Valuation of Health Benefits from Mercury Pollution Control

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NESCAUM

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Panel on Human Health Effects of Pollutants
Air Pollution as Climate Forcing: A Second Workshop
East-West Center, Honolulu
April 4-6, 2005
Economic Valuation of Human Health Benefits of Controlling Mercury Emissions from U.S. Coal-Fired Power Plants

February 2005
Overview

• The report covers diverse areas of policy-relevant research including:
  – Mercury emissions (including changes from coal plants), atmospheric transport and fate, modeling of Hg deposition
  – Relationship between Hg deposition and methylmercury levels in fish, current and future exposures in humans to mercury in fish
  – Dose response functions, and finally, monetization of benefits
Previous National Power Plant Mercury Studies

• Have not examined thoroughly relationship between dietary methylmercury intake and sources of consumable fish
• Ignored cardiovascular effects
What did this Report Monetize?

• “Monetized two end points:
  – IQ of children born to mothers with high blood-Hg levels
  – Myocardial infarction and premature mortality among adults
Global Mercury Cycle
Units: 100 tons/yr

Adapted from Lamborg et al., 2002
Most scientists agree on the total input of mercury to the atmosphere and the relative amount of this mercury that has come from anthropogenic sources (about two thirds of the total).

### Global Sources of Mercury to the Atmosphere (in metric tons per year)

<table>
<thead>
<tr>
<th>Source</th>
<th>Seigneur et al. 2004</th>
<th>Bergan et al.</th>
<th>Mason &amp; Sheu 2002</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct Anthropogenic</td>
<td>2143</td>
<td>1999</td>
<td>2400</td>
</tr>
<tr>
<td>Recycled Anthropogenic</td>
<td>2134</td>
<td>2000</td>
<td>2090</td>
</tr>
<tr>
<td>Total anthropogenic</td>
<td>4277</td>
<td>4160</td>
<td>4490</td>
</tr>
<tr>
<td>Natural</td>
<td>2134</td>
<td>1900</td>
<td>2110</td>
</tr>
<tr>
<td>Total (% of Anthropogenic Origin)</td>
<td>6411 (67%)</td>
<td>6060 (69%)</td>
<td>6600 (68%)</td>
</tr>
</tbody>
</table>
Total Hg Emissions for the 1999 ICR Plants

NOTE: Excludes 1 plant in AK and 1 plant in HI
Mercury Emissions from Power Plants Cause Human Exposure to Mercury

Mercury transforms into methylmercury in soils and water, then can bioaccumulate in fish.

Fishing
- commercial
- recreational
- subsistence

*Humans and wildlife affected primarily by eating contaminated fish*

**Impacts**
- Best documented impacts on the developing fetus: impaired motor and cognitive skills
- Also: cardiovascular, immune, and reproductive system impacts

**Emissions Reductions**
- Reduce Atmospheric Transport and Deposition

**Reduce Ecosystem Transport and Methylation**
- Reduce Human and Wildlife Exposure
- Reduce Health Impacts
# Blood Methylmercury Concentrations ($\mu$g/L)
in U.S. Women 16 to 49 Years of Age

<table>
<thead>
<tr>
<th>Fish Meals prior month&lt;sup&gt;a&lt;/sup&gt;</th>
<th>n</th>
<th>50&lt;sup&gt;b&lt;/sup&gt;</th>
<th>75</th>
<th>90</th>
<th>95</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>480</td>
<td>--</td>
<td>0.4</td>
<td>1.1</td>
<td>1.6</td>
</tr>
<tr>
<td>1-4</td>
<td>780</td>
<td>0.6</td>
<td>1.3</td>
<td>2.9</td>
<td>4.7</td>
</tr>
<tr>
<td>5-8</td>
<td>230</td>
<td>1.3</td>
<td>3.3</td>
<td>6.1&lt;sup&gt;c&lt;/sup&gt;</td>
<td>9.9</td>
</tr>
<tr>
<td>&gt;8</td>
<td>153</td>
<td>2.8</td>
<td>5.2</td>
<td>11</td>
<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>1707</td>
<td>0.6</td>
<td>1.7</td>
<td>4.4</td>
<td>6.7</td>
</tr>
</tbody>
</table>

Source: Mahaffey et al., 2004

<sup>a</sup> Fish meal - self-reported number of finfish and shellfish meals in prior 30 days.

<sup>b</sup> percentiles of total blood methylmercury concentration

<sup>c</sup> Shaded values exceed US EPA’s methylmercury RfD (5.9 $\mu$g/L)
Key Assumptions

- Equilibria exist between
  - Mercury deposition rates
  - Fish methylmercury levels
  - Human fish consumption patterns

- Proportional relationship
  - Methylmercury concentrations and mercury deposition

- Hold constant
  - Emissions from other sources
  - Source waters where fish caught for consumption
  - Types and rates of fish consumed

- No environmental lag time

- Change in methylmercury intake as consequence of change in deposition at location where fish is caught
Speciation of Power Plant Mercury Emissions

Elemental mercury 5-95%
RGM 5-95%

Use of an average species distribution does not accurately reflect actual variability.

Actual distribution depends on:
- Coal type
- Operating conditions
- Control technology
Source of fish affects ability to reduce methylmercury intakes via emissions controls
Distribution of Power Plant Mercury Emissions by US Region (tons/year):

Total VS Hg\(^{++}\) and Hg\(_P\)

Source: EPA, 2001
Nationwide Estimates of Changes in Mercury Deposition

- REMSAD model output (EPA, 2003)
- Model inputs include changes in mercury emissions from other sectors
- Power plant responses to regulations, changes in demand, etc. using IPM
- Power plant mercury emissions
  - 49 tons/year (Base case, 2001)
  - 26 tons/year (Clear Skies Act, 2010, Scenario 1)
  - 18 tons/year (Clear Skies Act, 2020, Scenario 2)
Reducing Annual Power Plant Emissions to **26 tons** and **18 tons** results in Mercury Deposition Rate Decrease:

Source: U.S. EPA 2003
Predicted Percent Decreases in Mercury Deposition in the Five Freshwater Regions Relative to Current Emissions

<table>
<thead>
<tr>
<th>Region</th>
<th>Baseline1</th>
<th>Scenario 1</th>
<th>Baseline 2</th>
<th>Scenario 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northeast</td>
<td>Current deposition rate: 12.6 µg/m²/yr (199 Receptors)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average Decrease</td>
<td>9%</td>
<td>12%</td>
<td>9%</td>
<td>13%</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>9%</td>
<td>9%</td>
<td>9%</td>
<td>9%</td>
</tr>
<tr>
<td>Mid-Atlantic</td>
<td>Current deposition rate: 14.1 µg/m²/yr (201 Receptors)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average Decrease</td>
<td>22%</td>
<td>31%</td>
<td>24%</td>
<td>34%</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>12%</td>
<td>12%</td>
<td>12%</td>
<td>12%</td>
</tr>
<tr>
<td>Southeast</td>
<td>Current deposition rate: 10.2 µg/m²/yr (661 Receptors)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average Decrease</td>
<td>17%</td>
<td>20%</td>
<td>18%</td>
<td>24%</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>12%</td>
<td>12%</td>
<td>13%</td>
<td>12%</td>
</tr>
<tr>
<td>Midwest</td>
<td>Current deposition rate: 12.5 µg/m²/yr (841 Receptors)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average Decrease</td>
<td>9%</td>
<td>12%</td>
<td>9%</td>
<td>14%</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>7%</td>
<td>9%</td>
<td>8%</td>
<td>10%</td>
</tr>
<tr>
<td>West</td>
<td>Current deposition rate: 6.5 µg/m²/yr (3001 Receptors)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average Decrease</td>
<td>3%</td>
<td>4%</td>
<td>3%</td>
<td>4%</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>5%</td>
<td>5%</td>
<td>5%</td>
<td>6%</td>
</tr>
</tbody>
</table>
Predicted Percent Decreases in Mercury Deposition to the Coastal Atlantic Ocean Region, the Gulf of Mexico Region, and All Other Water Regions Under CSI

<table>
<thead>
<tr>
<th></th>
<th>Coastal Atlantic Ocean</th>
<th>Gulf of Mexico</th>
<th>All Other Waters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current Deposition Rate ($\mu g/m^2/yr$)</td>
<td>22.6</td>
<td>22.1</td>
<td>NA</td>
</tr>
<tr>
<td>Baseline 1</td>
<td>5.87%</td>
<td>3.52%</td>
<td>0.6%</td>
</tr>
<tr>
<td>Scenario 1</td>
<td>7.04%</td>
<td>3.89%</td>
<td>1%</td>
</tr>
<tr>
<td>Baseline 2</td>
<td>6.00%</td>
<td>3.54%</td>
<td>0.6%</td>
</tr>
<tr>
<td>Scenario 2</td>
<td>7.53%</td>
<td>4.29%</td>
<td>1.2%</td>
</tr>
</tbody>
</table>
Methylmercury Intake via Commercial Fish Consumption

• Weighted mean methylmercury concentration: commercial fish
  – weighted by per capita consumption (Carrington and Bolger, 2002)
  – weighted by proportion of fish caught in each source water (NMFS, 2002)

• Predicted mercury deposition decreases used to estimate decrease in mean methylmercury concentration by fish type and source water

• Approach assumes commercial fish consumers diet well mixed
  – eat variety of commercial fish (reflected by per capita data)
  – eat fish from a variety of source waters (reflect NMFS, 2002)

• Assume NHANES blood methylmercury concentration distribution in U.S. females of reproductive age is reasonable surrogate for oral intake
  – (methylmercury 1-compartment toxicokinetic model)
  – (NHANES data as reported in Mahaffey et al., 2003)
Average Methylmercury Concentrations for "Top 24" Types of Fish Consumed in the U.S. Commercial Seafood Market
For "Top 24" Types of Fish in U.S. Commercial Seafood Market, the Percentage of Methylmercury Contributed by Fish Type

Fish Type

† Estimate based on the product of per capita fish consumption rates and mean methylmercury concentrations of each type of fish (Carrington and Bolger, 2002)
Methylmercury Intake via Non-Commercial Freshwater Fish Consumption cont.

• Fish consumption rate Distributions for composite freshwater fish consumers
  – (EPA, 1997)

• Implemented through Monte Carlo Approach

• Size of freshwater fish consuming population based on U.S. FWS

• Allocation of freshwater fishers to regions
  – days fished in each State
  – aggregate by region
Methylmercury intake via Non-Commercial Marine Fish

- **Step 1:** Derive Weighted Mean methylmercury concentration for typical fish
  - Atlantic Ocean
  - Gulf of Mexico
    - Weighted by recreational catch mass by fish type (NMFS, 2002)
    - Mean fish methylmercury concentrations (EPA, 2003)
  - Assume reasonable for non-commercial fish

- **Step 2:** Calculate future fish methylmercury concentrations
  - Current mean concentrations
  - Ratio of present and future deposition rates
    - Assume
      - mercury depositing in these waters is well-mixed
      - non-commercial fish consumers diet well mixed

- **Step 3:** Fish consumption rates for angler populations (EPA, 1997)

- **Step 4:** Estimated population sizes (NMFS and FWS)
Additional Comments on Non-Commercial Consumers

- 80% of anglers consume their catch
  – EPA, 1997
- On average anglers share catch with 1.5 others
  – EPA, 1997
Persistent IQ Decreases via *in utero* Exposures


- $\Delta$IQ = -0.5 IQ points/1 ppm hair mercury (New Zealand)
- With and Without Threshold of 0.1 $\mu$g/kg/day

Diagram:
- IQ $\rightarrow$ School
- IQ $\rightarrow$ Wages
- IQ $\rightarrow$ Participation

**Key:**
- $IQ_W$: effect of IQ on wage
- $IQ_S$: effect of IQ on scholastic achievement
- $IQ_P$: effect of IQ on work force participation
- $S_W$: effect of scholastic achievement on wages
- $S_P$: effect of schooling on participation

-Adapted from Salkever, 1995; Schwartz, 1994
Fractional Contribution of Consumers of Non-Commercial Fish in Each Region and Commercial Fish to Total IQ Point Loss, Assuming No Neurotoxicity Threshold

- Atlantic: 4.5%
- Gulf: 3.4%
- Northeast: 3.4%
- Mid-Atlantic: 0.3%
- Southeast: 9.1%
- Midwest: 10.8%
- West: 9.4%

Population consuming commercial fish: 59.1%
EPA Methylmercury RfD

- EPA’s Position (general scientific consensus): low-dose fetal neurotoxicity is a credible effect
- Outcome: neurological impairment in 7 year-olds
  - Faroe Islands and New Zealand studies
  - Faroe study: 1 change persists in 14 year-olds
- Exposure: Intrauterine methylmercury
- Central tendency
  - BMDL$_{05} = 0.6$ µg/kg day → 1 µg/kg day
- Composite Uncertainty Factor = 10
- RfD = 1E-4 mg/kg/day
Methylmercury Threshold

• Functional form

\[ \begin{align*}
D \leq T, & \quad P(D) = 0 \\
D > T, & \quad P(D) = m \times (D - T)
\end{align*} \]

Where:

- \( P(D) \): Probability of effect at dose \( D \)
- \( T \): Population toxicity threshold (\( \mu g/\text{kg/day} \))
- \( D \): average dose (\( \mu g/\text{kg/day} \))
- \( m(*) \): slope dose-response function for (per \( \mu g/\text{kg/day} \))

• Threshold may be between 0 and 1 \( \mu g/\text{kg/day} \) Illustrate assuming threshold of 0.1 \( \mu g/\text{kg/day} \) (RfD)
Mercury Cardiovascular Toxicity

Salonen, 1995

- n = 1833 Male Finns, aged 42-60 years
- Estimated mean hair concentrations = 1.9 ppm, included 7 year follow-up
- Freshwater fish: high methylmercury and low polyunsaturated fatty acids concentrations

Rissanen 2000 - methylmercury intake may attenuate benefits of polyunsaturated fatty acids
Salonen 2000 - methylmercury may promote progression of arteriosclerosis (carotid thickening)

<table>
<thead>
<tr>
<th>Salonen</th>
<th>Fatal and Nonfatal AMI</th>
<th>All Cause Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>RR</td>
<td>p value</td>
</tr>
<tr>
<td>Hair Mercury (ppm)</td>
<td>1.07</td>
<td>0.18</td>
</tr>
<tr>
<td>Hair Mercury (&gt;2 ppm)</td>
<td>1.69</td>
<td>0.04</td>
</tr>
</tbody>
</table>

Difficult to separate cardioprotective components from potentially harmful components in epidemiology studies.
Relative Risk of Acute Coronary Events Based on Serum Fatty Acid Composition, Stratified by Hair Mercury Levels

<table>
<thead>
<tr>
<th>Hair mercury concentration</th>
<th>Quintiles, by Proportion of Serum Fatty Acids comprised of DHA and DPA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;2.4%</td>
</tr>
<tr>
<td>&lt; 2 ppm</td>
<td>0.9</td>
</tr>
<tr>
<td>&gt; 2 ppm</td>
<td>1.0</td>
</tr>
</tbody>
</table>

DHA = Docosahexaenoic Acid
DPA = Docosapentanoic Acid
DHA and DPA are fish-derived fatty acids

Source Rissanen, 2000
Spectrum of Health Effect Weight-of-Evidence

1. Persistent IQ deficits from fetal exposures above MeHg RfD
2. Persistent IQ deficits in all children from fetal MeHg exposures
3. Cardiovascular effects and premature mortality in male consumers of non-fatty freshwater fish with high MeHg levels
4. Cardiovascular effects and premature mortality in male fish consumers
5. Cardiovascular effects and premature mortality in all fish consumers

Decreasing Weight-of-Evidence
### Summary of Cost-of-Illness and Value-of-Statistical Life Approaches for Neurotoxicity and Cardiovascular Toxicity

<table>
<thead>
<tr>
<th>Scenario 1</th>
<th>Neurotoxicity Threshold</th>
<th>No Neurotoxicity Threshold</th>
<th>Costs AMI+ ACM (VSL) Male Pike Consumers</th>
<th>Costs AMI+ ACM (COI)</th>
<th>Costs AMI+ ACM (VSL)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$75,311,000</td>
<td>$193,940,000</td>
<td>$48,436,000</td>
<td>$154,814,000</td>
<td>$3,286,000,000</td>
</tr>
<tr>
<td>Scenario 2</td>
<td>$119,002,000</td>
<td>$288,247,000</td>
<td>$86,713,000</td>
<td>$231,244,000</td>
<td>$4,907,000,000</td>
</tr>
<tr>
<td>Scenario 1</td>
<td>Summary of neurotoxicity costs and cardiovascular toxicity costs (no threshold)</td>
<td></td>
<td>$242,376,000</td>
<td>$348,754,000</td>
<td>$3,480,000,000</td>
</tr>
<tr>
<td>Scenario 2</td>
<td>Summary neurotoxicity costs and cardiovascular toxicity costs (no threshold)</td>
<td></td>
<td>$374,959,000</td>
<td>$519,491,000</td>
<td>$5,195,000,000</td>
</tr>
</tbody>
</table>
### Spectrum of Health Effect Certainty

<table>
<thead>
<tr>
<th>Persistent IQ deficits in all children from fetal MeHg exposures</th>
<th>Persistent IQ deficits from fetal exposures above MeHg RfD</th>
<th>Cardiovascular effects and premature mortality in male consumers of non-fatty freshwater fish with high MeHg levels</th>
<th>Cardiovascular effects and premature mortality in male fish consumers</th>
<th>Cardiovascular effects and premature mortality in all fish consumers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scenario 1</td>
<td>$75M</td>
<td>$194M</td>
<td>$48M</td>
<td>$1.5B</td>
</tr>
<tr>
<td>(26 TPY)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scenario 2</td>
<td>$119M</td>
<td>$288M</td>
<td>$86M</td>
<td>$2.3B</td>
</tr>
<tr>
<td>(18 TPY)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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**Decreasing Certainty**

**Increasing Benefit**

Spectrum of Certainty of Causal Association of Health Effect with Mercury Exposure with Estimated Benefit Overlay in Millions ($M) and Billions ($B) of Dollars (2000$)
Value of Monetized Benefits for about 70 percent control

- Annual Benefits: 100 to 300 million dollars for IQ gain
- Annual benefits: 3 to 5 Billion dollars for avoided mortality and avoided non-fatal CHD
Moving Forward... Influence Diagram for Controlling Methylmercury Intake

- Exposure Control Strategy
- Utility Mercury and PM
- Population Methylmercury and PM Exposure
- Population Morbidity and Mortality
- Morbidity and Mortality Costs
- Total Social Cost
- Control Costs

Emissions Control Strategies:
1. Control Hg, PM and its precursors
2. Control Hg (specific)

- Environmental Fate
- Methylmercury Neurotoxicity Dose-Response
- Methylmercury Cardiovascular Disease Dose-Response
- PM Mortality Exposure Response
Cost and Performance of Sorbent-Based Mercury Control

Mercury Removal (%) vs. Sorbent Costs (mills/kWh)

- FF Bituminous
- FF PRB (EPRI Pilot)
- ESP Bituminous
- ESP PRB
Some Observations on Policy

§ Many states in the U.S. are moving at a faster and a more certain pace than the federal regulation, based on the assumption that environmental regulation drives technology innovation and implementation.

§ Hg Control technologies are now commercially available; new technologies are rapidly emerging; 90% and higher control is feasible.

§ Cost effectiveness of Hg control is quite comparable to, and more attractive than, the cost effectiveness of SO$_2$ and NOx controls from power plants (Hg:SO$_2$:NOx:1 to 3 mills/kwhr: 3-5 mills/kwhr: 2-3 mills/kwhr).
Some Observations: Effect on Global Climate Change

§ “Hg co-benefits” through control of SO$_2$ with wet and dry scrubbers has substantial effect on sulfate aerosols (40 percent of fine PM mass in the U.S.)

§ Application of bag houses (fabric filters) instead of or in addition to ESPs to control Hg results in large reductions in primary PM emissions