in the Faroe Islands. Also, the Faroese Food and Environmental Agency has issued general advisories for avoiding or restricting pilot whale organs, meat, and blubber as early as the late 1970s and specifically to no more than 5-7 g of pilot whale meat in 1989 (pp 202-203 of Weihe et al. 2005[29]).

Additionally, a 2000-2001 survey found that pregnant women in the Faroes consumed an average of 1.45 g per day of pilot whale meat[30] (hence the low weekly MeHg intake levels of 30-40 μg shown in **Fig. 13**). In another estimate related to births of 1152 Faroese children from December 1997 through February 2000, Weihe et al. (2003) estimated (through analyses of dietary questionnaires) total mean maternal intake of 326 g during pregnancy, yielding a daily pilot whale mean consumption of about 1.16 g. Again, BZ05 assumed an inflated daily intake of 12 g.

Figure 13

BZ05 estimates of weekly intake levels of MeHg for Faroese adult population are likely exaggerated on two counts: (1) assumed whale mean MeHg burden Level of 1.6 ppm and (2) assumed human whale meat intake of 12 g/person/day



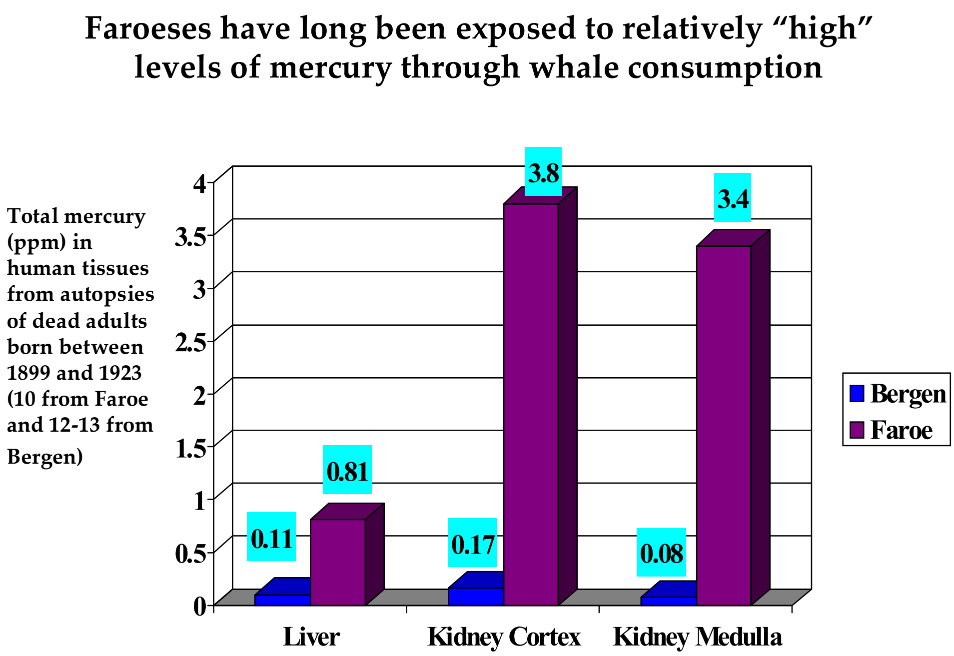
A drastic lowering of adult's weekly MeHg intake from ~162 μ g to 92 μg if a mean MeHg of 0.77 ppm in pilot whale muscle is adopted instead

Thus, it seems reasonable to suggest that with both the relatively high level of awareness about MeHg in pilot whale and the documented drastic reduction of pilot whale meat consumption by pregnant women, even the general adult population in the Faroe Island may consume less than the 12 g/day assumed in BZ05. This would also mean a lower total weekly intake of MeHg, further weakening conclusions in BZ05.

This brings us to consider yet another question for BZ05. If modeling assumptions regarding future health concerns in the Faros are valid, then where is the epidemic of health problems from past exposure? Faroeses have long been exposed to "high" levels of mercury (plus a synergistic cocktail of other toxic chemicals such as PCBs and DDT) through traditional whale consumption.

Studies comparing Faroeses body burdens of mercury with adults from Bergen, Norway revealed startling differences (**Fig. 14**). Mercury contents in kidney and liver tissues measured in the autopsies[31] of the 10 Faroese bodies were found to be about 10-40 times higher than those measured in 12-13 bodies from Bergen, Norway. And yet, the authors (Andersen et al. 1987[32]; Julshamn et al.1989[33]) of this pioneering research observed:

Figure 14



> "It is remarkable that all whales caught have been consumed by the population of the Faroe Islands for centuries without any signs of poisoning or reduced life span being recorded in the population."

As an important side note, we have elsewhere examined and clarified in detail why the extremely conservative "safety" MeHg thresholds set by USEPA and WHO are not only scientifically unsupportable but potentially harmful in generating fear and confusion leading to drastic dietary reductions for fish intake.[34]

Potential Harm to Human Health from Restricting Fish Consumption

Our final and most serious concern centers on the one-sided framing of the issues surrounding trace levels of methylmercury contained in fish (or marine mammals) taken from the world's oceans. Apparently, BZ05 have chosen to highlight only reputed health risks (like impacts on child cognitive abilities) related to MeHg exposures, neglecting balance from a host of health benefits associated with the consumption of highly nutritious fish.

Recent CSPP reports, (1) ***Making Sense of State Fish Advisories***[35], (2) ***Fish, Mercury and Cardiac Health***[36], (3) ***How Safe Are We From the Fish We Eat?***[37], and (4) ***Doing Harm – The Mercury Scare***[38] discuss the negative health concerns briefly mentioned and referenced in BZ05, clarifying risks and benefits of fish consumption.

Herein, we update with additional specific notices on the beneficial role of fish consumption for omega-3 polyunsaturated fatty acids (hereafter variously labeled as PUFA, DHA or EPA).

On Breast Cancer:
Gago-Dominguez et al. (2003)[39] noted:

> "Ecological studies support the notion that high consumption of fish is associated with low incidence of breast cancer. Coastal- and rural-dwelling Japanese and Eskimos, who traditionally consume large quantities of marine n-3 fatty acids, have low breast cancer rates. We investigated the effects of individual fatty acids on breast cancer in a prospective study of 35,298 Singapore Chinese women aged 45-74 ... Our observations may have practical implications in prevention and treatment strategies for breast cancer, suggesting that an intake level of approximately 40 g of fish/shellfish per day can reduce breast cancer risk by 25%. In the present study, the positive association between the family history of breast cancer and personal risk was especially pronounced among women with low intake of fish"

Terry et al. (2003)[40] reported:

> "One study in Japan found that women who consumed ≥ 5 servings of undried or dried fish/wk had a 10% or 20% lower risk [of breast cancer], respectively, than did women who consumed ≤ 1 serving/wk. In a Norwegian study, women who consumed ≥ 5 servings of poached fish/month had a 30% lower risk than did those who ate poached fish ≤ 2 times/month ..."

On Prostate Cancer:
Terry et al. (2001)[41] suggested:

> "Consumption of fatty fish might reduce the risk of prostate cancer ... We studied the association between fish consumption and prostate cancer in a population-based prospective cohort of 6272 Swedish men. During 30 years of follow-up, men who ate no fish had a two-fold to three-fold higher frequency of prostate cancer than those who ate moderate or high amounts did. Our results suggest that fish consumption could be associated with decreased risk of prostate cancer."

On Alzheimer Disease (AD):
Friedland et al. (2003)[42] commented:

> "Attention was drawn to dietary influences on health decades ago by observation of the low risk for cardiovascular disease in Japanese and also in the Inuit of Greenland, populations with high levels of consumption of fish. Hendrie and colleagues have reported a low risk of AD in the Cree in northeastern Canada, another population with high fish intake. We have found a high prevalence of AD in Arab population in Israel with a low rate of fish consumption."

Morris et al. (2003) found:

> "A primary component of membrane phospholipids in the brain is the n-3 [PUFA], docosahexaenoic acid (DHA; 22: 6n-3). High levels of DHA (Docosahexaenoic Acid) are found in the more metabolically active areas of the brain, including the cerebral cortex ... Fish is a direct source of preformed DHA. Consumption of the n-3 [PUFAs] and fish was associated with reduced risk of incident Alzheimer disease in this large prospective study [in a biracial community Chicago with 815 participants aged 65-94 years]. Persons who consumed at least 1 fish meal per week had 60% less risk of Alzheimer disease than did persons who rarely or never ate fish. Total intake of n-3 [PUFAs] was associated with reduced risk of [AD], as was intake of [DHA]. We did not observe a protective benefit from EPA (Eicosapentaenoic Acid); however, the range of intake was low [i.e., < 0.03 g/d], and we cannot rule out an effect at higher dose levels obtained from cold-water fatty fish or fish oil supplements. This study supports the protective associations found by 2 other epidemiologic studies."

On Adult Cognitive Performance:
New results by Kalmijn et al. (2004) reported that:

> "The notion that dietary factors influence cognitive functions and subsequently the risk of dementia is growing. Besides the observation that antioxidant intake is associated with a lower risk of dementia, saturated fat and cholesterol intake were found to be associated with a higher risk of dementia. ... Marine omega-3 [PUFAs] was inversely related to the risk of impaired overall cognitive function and speed [based on the Doetinchem (Netherlands) Cohort Study with 1613 men and women aged 45 to 70 years]. Results for fatty fish consumption were similarly inverse. Higher dietary cholesterol intake was significantly associated with an increased risk of impaired memory and flexibility. The observed association will probably have no functional significance yet, because participants were middle aged and had only subtle impairments. Various studies showed that subjects with mild cognitive impairment progressed to dementia or AD at a rate of 10 to 15% per year, and the risk of dementia was higher when

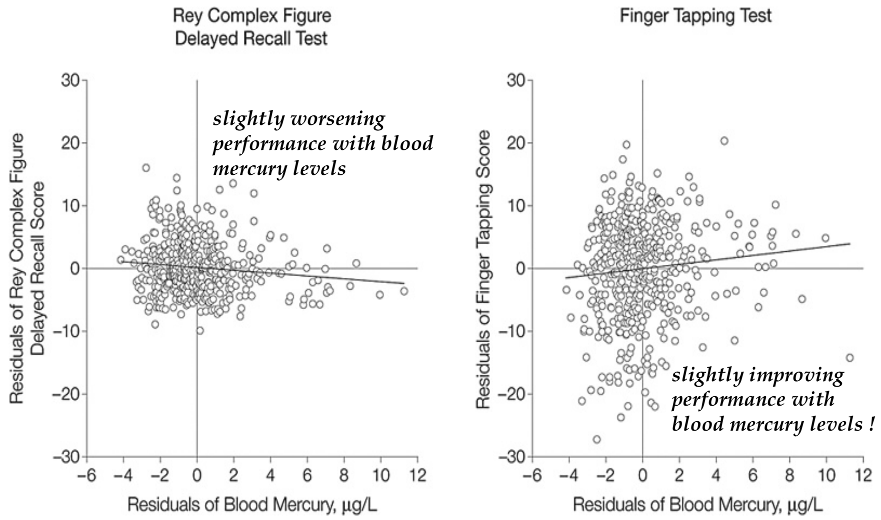
rate of decline was higher. Therefore the association with fatty acids is expected to become clinically important at old age, which is indeed suggested by some previous studies on fatty acids and dementia.”

Fig. 15 shows recent results from the first study attempting to assess potential associations of mercury exposure with adverse neurobehavioral outcomes in older adults within the general U.S. population (2005)[43]

Some 474 participants were randomly selected from a pool of 1140 residents (aged 50 to 70 years) in the Baltimore Memory Study in order to confirm potential associations of adverse neurobehavorial performance as functions of mercury levels measured in their blood. As shown in **Fig. 15**, the statistical nature of one *positive* detection could easily be discounted by an associated *negative* detection. Thus Weil et al. (2005) concluded:

> “Overall, the data do not provide strong evidence that blood mercury levels are associated with worse neurobehavorial performance in [the] population of older urban adults [from their Baltimore Memory Study].”

Figure 15 **“Overall, the [Baltimore Memory Study] data do not provide strong evidence that blood mercury levels are associated with worse neurobehavioral performance in this population of older urban adults.”**



The above health studies offer challenge to BZ05 claims that:

> “Methyl mercury affects human health as a result of direct discharges and atmospheric transport [none of which are modeled in BZ05]. This pollutant is of particular concern to indigenous people of the Arctic, who often rely heavily on marine resources, and especially marine mammals, for part of their traditional diets. For example, in Greenland, approximately 43% of blood samples taken from indigenous women of reproductive age had blood mercury levels exceeding guidelines (UNEP 2003). However, increasingly pollutants [sic.] found in marine resources, such as mercury, PCBs, and dioxins, are of growing concern also to westernized societies, given the growing demand for and consumption of seafood. This is illustrated by the advisory regarding seafood consumption by pregnant women issued by the U.S. Food and Drug Administration in March 2004 (U.S. FDA 2004) [factually, this is a *joint* advisory with USEPA].”

It is unfortunate that BZ05 fails to point out that the study by Weihe et al. (2002) actually found Greenland's Inuit children "did not appear to be clinically adversely affected by the exposure." And "neuropsychological tests failed in revealing clear mercury-related deficits."[44] Again, this suggests the arbitrary nature of most mercury guidelines.

A final note of caution to potential readers of BZ05 (and anyone concerned about the public health of native populations of the Arctic) from scientists with the Alaska Division of Public Health, Epidemiology Section (Arnold et al. 2005[45]). They warn about the undue restrictions on fish consumption issued by USEPA and FDA (complicit in BZ05):

> "Both the U.S. Environmental Protection Agency (EPA) and the U.S. Food and Drug Administration (FDA) have issued national fish consumption advisories that recommend women of childbearing age restrict their consumption of fish to avoid excessive exposure to methylmercury. These advisories were issued on the basis of methylmercury levels found in fish in specific locations across the country and estimates of dietary consumption, and they were issued across the country regardless of actual levels of mercury found among human populations. Thus, we question the wisdom and the validity of the scientific basis of these advisories. First, **by ignoring the benefits associated with consuming fish and the potentially decreasing fish consumption among women of childbearing age, these advisories may violate the ethical principles of beneficence: do not harm, and maximize possible benefits and minimize possible risks** [emphasis added]. Second, the advisories ignore the evolution of public health's ability to measure actual exposure to environmental contaminants among specific populations."

We question the wisdom and the validity of the scientific basis of these advisories. Highly restrictive generic fish consumption advisories such as the ones issued by the EPA and the FDA, can cause harm by unnecessarily warning people not to consume fish. Alaska's public health response to EPA/FDA advisories has been to recommend unrestricted consumption of fish caught in Alaska waters.

Arnold et al. (2005) specifically clarified and warned of potential public health crisis from scientifically unsubstantiated calls for restricting fish intakes for Alaskan natives and other Americans:

> "Although national fish advisories are not intended as research, they overemphasize risks and undervalue the benefits of consumption. Highly restrictive generic fish consumption advisories such as the ones issued by the EPA and the FDA, can cause harm by unnecessarily warning people not to consume fish. Among Alaska Natives who rely heavily on these foods for their nutritional, spiritual, and cultural health, the results can be disastrous. ... Many Alaskans have no readily available alternative to fish. In fact, a large number of Alaskans rely on locally caught fish as their primary protein source. Thus, for Alaska public health officials, the EPA/FDA advisories have been particularly problematic. *Even though available data show methylmercury concentrations in the most frequently consumed Alaska fish (e.g., chinook, coho, sockeye, chum, and pink salmon) are among the lowest of all fish species (average <0.05 mg/kg [or ppm]), many Alaskans, particularly Alaska Natives, have begun questioning*

the safety of their traditional diets. [Emphasis added] The adverse effects on public health in communities that have moved away from traditional foods have been well documented. In Alaska, there is now evidence that Alaska Natives are replacing their traditional diets with foods that are less healthy, and Alaska Natives are experiencing a significant increase in the prevalence of diabetes and overweight/obesity. Additionally, many Alaskans have serious problems with alcohol use and lack physical exercise, conditions that may be partially attributed to the abandonment of a traditional diet and lifestyle. Alaska's public health response to EPA/FDA advisories has been to recommend unrestricted consumption of fish caught in Alaska waters. Furthermore, a biomonitoring program has been implemented to determine actual exposure levels of environmental contaminants among concerned populations."

Conclusions

This brief criticism of Booth and Zeller (2005) should serve as a serious caution for interpreting results and claims even from peer-reviewed literature.

BZ05, at best, appears scientifically inaccurate and premature. The paper immodestly attempts to fold complex sciences surrounding (1) the cycling of micro-trace mercury in aquatic systems, (2) fishing dynamics and pressures in world oceans, (3) unique dietary practices in the Faroe Islands, (4) human epidemiology and (5) global warming into one rather simplistic, and yet sweeping synthesis.

> *"Shoddy science, bad peer review and a failure of the science community to demand high standards is not the best recipe for helping science to contribute effectively to policy."*

In other words, the thesis of BZ05 rests on a litany of assumptions and estimates of highly variable parameters linked by an unproven modeling attempt for exceedingly complex and poorly understood phenomena. The margin for error is vast while the forecasted increase in MeHg is small.

Speculation of a remote possibility for a minor increase in health risk for the small population of a few remote islands 100 years from now when an easy, obvious solution is already in effect is a subject ripe for serious scientific skepticism. Further, that a rise in fish methyl mercury levels can tenuously be linked to global warming is simply beyond present belief. If nothing else, BZ05 may represent another striking example of just how far some will flush global warming concerns from any moorings of reality.

BZ05 may represent another striking example of just how far some will flush global warming concerns from any moorings of reality.

We've attempted to document from the current literature the numerous weaknesses and shortcomings in this approach. While there are certain aspects in BZ05 that may appear intrinsically scientific and of interest, its analysis and conclusions are unconvincing and unsupportable. Worse, they encourage public policy responses *already doing harm*, not good.

Given its many problems, it is amazing that BZ05 survived peer review at all. Whether *Environmental Health Perspectives* has a quality control problem or an inability to question analysis that may be politically inconvenient in the present environment, publication of BZ05 certainly raises unpleasant questions.

As one scientist has observed in another context, "Shoddy science, bad peer review and a failure of the science community to demand high standards is not the best recipe for helping science to contribute effectively to policy. Those who perpetuate such claims...are either ill-informed or dishonest."[46]

We leave it to readers to weigh the major shortcomings in BZ05 when judging its usefulness or value in policy formulation.

Robert Ferguson has 26 years of Capitol Hill experience, having worked in both the House and Senate. He served in the House Republican Study Committee, the Senate Republican Policy Committee; as Chief of Staff to Congressman Jack Fields (R-TX) from 1981-1997, Chief of Staff to Congressman John E. Peterson (R-PA) from 1997-2002 and Chief of Staff to Congressman Rick Renzi (R-AZ) in 2002. He also served as the Executive Director of the Center for Science and Public Policy. He has considerable policy experience in climate change science, mercury science, energy and mining, forests and resources, clean air and the environment. His undergraduate and advanced degrees were taken at Brigham Young University and George Washington University, respectively.

The views and opinions expressed in this paper are those of the author, and not necessarily those of the Center for Science and Public Policy.

End Notes

[1] See the CSPP mercury library at: (http://ff.org/centers/csspp/misc/library/mercury.html)
[2] Pal Weihe, Open Letter to the Boston Herald, Feb-2-04.
[3] See Doing Harm – The Mercury Scare (http://ff.org/centers/csspp/pdf/doingharm_072006.pdf)
[4] A level of awkwardness not dissimilar, for example, to the statistical conclusions reached in Weihe et al. (2003, In the *AMAP Greenland and the Faroe Islands 1997-2001* report issued by the Danish Environmental Protection Agency, pp. 135-212) that (1) more alcohol leads to a higher gestational age for the Faroese pregnant women (p. 151) and (2) women who consumed more whale meat during pregnancy are more likely to undergo abortion in late pregnancy, during the 16-28 weeks of gestation (p. 153). This is why we conclude that modeling results from Booth and Zeller (2005) cannot be extrapolated (or rather it is meaningless) to call for increasing fishing pressure in order to try to drive a reduction of MeHg accumulation in pilot whale caught off the Faroe Islands.
[5] BZ05 does not appear to define the Key Parameter "Fishing Mortality" (F) nor do they explain why or how it should affect methyl mercury levels in different organisms. Also no probability estimate is made for

either an increase or decrease in F. The authors simply simulate 20% increases and decreases and then for no explained reason concentrate discussion on the one that produces increases in methyl mercury in cod and pilot whales.

[6]It may be of interest to note that the annual catch of pilot whale in the Faroes is notably a sustainable practice. A recent report by the North Atlantic Marine Mammal Commission (NAMMCO) suggests that "Pilot whales are likely one of the most abundant odontocetes in the North Atlantic. The harvesting of pilot whale that continues today in the Faroe Islands has proven sustainable over a period of more than 300 years, and in 1997, NAMMCO concluded that the drive hunt [of pilot whale] was sustainable. The population off Newfoundland is likely recovering after a period of excessive harvesting.")

[7] Marvin-DiPasquale et al., 2003, Environmental Geology, vol. 43, 260-267.

[8] Trudel and Rasmussen (1997) Environmental Science and Technology, vol. 31, 1716-1722.

[9] Siciliano et al. (2005) Environmental Science and Technology, vol. 39, in press.

[10] Mason et al. (2005) Environmental Science and Technology, vol. 39, A14-A22.

[11] 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.)

[12] A comparison of MeHg levels in ancient and modern Alaskans found a higher mean level in 550-year old mummies.

Exposure to MeHg in Alaska: Today versus 550 years ago

Today's distribution of pregnant women (n=177) in Alaska with a mean of 0.7 ppm (or median value of 0.47 ppm)

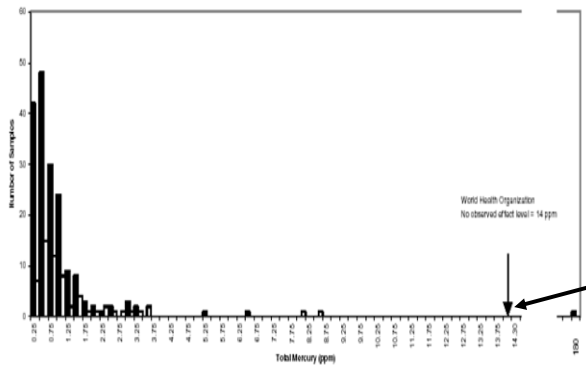
Figure 20. The Frequency Distribution of the Total Mercury Concentrations Detected in the Hair of Pregnant Women (n = 177; black bars; median = 0.47 ppm) Statewide and Nonpregnant Women of Childbearing Age (n = 60; white bars; median = 0.63 ppm) Participating in the Statewide Maternal Hair Mercury Biomonitoring Program.

Compare this 0.7 ppm to the mean level of MeHg in 550-year old Aleutian mummies:

1.2 ppm (mean of 4 adults)

1.44 ppm (mean of 4 infants)

one with MeHg as high as 4.6 ppm!



Note: the WHO "no observed effect" level for MeHg exposure is *14 ppm*!

State of Alaska Epidemiology Bulletin No. 11 (December 2, 2004)

[13]Mercury in Humans – Historical Evidence
Archeological data of ancient human hair demonstrate past exposure:
(1) *Barrow, Alaska*: total mercury 4.8 ppm in a mummy 25 years of age at death, and 1.2 ppm in a 50-year-old mummy, both dating back to 1460 A.D. (Toribara et al. 1984).
(2) *Canada*: 5th and 12th century methylmercury levels ranged from 0.8 ppm to 3.7 ppm with a mean level of 1.7 ppm among eight individuals (Wheatley et al. 1988).
(3) *Greenland*: the mean total hair mercury level of 15th century mummies was 3.1 ppm among six adults and 10.0 ppm among 2 children (Hansen et al. 1989).
(4) *Kodiak, Alaska*: mean total mercury concentration in sixteen human hair samples dated 1170 A.D. to 1660 A.D. in was 1.33 ppm (Egeland et al. 1999).
(5) *Alaska's Aleutian Islands*: dating to about 1450 A.D. , average level of methylmercury in 4 adults was 1.2 ppm and in 4 infants 1.4 ppm.

[14] Another question unanswered in BZ05 is if a projected 1°C rise in temperature can be expected to produce such a detrimental increase in methyl mercury in high northern latitudes why is it not already evident in the vastly warmer tropics?
[15] Levitus et al. (2005) Geophysical Research Letters, vol. 32, L02604.
[16] Shotyk et al. (2005) Geochimica et Cosmochimica Acta, vol. 69, 1-17.
[17] 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 not only mercury but also other trace metals deposited from the atmosphere.
[18] Barber et al. (1984) Environmental Science and Technology, vol. 18 (no. 7), 552-555.
[19] Kraepiel et al. (2003, Environmental Science & Technology, vol. 37, 5551-5558)
[20] Zhang et al. (2002, Ambio, vol. 31, 482-484)
[21] BZ05 attempts (p. 525) to reinterpret away these findings, and their conclusions are speculative and unconvincing in the face of other, similar findings for both ocean and fresh water aquatic systems.
[22] Greenfield et al. (2004) Science of the Total Environment, in press.
[23] Yamaguchi et al., 2003, Chemosphere, vol. 50, 265-273
[24] See also Mercury science findings in People of the State of California vs. Tri-Union Seafoods, (http://ff.org/centers/csspp/pdf/20060516.pdf)
[25] Julshamn et al. (1987) Science of the Total Environment, vol. 65, 53-62.
[26] Julshamn et al. (1987) Science of the Total Environment, vol. 65, 53-62.
[27] It is important to note that many reported high values for "mercury" in pilot whales, like those reported in Dam and Bloch (2000, Marine Pollution Bulletin, vol. 40, 1090-1099), are actually measurements for total mercury (Hg_T) rather than MeHg values. Relatively high percentages of MeHg are often assumed, but not actually proven; and that assumption can certainly be shown as wrong for the well-known, low relative amount of MeHg (only 3-12% of Hg_T) detected in livers and kidneys of marine mammals in the Arctic (Wagemann et al., 1998, Science of the Total Environment, vol. 218, 19-31). Professor Kare Julshamn of Norway's National Institute of Nutrition and Seafood Research, as recent as January 4, 2005, commented that "There is, however, still little knowledge on what is the amount of total mercury in different fish [we add, marine mammals] samples that in fact is organic mercury [for example, MeHg]." (http://www.nifes.no/mercury_methyl_nfo.html)
[28] Caurant et al. (1996) Science of the Total Environment, vol. 186, 95-104.
[29] Weihe et al. (2005) Environmental Research, vol. 97, 201-208.
[30] Weihe et al. (2003) Chapter 6 in *AMAP Greenland and the Faroe Islands 1997-2001* report issued by the Danish Environmental Protection Agency (eds. Bente Deutch & Jens C. Hansen) , pp. 135-212.
[31] Although the time of death of these Faroese and Bergen residents (that were noted to be born between 1899 and 1923) was not directly listed in the paper, we may clarify that those deaths were probably around late 1980s from available aspects of quick collection (i.e., within 48 hours of deaths) and analyses of the specimen mentioned in the paper.
[32] Andersen et al. (1987) Science of the Total Environment, vol. 65, 63-68.
[33] Julshamn et al. (1989) Science of the Total Environment, vol. 84, 25-33.
[34] See: *When Mercury Fears Harm* (http://ff.org/centers/csspp/pdf/szwarc_072006.pdf)
[35] http://ff.org/centers/csspp/pdf/20050228_hgfishadvisories.pdf
[36] http://ff.org/centers/csspp/pdf/20050119HGHeart.pdf
[37] http://ff.org/centers/csspp/pdf/mercury092804.pdf
[38] http://ff.org/centers/csspp/pdf/doingharm_072006.pdf
[39] Gago-Dominguez et al. (2003) British Journal of Cancer, vol. 89, 1686-1692.
[40] Terry et al. (2003) American Journal of Clinical Nutrition, vol. 77, 532-543.
[41] Terry et al. (2001) Lancet, vol. 357, 1764-1766.
[42] Friedland et al. (2003) Archives of Neurology, vol. 60, 923-924.
[43] Weil et al. (2005) Journal of the American Medical Association, vol. 293, 1875-1882.
[44] p. 48 of Weihe et al. (2002) International Journal of Circumpolar Health, vol. 61, 41-49.
[45] Arnold et al. (2005) American Journal of Public Health, vol. 95, 393-397.
[46] Roger Pielke, Jr. (Prometheus web blog, 22 August 2005) (http://sciencepolicy.colorado.edu/prometheus/archives/climate_change/000537the_other_hockey_sti.html