

**Testimony on Pennsylvania's Proposed Mercury Reduction Plan  
Provided to Pennsylvania's Senate Environmental Resources  
and Energy Committee  
Senator Mary Jo White, Chairman  
by Gail Charnley, PhD  
6 June 2006**

Senator White and other committee members, good morning and thank you for inviting me to share my views with you today. I am providing this testimony at the invitation of Senator White and am not representing any organization. I would like to make three main points:

- First, nobody disputes that methylmercury can be toxic to the developing nervous system and if women are exposed to too much of it during pregnancy, it can affect their babies. What is disputed is how much methylmercury exposure has to occur before it becomes a problem. Current methylmercury exposures do not appear to pose a public health threat for most US women and children.
- Second, fish is good for you and the children of women who eat fish during pregnancy, even when it contains methylmercury, apparently perform better on tests of neurologic development than the children of mothers who avoid fish, thanks to important nutrients in fish.
- And third, while reducing power plant mercury emissions is certainly desirable, it is inappropriate to assume that doing so will lead directly to reduced risks from methylmercury. I believe that the public health benefits of the Governor's proposed rule are unlikely to exceed those attainable through implementing EPA's CAMR and are being both oversimplified and oversold.

I will also address the issue of the potential for risks to children from inhaled mercury vapor associated with power plants.

## Methylmercury exposure and risk

At one of your previous hearings you heard a little about the relationship between the levels of methylmercury that have been associated with adverse effects in kids and the levels of methylmercury to which we are actually exposed. I would like to provide more detail on that if I may, because I think it's helpful to see how the levels of methylmercury that have been associated with adverse effects compare to the levels we're generally exposed to. Such comparisons provide some insight into the extent to which methylmercury may or may not be a public health problem in the US or in Pennsylvania.

As the earlier witness testified, based on its ongoing biomonitoring survey, the US Centers for Disease Control (CDC) reports that children and women of childbearing age in the US have methylmercury levels in their blood well below those that have been reported to produce adverse effects.<sup>1</sup> Figure 1 in the handout illustrates the relationships among the numbers I'm about to give you. According to a National Academy of Sciences report, an umbilical cord blood mercury level of 85 micrograms per liter was associated with a 5% likelihood of poorer performance on the Boston Naming Test, a test of memory, among children in the Faroe Islands, where people rely primarily on seafood as their source of dietary protein.<sup>2</sup> US EPA used a blood mercury level of 58 micrograms per liter—a conservative, health-protective lower limit on 85 micrograms per liter—as the basis for calculating a methylmercury reference dose. A reference dose is defined by EPA as “an estimate of an exposure . . . that is likely to be without an appreciable risk of adverse effects over a lifetime.” The reference dose for methylmercury, 5.8 micrograms per liter, was obtained by dividing 58 micrograms per liter by 10, in order to protect any unusually sensitive individuals. The average mercury blood level in women of childbearing age reported by the CDC was 0.83 micrograms per liter, with 5.7% of the women tested having

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<sup>1</sup>US Centers for Disease Control and Prevention (2005) *Third National Report on Human Exposure to Environmental Chemicals*. <http://www.cdc.gov/exposurereport/>

<sup>2</sup>National Academy of Sciences (2000) *Toxicological Effects of Methylmercury*. National Academy Press

blood mercury levels between 5.8 and 58 micrograms per liter (i.e., exceeding EPA's reference dose). None of this representative cross-section of women who have been tested in the US had blood mercury levels even approaching 85 micrograms per liter, the value calculated in the National Academy of Sciences report as being associated with a 5% change in performance on the memory test.

Figure 2 in the handout shows another way to compare exposures. In addition to blood levels, mercury exposure can be reflected in hair. Figure 2 shows (1) measurements of mercury levels in the hair of the women in the Faroe Islands who ate methylmercury and PCB-contaminated pilot whales and whose children tended to perform more poorly on the Boston Naming Test as their mothers' exposure to mercury increased;<sup>3</sup> (2) EPA's reference dose, or recommended limit on methylmercury exposure;<sup>4</sup> (3) the average mercury level found in the hair of US women of childbearing age tested by the CDC;<sup>5</sup> (4) the upper 90<sup>th</sup> percentile mercury level in US women of childbearing age; and (5) the mercury level reported for a sample of mothers in Japan.<sup>6</sup> Both Figures 1 and 2 show that the mercury level associated with neurodevelopmental deficits in the Faroe Islands is much higher than the levels of mercury exposure in US women. The Japanese data provide an interesting contrast and were used by the investigators to calculate that more than 90% of Japanese women have mercury levels that exceed EPA's reference dose for methylmercury. As far as I know, there is no epidemic of poor neurodevelopmental performance in Japan.

It is the finding that 5.7% of US women have blood methylmercury levels exceeding EPA's reference dose that has served as the basis for a variety of claims about how many babies

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<sup>3</sup>Grandjean et al. (2001) *Neurotoxicology and Teratology* 23:305

<sup>4</sup>US EPA (2001) *Water Quality Criterion for the Protection of Human Health: Methylmercury*. <http://www.epa.gov/waterscience/criteria/methylmercury/document.html>

<sup>5</sup>US Centers for Disease Control (2001) *Morbidity and Mortality Weekly Report* 50:140  
file:///C:/MyFiles/ABEC/MMWR%20NHANES%20Blood%20&%20Hair%20Hg%201999.htm

<sup>6</sup>Iwasaki et al (2003) *Tohoku Journal of Experimental Medicine* 200:67

in the US are born each year adversely affected by methylmercury, including a claim on PA DEP's website.<sup>7</sup> For example, much has been made out of an EPA scientist's assertion that because about 6% of the women in the CDC survey had levels that exceed EPA's reference dose, either 300,000 or 600,000 children, depending on the assumptions, are born each year "at risk" of developmental neurotoxicity. First of all, that is an assertion made by an EPA scientist and is not EPA's position. Second of all, exceeding a reference dose does not put someone "at risk." For example, page 248 of EPA's Regulatory Impact Assessment for the Clean Air Mercury Rule states, "It is . . . important to note that the [reference dose] does not define a bright line, above which individuals are at risk of adverse effect."<sup>8</sup>

Finally and most importantly, EPA's reference dose is based solely on a study performed in the Faroe Islands that no other public health organization in the world relies on for the development of methylmercury exposure limits. The study is not relied on by others for two reasons: (1) the primary source of methylmercury exposure was pilot whale, not fish, so the well established benefits of fish consumption during pregnancy that appear to be protective against the effects of methylmercury were not applicable and (2) the pilot whales were contaminated with high levels of PCBs, which have been associated with the same neurodevelopmental effects that were reported for methylmercury in that study. Although the National Academy of Sciences committee that evaluated methylmercury did conclude that PCBs did not confound the results of that study, the committee did not have access to research and data that have become available subsequently. The committee did not consider postnatal exposure to PCBs via breast milk, so potential effects associated with postnatal exposure to PCBs were not assessed. And, according to the study investigators themselves, when the effects of prenatal exposure to PCBs were properly controlled for, the correlation between methylmercury exposure and poorer

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<sup>7</sup><http://www.depweb.state.pa.us/mercury/site/default.asp>

<sup>8</sup>US EPA (2005) *Technical Support Document: Methodology Used to Generate Deposition, Fish Tissue Methylmercury Concentrations, and Exposure for Determining Effectiveness of Utility Emission Controls: Analysis of Mercury from Electricity Generating Units*. Pages 6-11

performance on the Boston Naming Test was no longer statistically significant.<sup>9</sup> A subsequent review co-authored by a different EPA scientist concluded that PCB exposure was associated with poorer performance on the Boston Naming Test in the Faroes.<sup>10</sup>

As Figure #3 in the handout shows, the level of PCBs to which the children were exposed in the Faroe Islands was almost double the level demonstrated to produce neurologic effects in infant monkeys and almost 1,000 times higher than EPA's reference dose for PCBs. So it is my opinion and that of many other scientists that the results of the Faroe Islands study at best should be attributed to combined exposure to methylmercury and PCBs and that EPA's reference dose is lower, or more stringent, than it would be if it were based on the combined exposure. The data also do not rule out the conclusion that the outcomes reported in the Faroe Islands could be due solely to PCBs and not methylmercury at all. In that case, EPA's reference dose would be meaningless. In either case, and for the various reasons I have just discussed, concluding that there are hundreds of thousands of US children either "at risk" or actually experiencing neurodevelopmental effects from methylmercury because their moms' blood methylmercury levels exceed EPA's reference dose is disingenuous and inflammatory.

I don't doubt that there are groups of subsistence fishers in the US who may be exposed to higher levels of methylmercury if they rely on freshwater predator fish as their primary protein source, and whose children may truly be at risk as a result, but I don't know how many of those live in Pennsylvania or whom, exactly, the governor believes he is protecting with his proposed rule. Shutting down all the coal-fired generation in Pennsylvania tomorrow will have no impact on most people's methylmercury exposure, which comes primarily from ocean fish, especially canned tuna, not from fish caught in Pennsylvania. I understand that there are over a million sports anglers in Pennsylvania but I couldn't find any data specific to what they do or do not eat in terms of what they catch. I also understand that there is a catfish farming industry in Pennsylvania but could not find data on the extent to which those fish are or are not

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<sup>9</sup>Grandjean et al. (1997) *Neurotoxicology and Teratology* 19:417

<sup>10</sup>Schantz et al. (2003) *Environmental Health Perspectives* 111:357

contaminated with methylmercury, although I know that in general, catfish are considered one of the least mercury-contaminated fish species.<sup>11</sup> Even Physicians for Social Responsibility considers farmed catfish to be safe.<sup>12</sup> US EPA has ranked Pennsylvania 24<sup>th</sup> out of 37 states in terms of largemouth bass methylmercury contamination and 17<sup>th</sup> out of 26 states for carp methylmercury contamination, although those data are from the 1990s.<sup>13</sup>

In the US overall, according to the National Marine Fisheries Service, more than 75% of the fish we eat is imported and half of what we eat comes from a can.<sup>14</sup> According to The Annapolis Center, of those who eat fish in the US, about 10% of the fish they eat comprise freshwater fish<sup>15</sup> (not all of which would be methylmercury-contaminated). In the State of New York, 98% of sports anglers surveyed reported that they either don't eat what they catch at all or eat it less frequently than once per month.<sup>16</sup> In Wisconsin, of the women of childbearing age who reported eating fish, less than one-third reported eating sport fish and there was no difference between their hair mercury levels and those of women who did not eat sport fish.<sup>17</sup> It would be good to include in your deliberations whatever specific data might be available on who the people are in Pennsylvania who are actually potentially at risk from methylmercury present in Pennsylvania fish, so that a more informed discussion of the potential benefits of either CAMR or the proposed rule might be conducted.

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<sup>11</sup>US EPA, <http://www.epa.gov/ost/fishadvice/advice.html>

<sup>12</sup>PSR, <http://home.earthlink.net/~healthyfishing/red.html>

<sup>13</sup>US EPA, <http://www.epa.gov/ost/fish/mercurydata.pdf>

<sup>14</sup>US NMFS, [http://www.st.nmfs.gov/st1/fus/fus03/08\\_perita2003.pdf](http://www.st.nmfs.gov/st1/fus/fus03/08_perita2003.pdf)

<sup>15</sup>Annapolis Center (2003) *Mercury in the Environment: The Problems, the Risks, and the Consequences*. <http://www.annapoliscenter.org>

<sup>16</sup>Li et al. (2005) *Environmental Research* 97:142

<sup>17</sup>Knobeloch et al. (2005) *Environmental Research* 97:220

## Benefits of fish consumption

There is a large body of evidence demonstrating the cardiovascular benefits of fish consumption in adults<sup>18</sup> and an emerging body of evidence that women who eat more fish while pregnant have smarter kids than do women who avoid fish during pregnancy. For example, a study of kids in the Seychelle Islands, whose moms consumed an average of 12 fish meals weekly while pregnant, found that the kids whose moms had higher hair mercury levels when they were pregnant because they ate more fish tended to perform better than the kids of moms who had lower hair mercury levels and ate less fish. The authors of that study concluded that their data “do not support the hypothesis that there is a neurodevelopmental risk from prenatal [methylmercury] exposure resulting solely from ocean fish consumption.”<sup>19</sup> The average methylmercury concentration in fish in the Seychelles is similar to that in commercial fish consumed in the US (0.3 ppm) but because the Seychelle women ate more fish meals per week than most US women, their exposures were much higher. A recent study of children in the UK reported that increasing cord blood mercury levels were not associated with increased cognitive impairment but that increasing prenatal fish consumption was associated with improved cognition.<sup>20</sup> A different study of the same group of kids in the UK found that the children of mothers who ate more fish during pregnancy and were exposed to more methylmercury had IQs five points higher than the children of mothers who ate less.<sup>21</sup> A preliminary study in Massachusetts found similar results.<sup>22</sup> In all cases, the cognitive benefits were attributed to the omega-3 fatty acids found in fish. Omega-3 fatty acids are essential for appropriate nervous system development and function. Other micronutrients found in fish are also considered

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<sup>18</sup>See review by Kris-Etherton et al. (2002) *Circulation* 106:2747

<sup>19</sup>Myers et al. (2003) *Lancet* 361:1686

<sup>20</sup>Daniels et al. (2004) *Epidemiology* 15:394

<sup>21</sup>Hibbeln (2006) *Nutrition, the brain and mental ill health*. Cleave Lecture. Presented at the conference “Generating Healthy Brains,” 17 January 2006. London

<sup>22</sup>Oken et al. (2005) *Environmental Health Perspectives* 113:1376

important contributors to successful brain development or even capable of preventing neurotoxicity.<sup>23</sup> These studies do not suggest that methylmercury is good for children, but they do demonstrate that the benefits of fish clearly can overcome its potential threats, even in the kids of women who derive most of their dietary protein from fish and are exposed to higher levels of methylmercury as a result.

A presumably unintended consequence of overstating risks from fish is that some of the important contributions of fish nutrients to the developing brain may be lost. I personally have known several pregnant women who refused to eat fish because of what they had heard about mercury. Pregnant women and children should follow the advice of EPA and FDA and limit their consumption of high-mercury-containing fish while maintaining the health benefits of fish consumption by eating a variety of other fish. Figure 4 shows how much of different fish species can be safely consumed by pregnant women, based on EPA's methylmercury reference dose.

#### Relationship between power plant mercury emissions and fish methylmercury levels

Much of what appears in the media about fish, mercury, and power plants implies that mercury comes directly out of power plants, falls directly onto nearby fish, turns into methylmercury, and threatens the development of our children's brains. The implication of such a scenario would be that reducing mercury emissions from power plants would lead directly to less methylmercury in fish and safer children. It implies that reducing power plant mercury emissions by a certain percentage would lead to the same percentage decrease in fish methylmercury levels. Such a simple and direct relationship between mercury emissions and fish methylmercury levels is not supported scientifically.

While it seems logical to assume that reducing power plant or other mercury emissions will lead to reductions in local fish methylmercury levels, available data do not provide much support for that conclusion. The relationship between mercury emissions and fish methylmercury

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<sup>23</sup>Clarkson & Strain (2003) Journal of Nutrition 133:1539S

levels appears to be highly site-specific, so it is likely that reducing power plant mercury emissions could lead to lower fish methylmercury levels in some places and not in others. Predicting where changes might occur is not yet possible.

You have heard testimony from Nathan Willcox to the effect that a 2003 study in Florida reported that the levels of methylmercury in largemouth bass in the Everglades declined about 80 percent several years after state and federal agencies required incinerators to cut their mercury emissions. He used that statistic to support his contention that reducing mercury emissions anywhere will inevitably lead to lower local fish methylmercury levels. While his description of the Everglades data was accurate, he left out two important things: (1) EPA's CAMR requires power plants to cut their mercury emissions, so would be expected to lead to lower levels of methylmercury in largemouth bass if the Florida data are correct and (2) data were collected from 12 Florida locations, not just in the Everglades. If you look at Figure 5 in the handout, you can see that overall, the largemouth bass taken from all 12 locations were fairly evenly split between declining trend and no or increasing trend. Consistent declining trends across age groups were seen at three of the locations sampled, a consistent lack of trend was seen at four locations, and an increasing trend was seen at one location. The other locations showed some declines and some absence of change depending on the age of the fish.<sup>24</sup> It seems odd that Mr. Willcox would share with you only the data that support his conclusion and omit those that contradict it.

Mr. Willcox also failed to mention why it is likely that extrapolating the potential impact of lower incinerator mercury emissions in the Everglades to the potential impact of lower power plant mercury emissions in Pennsylvania is tenuous at best. First of all, the mercury emitted from incinerators is different from the mercury emitted from power plants. Most incinerator mercury is water-soluble and deposits close to incinerators; most power plant mercury is elemental, not water soluble, and ends up becoming part of global atmospheric mercury. Second of all, the Everglades are a unique, tropical ecosystem that is strikingly different from the decidedly non-

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<sup>24</sup>Florida Department of Environmental Protection (2003) *Integrating Atmospheric Mercury Deposition with Aquatic Cycling in South Florida: An approach for conducting a Total Maximum Daily Load analysis for an atmospherically derived pollutant*. [www.floridadep.org/labs/mercury/index.htm](http://www.floridadep.org/labs/mercury/index.htm)

tropical ecosystems found in Pennsylvania. They differ greatly in terms of water chemistry and other factors that determine the extent to which deposited mercury becomes fish methylmercury. And third, there was no contemporaneous determination of whether reduced mercury emissions actually led to reduced local mercury deposition, which would have had to have occurred if there was a connection between local mercury emissions and local freshwater fish methylmercury concentrations. Incinerator controls were initiated in 1987 but deposition measurements were not taken until 1995.

The extent to which mercury deposited into water bodies—whether natural, global, local, and/or the result of human activities—actually turns into methylmercury and gets into fish is very site-specific, depending on factors like water chemistry, pH, temperature, sunlight, nutrient levels, and other site-specific characteristics.<sup>25</sup> As a result, the amount of methylmercury in fish is not related simply to the amount of mercury that is available. A lake with high mercury levels in its sediment can have fish with low methylmercury levels and a lake with lower mercury levels can have fish with higher methylmercury levels. Lakes in the same region with the same atmospheric mercury deposition can have widely varying fish methylmercury levels. While it is true that coal-fired power plant mercury emissions eventually may be deposited on the surrounding soil, soil concentrations have not been correlated with distance or direction from such plants.<sup>26</sup> In a recent report from a workshop convened by the Society for Environmental Toxicology and Chemistry, the authors concluded, “It is not clear whether changes in mercury input will result in a linear change in mercury methylation. Computer models, such as one developed for the Florida Everglades, tend to predict a linear response, but there are little data to support the predictions.”<sup>27</sup>

Thus, for freshwater fish caught in the vicinity of coal-fired power plants, available data neither support nor rule out a relationship between fish methylmercury levels and plant emissions;

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<sup>25</sup>Summarized in Center for Science and Public Policy (2005) *Making Sense of State Fish Advisories*. [http://ff.org/centers/csspp/pdf/20050228\\_hgfishadvisories.pdf](http://ff.org/centers/csspp/pdf/20050228_hgfishadvisories.pdf)

<sup>26</sup>Sato & Sada (1992) *Atmospheric and Environmental Pollution* 26A:325

<sup>27</sup>Mason et al. (2005) *Environmental Science and Technology* 39:14A

such relationships appear to be site-specific and unpredictable with current data. It is therefore not possible to predict whether and to what extent reducing power plant mercury emissions will result in reduced fish methylmercury concentrations in Pennsylvania. It certainly will not be possible to distinguish between the effects of the Governor's 90% reduction versus EPA's 86% reduction in terms of its impact on public health, even if it were possible to achieve one earlier than the other, although I gather that the costs of achieving one versus the other vary quite a bit. Any claims that Pennsylvania's state-specific proposed rule will protect high consumers of Pennsylvania fish any better than will the federal rule are not scientifically supportable.

### Mercury inhalation

Senator White has received a letter from the American Academy of Pediatrics identifying coal-fired power plants as a major threat to health resulting from inhalation of elemental mercury vapor. I have tried diligently to identify studies that demonstrate, or even suggest, an association between mercury emitted by power plants and ambient levels of mercury vapor, to no avail. Without providing data, the letter Senator White received implies that this is a real problem but—oddly—omits any mention of methylmercury and fish. Apparently the letter's implied link between power plant mercury emissions and elemental mercury vapor toxicity is based on a publication by Goldman et al.<sup>28</sup> That publication asserts in its abstract that “industry emissions with resulting ambient air pollution remain the most important source of inhaled mercury” but—mysteriously—that statement is not discussed at all in the article itself nor are any supporting citations provided.

The vapor phase mercury that is emitted from power plants does not become breathable mercury at ground level. That mercury becomes part of the global atmospheric cycle of mercury and is found in the troposphere. Vapor phase mercury measurements taken by Electric Power Research Institute (EPRI) scientists right next to power plant stacks at ground level are all consistent with the naturally occurring background concentration of mercury, 1.6 nanograms per

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<sup>28</sup>Goldman et al. (2001) Pediatrics 108:197

cubic meter of air.<sup>29</sup> The US Geological Survey has an instrumented trailer about 10 miles downwind from a coal-fired power plant in Alabama and is finding vapor phase mercury levels of 1.6 nanograms per cubic meter of air, the naturally occurring global background level, even when the wind is coming directly from the power plant's stack. EPRI's measurements right in the middle of a plume just emitted from a power plant showed an average concentration of only 11 nanograms per cubic meter of air. And, as I said, that mercury ends up in the global atmospheric mercury pool, not at ground level.

The US Agency for Toxic Substances and Disease Registry (ATSDR) at the Centers for Disease Control and Prevention identifies a minimal risk level for elemental mercury of 0.2 micrograms per cubic meter of air.<sup>30</sup> A minimal risk level is a public-health-protective estimate of daily exposure considered likely to be without appreciable risk that is intended to be protective of particularly sensitive individuals such as children. Both children's and adults' actual exposures to vapor phase mercury are thus 100 times less than the minimal risk level. In any case, power plants do not contribute to ground-level vapor phase mercury concentrations and the American Academy of Pediatrics' suggestion otherwise appears to be a red herring unsupported by scientific data.

### Mercury "hot spots"

You have heard a lot from some witnesses and other sources about how EPA's CAMR will cause hot spots of mercury to occur in Pennsylvania, leading to more contaminated fish and more children at risk. I am not an expert in emissions trading but I have never seen any actual evidence that hot spots are a real problem. Everything I have read on the subject indicates the opposite. Emissions trading smooths out hot spots, it doesn't create them. I have offered into the record a report prepared by Byron Swift, who *is* an expert on hot spots, based on work he did at

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<sup>29</sup>Personal communication, Dr. Leonard Levin, Electric Power Research Institute

<sup>30</sup>ATSDR Toxicological Profile for Mercury, <http://www.atsdr.cdc.gov/toxprofiles/tp46.html>,  
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the Environmental Law Institute (ELI),<sup>31</sup> published in ELI's Environment Reporter and reproduced by the Bureau of National Affairs. His report evaluates the effects of EPA's clean air trading programs that are already in place and finds—based on actual performance data—“that none has resulted in a regional shift of emissions, and all trading programs examined have led to proportionately greater emissions reductions from the larger sources . . . Overall, the data from the programs reviewed indicate that trading has not created geographic hot spots and, in promoting reductions at the largest plants, has smoothed out pollutant emissions instead of concentrating them.” I haven't been able to locate any data contradicting Swift's conclusion, so I offer his report for your consideration as you evaluate the pros and cons of the proposed rule.

#### Final note

Finally, just for the record, I am very tired of opponents of the Bush Administration continually referring to how it's “rolling back” or “weakening” power plant mercury controls. The Bush Administration is the first administration to actually regulate power plant mercury emissions. The Clinton Administration had 8 years to regulate power plant mercury emissions but did not. How can the Bush Administration be rolling back something that didn't exist before??

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<sup>31</sup>Swift's work was funded by The Joyce Foundation and A.W. Mellon Foundation