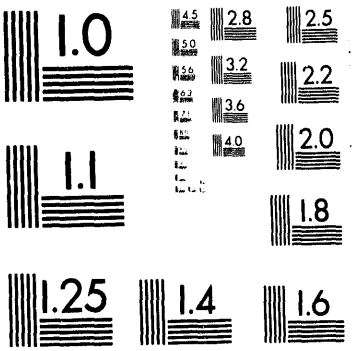
1.0
1.1
1.25
1.4
1.6
1.8
2.0
2.2
2.5
2.8
3.2
3.6
4.0
4.5
5.0

1 of 1

BNL-49862

AN ASSESSMENT OF ADULT RISKS OF PARESTHESIA DUE TO MERCURY FROM COAL COMBUSTION

F. Lipfert, P. Moskowitz, V. Fthenakis, M. DePhillips,

J. Viren and L. Saroff

November 1993

Prepared for presentation at Power-GEN Americas '93
Dallas, TX 11/16-18/93

Biomedical and Environmental Assessment Group
Analytical Sciences Division
Department of Applied Science
Brookhaven National Laboratory
Upton, New York 11973

This research was performed under the auspices
of the United States Department of Energy under Contract No.
DE-AC02-76CH00016

DISCLAIMER

Prepared for presentation at POWER-GEN Americas '93, Dallas, TX, November 19, 1993

AN ASSESSMENT OF ADULT RISKS OF PARESTHESIA DUE TO MERCURY FROM COAL COMBUSTION

F. Lipfert, P. Moskowitz, V. Fthenakis, and M. DePhillips
Department of Applied Science
Brookhaven National Laboratory
Upton, NY 11973

J. Viren
Consultant
4643 144th Place, S.E.
Bellevue, WA 98006

L. Saroff
Program Manager
Office of Fossil Energy
U.S. Department of Energy
Washington, DC 20585

Abstract

This paper presents results from a probabilistic assessment of the mercury health risks associated with a hypothetical 1000 MW coal-fired power plant. The assessment draws on the extant knowledge in each of the important steps in the chain from emissions to health effects, based on methylmercury derived from seafood. Because of the non-linear nature of the dose-response functions, both global background levels and local source increments are considered. We rely on "black-box" assessment approaches only in the absence of adequate information, and we seek realism rather than conservatism throughout the analysis. We explicitly recognize that the general public displays a wide range of fish consumption patterns, mainly comprising a variety of species. Further, we recognize that health effects are keyed to equilibrium levels of blood Hg, not to acute individual doses. For this assessment, we define three separate sources of dietary Hg: canned tuna (affected by global Hg), marine shellfish and finfish (affected by global Hg), and freshwater gamefish (affected by both global Hg and local deposition from nearby sources). We consider emissions of both reactive and elemental mercury from the hypothetical plant (assumed to burn coal with the U.S. average Hg content) and estimate wet and dry deposition rates; atmospheric reactions are not considered. Mercury that is not deposited within 50 km is assumed to enter the global background pool. The incremental Hg in local fish is assumed to be proportional to the incremental total Hg deposition. Three alternative dose-response models were derived from published data on specific neurological responses, in this case, adult paresthesia (skin prickling or tingling of the extremities). The Monte Carlo methods used in the assessment specifically incorporate the uncertainties in the dose-response functions, as well as in the dose terms. Preliminary estimates show the upper 95th percentile of the baseline risk attributed to seafood consumption to be around 10^{-4} (1 chance in 10,000). Based on a doubling of Hg deposition in the immediate vicinity of the hypothetical plant, the incremental local risk from seafood would be about a factor of 4 higher. These risks should be compared to the estimated background prevalence rate of paresthesia, which is about 7%. The paper recommends that a similar approach be taken to assess the fetal risks implied by maternal seafood consumption, which are thought to be more critical than adult paresthesia.

This project is sponsored by the Office of Clean Coal Technology, U.S. Department of Energy, Washington, DC. The following is a synopsis of a draft progress report to that office.

INTRODUCTION

Two special studies of mercury in the environment are being prepared in response to the Clean Air Act Amendments of 1990. The National Institute of Environmental Health Sciences will prepare a report on the maximum acceptable average daily intake levels for mercury, and the U.S. Environmental Protection Agency will prepare a study of mercury emissions from electric utilities. These studies are to be used in an analysis of the need to regulate mercury emissions from utilities.

This report presents results from a probabilistic assessment of the mercury health risks associated with a hypothetical 1000 MW coal-fired power plant, in support of this forthcoming regulatory analysis. We assume that methylmercury in seafood is the dominant exposure pathway. The approach draws on the extant knowledge in each of the important steps in the chain from emissions to health effects, recognizing that both global background levels and local source increments must be considered, because of the non-linear nature of the dose-response functions. Only in the absence of adequate information do we rely on "black-box" approaches, and we seek realism rather than conservatism throughout the analysis. We explicitly recognize that the general public displays a wide range of fish consumption patterns, mainly comprising a variety of species. Further, we recognize that health effects are keyed to equilibrium levels of blood Hg, not to acute individual doses. For this assessment, we define three separate sources of dietary Hg: canned tuna (affected by global Hg), marine shellfish and finfish (affected by global Hg), and freshwater gamefish (affected by both global Hg and local deposition from nearby sources). We estimate wet and dry deposition of Hg from the plant, and any mercury that is not deposited within 50 km is assumed to enter the global background pool. The incremental Hg in local fish is assumed to be proportional to the incremental total Hg deposition. We do not use existing "reference doses" as an index of health effects because of their embedded conservatism; rather, we use alternative dose-response models based on the original data on specific neurological responses, in this case, adult paresthesia. The Monte Carlo methods used in the assessment specifically incorporate the uncertainties in the dose-response functions, as well as in the dose terms.

PREVIOUS ASSESSMENTS OF METHYLMERCURY HEALTH EFFECTS

Previous assessments of the health risks of methylmercury (MeHg) in the environment have been lacking in any of several respects:

1. Use of reference dose levels as the measure of acceptable risk, thus ignoring the conservatism that is built into these regulatory guidelines.

2. Incomplete consideration of the dietary dose of MeHg, especially with regard to baseline risk levels.

3. Unrealistic assumptions about the contributions of local sources of atmospheric Hg.

4. Incremental risks based on an assumed bioaccumulation factor, which can only be even approximately correct for a single species of fish, whereas almost all fish diets comprise a variety of species.

5. Failure to account for the temporal averaging process inherent in the build-up of equilibrium Hg levels in blood.

MERCURY IN THE ENVIRONMENT

Mercury is extracted from the earth primarily as cinnabar (HgS); annual worldwide production is about 10,000 tons. Coal combustion releases mercury to the atmosphere primarily as gaseous compounds, while municipal incineration releases both gaseous and particulate forms. Certain industrial processes, chiefly chlor-alkali plants, were known to release large amounts of mercury in aqueous waste streams, but these have now been largely brought under control in the developed world. Natural emissions emanate from volcanoes, soil, biota, sea spray, and forest fires. While there are considerable uncertainties, natural and anthropogenic emissions of mercury are thought to be roughly comparable. Suffice it to say, there are no major individual sources of mercury emissions on the planet, and the problem should be regarded as global in scope.

The mercury cycle is quite complicated, since the residence time of Hg vapor in the atmosphere is long (weeks to months) and there are few permanent sinks. Aquatic sediments provide relatively long-term storage, but organic Hg released to the water column can be reduced to mercury vapor and re-emitted to the atmosphere. Even mercury in dental amalgams can be recycled through cremation.

The pathways for human exposure to mercury include inhalation of airborne mercury, which is mainly inorganic, and through ingestion of foods containing mercury, mainly as MeHg, which is defined as the CH_3Hg^+ ion in whatever form it appears. Many studies have shown that the most important pathway is through consumption of predatory fish, since they can bioconcentrate methylmercury by many orders of magnitude as it works up the food chain. Mercury in water and other foods does not constitute an important pathway (except perhaps for the low doses characteristic of non fisheaters).

Mercury is present in the atmosphere in three oxidation states: elemental, Hg^0; monovalent, Hg^+; and divalent mercury, Hg^{++}. Mercury is emitted by anthropogenic sources in elemental and various oxidized forms. Elemental Hg and $(CH_3)_2Hg$ also enter the atmosphere via biological processes. Oxidized inorganic forms (e.g., $HgCl_2$) are extremely water-soluble, methyl chlorides (e.g.,$HgCH_3Cl$) are also quite soluble, whereas elemental Hg and dimethyl mercury $(CH_3)_2Hg$ are insoluble forms. Until recently it was generally assumed that the main form of volatile mercury in air is elemental Hg; this is still the case for Hg generated from natural sources, but there is evidence that plumes emitted from coal-burning power plants initially contain a large fraction of water soluble $HgCl_2$. Background ambient concentrations of Hg in remote areas are in the range of 1-3 ng/m^3, and in urban areas as high as 10 ng/m^3. In the plume of a coal-fired power plant, concentrations of total gaseous mercury in excess of 1000 ng/m^3 have been measured within a few km from the source. The degree that these compounds are removed from the atmosphere by rain depends on their solubility in water; divalent Hg (Hg^{++}) is more soluble and dominates wet deposition; mercury vapor (Hg^0) has higher ambient concentrations and dominates dry deposition.

Several studies have been done in Europe and the U.S. on the effect of deposition of atmospheric mercury in lakes. A study of Hg levels in 220 lakes in southern and central Sweden showed an implied relationship with atmospheric deposition. Data compiled by Lindqvist (1985) indicate that the predominant mercury compounds in fresh waters are inorganic and monomethyl mercury compounds. The chlorides $HgCl_2$ and CH_3HgCl are the main mercury compounds in neutral and acidic water environments, whereas in alkaline environments the hydroxide compounds $Hg(OH)_2$ and CH_3HgOH prevail. Thus, we expect lake pH to play an important role in the bioaccumulation of mercury in aquatic organisms. Field data have confirmed this trend and also shown that dissolved organic carbon (DOC) can increase the rate of methylation. The processes involved in overland transport of deposited Hg through the watershed are less clear.

THE BNL RISK ASSESSMENT

Table 1 presents the major features of the BNL risk assessment model developed for this project.

Table 1 Features of the BNL Mercury Assessment Model

Emissions	Fraction of Hg^{++} is estimated based on Cl^- content of coal. Effects of air pollution controls are estimated.
Transport, dispersion, and deposition	Gaussian model for distances to 50 km. Constant values of V_d and washout ratio for 3 classes of Hg compounds. No atmospheric chemical reactions.
Accumulation in surface waters	All Hg deposited within the watershed is assumed to enter the water body. Incremental deposition is estimated (wet+dry).
Contributions to global background	Any Hg not deposited within 50 km is assumed to enter global pool. Global deposition is increased in proportion to (net) emissions.
Effects of local sources on fish Hg content	Existing average Hg in each fish species is assumed to increase in proportion to the Hg deposition increment (local plus global).
Background dose from seafood	Dose is calculated by summing 3 independent log-normal distributions (freshwater fish, marine species, canned tuna), using Monte Carlo methods. The Hg concentration in each distribution is assumed to be independent of consumption rate.
Equilibrium metabolic model	Long-term average dose is then estimated separately, by frequency of eating. The distribution of blood Hg is estimated from distributions of dose, body mass, and half life of Hg, using Monte Carlo methods. The last 2 factors are negatively correlated.
Dose-response functions	Three different dose-response models are developed from the Iraqi paresthesia data, using Monte Carlo methods, and are used to estimate levels of risk for the baseline case and for the incremental effects of a hypothetical power plant.

EMISSIONS AND ATMOSPHERIC PROCESSES

The plant is assumed to burn coal with an average content of mercury (0.8 ppm), to be equipped with an electrostatic precipitator, and to have a capacity factor of 80%. In general, we found little reliable data on the parameters controlling mercury deposition from power plant plumes. There is more information on the relevant processes for background Hg, but these data may not apply to the near field (i.e., footprint) of a plume. We use results from conventional (short-range) air quality modeling, based on the assumption of Gaussian profiles in the power plant plume, to estimate annual air concentrations at the surface, which is assumed to be flat.

Although recent data indicate that a high proportion of mercury in coal-fired power plant stacks is Hg^{++}, we have no information on its fate as it travels downwind in the plume. It has been suggested that the Hg^{++} may be reduced back to Hg^0 by SO_2. If the Hg^{++} remains as such, we expect that it will behave as a reactive gas such as HNO_3 and deposit rapidly, but it may also sorb onto particles. If these are fine particles, dry deposition rates will be slowed dramatically. Dry deposition is modeled by assuming a value for the dry deposition velocity (V_d), defined as the ratio of the deposited flux to the air concentration. Different values of V_d are assigned to elemental, water-soluble, and particulate forms of Hg.

We modeled wet deposition in two different ways, using either the washout ratio (ratio of concentration in precipitation to air concentration) or a dynamic plume depletion model, and obtained similar results. Both methods require site-specific data on precipitation frequencies, durations, and wind directions. The model suggests that local effects may double or triple the total background deposition of mercury, but that, in any event, more than 99% of the Hg emissions from a tall stack will travel beyond the 50 km radius. At this radius, the incremental Hg deposition from the plant is of the order of 1% of background levels.

BASELINE MERCURY LEVELS IN SEAFOOD

We find substantial variations in the mercury concentrations reported for a given fish species. Some of these may be due to differences in laboratory techniques over time and the reporting of total Hg vs. MeHg, but the main sources of variability within a given species are fish size and age, and for freshwater species, lake DOC and pH in addition. We were not able to identify an effect of watershed/lake surface area ratio on Hg content, which raises questions as to the mobility of Hg deposited in a watershed. The variations among the average levels of Hg in different species were of about the same order as those within a given species, and were probably related to the trophic level of the fish. When weighted by the quantities caught, there was no difference between average mercury levels in marine shellfish and marine finfish (about 0.1 ppm), if tunafish are considered separately (averaging about 0.2 ppm). Mercury levels for freshwater gamefish were higher (averaging about 0.3 ppm) and more variable. There was the suggestion of a downward trend in Hg levels over time, especially for canned tuna, but this could not be confirmed statistically. Better data on mercury levels in seafood are badly needed. These considerations apply to the baseline status of mercury in seafood, which may include the distributed regional effects of existing coal combustion. To estimate the incremental local effects of a hypothetical plant, we assume proportionality between mercury deposition from the atmosphere and the mercury content of local fish.

FISH CONSUMPTION RATES AND THE DISTRIBUTION OF BASELINE MEHG DOSES

In order to derive seafood consumption statistics appropriate for this assessment, we found it necessary to combine data from various sources. For example, much of the detailed data on distributions of consumption date from 1973-74, and overall seafood consumption has increased substantially since then. Thus, we use the trend data from national overall production statistics to adjust the older distributional statistics upward to more nearly reflect current consumption levels. However, this procedure may entail errors if public preferences for certain species have changed over the years. We find good support from both surveys and production statistics for an overall average seafood consumption rate of about 25 g/d (even in "urban" Alaska), with a

95th percentile level of about 80 g/d. About 95% of the U.S. population consumes some seafood over the course of a year. The average MeHg daily (baseline) dose for consumers of freshwater fish in the upper Midwestern U.S. was estimated to be about 2.6 ug/d, and the 99th percentile was well below the estimated threshold for adult health effects. As discussed above, local increases in mercury deposition are assumed to have proportional effects on the mercury dose to the population (consumption levels are assumed to remain at baseline levels).

THE STEADY-STATE DIET-BLOOD MeHg RELATIONSHIP

We examine several sources of information on the relationship between MeHg in the diet and the levels of Hg reached in blood, which then form the basis for neurological health effects. The most commonly used relationship is based on acute doses by only 5 subjects, and does not present a realistic picture of the uncertainties. We find that epidemiological approaches to this topic suffer from imprecision as to the dietary doses, which biases the resulting slope of blood MeHg with respect to dietary MeHg downward. It is also possible that actual fish consumption under long-term conditions includes factors that may mitigate the uptake of MeHg, such as selenium or salt. We base our risk estimates on data derived from 20 volunteers who consumed controlled diets of halibut for about 100 days, with four basic levels of MeHg. The relationship between diet and blood was linear, but showed a different dependence on body mass than was seen in the earlier study.

DOSE-RESPONSE FUNCTIONS

We use data on adult paresthesia from about 120 Iraqi adults who were affected by consuming contaminated grain in 1971-2. These data are only available as 7 groups of individuals, classified according to their estimated intake of MeHg (Figure 1). Thus, to include the possible variability within each group, we use Monte Carlo methods to generate a range of dose response functions, for three different mathematical models: 2 "hockey-stick" or threshold models, and a continuous risk model employing the logistic function. This process adds the fundamental uncertainties in the dose response functions and their supporting data to the other uncertainties that are propagated through the risk analysis process. The differences in estimated risk levels that are entailed by the choice of model also reflect the fundamental uncertainties for this end-point. These are among the uncertainties that prompt regulatory authorities to use safety factors when establishing reference dose levels.

ASSESSMENT OF BASELINE AND INCREMENTAL RISKS FROM A 1000 MW PLANT

Preliminary results are as follows: The logistic risk model developed for this project predicts a baseline risk level of about 10^{-4} (1 chance in 10,000), at the upper 95th percentile. Using "hockey-stick" or threshold models in a Monte Carlo simulation with 5000 trials, we find zero risk of adult paresthesia for baseline dose levels, even for subsistence populations. When power plant increments are added, the expected average risk levels are of the order of 5×10^{-4}. There is considerable uncertainty as to the appropriate parameters to use in estimating the incremental effects of local sources, and these uncertainties have not been modeled with Monte Carlo methods. The effects of emissions of a single power plant on global mercury levels are seen to be trivial, and this is probably the case for the entire U.S. utility industry as well.

RECOMMENDATIONS

It is clear that the risks of adult paresthesia from fish consumption are low. This analytical framework should be extended to the case of maternal fish consumption and fetal effects on retarded child development, which are thought to be considerably more sensitive. It is also clear that the basic data used in this analysis are in need of improvement.

1. Mercury levels in fish are based on conflicting and outdated numbers.

2. Fish consumption patterns of sensitive subpopulations (such as pregnant women) have not been established.

3. Appropriate methods for estimating Hg deposition from power plants have not been validated in the field.

4. The intersections of the sets of U.S. coal-fired power plants, sensitive water bodies, and susceptible subpopulations have not been established.

5. Annual total Hg emissions from coal burning and from other sources, including natural sources, are still uncertain.

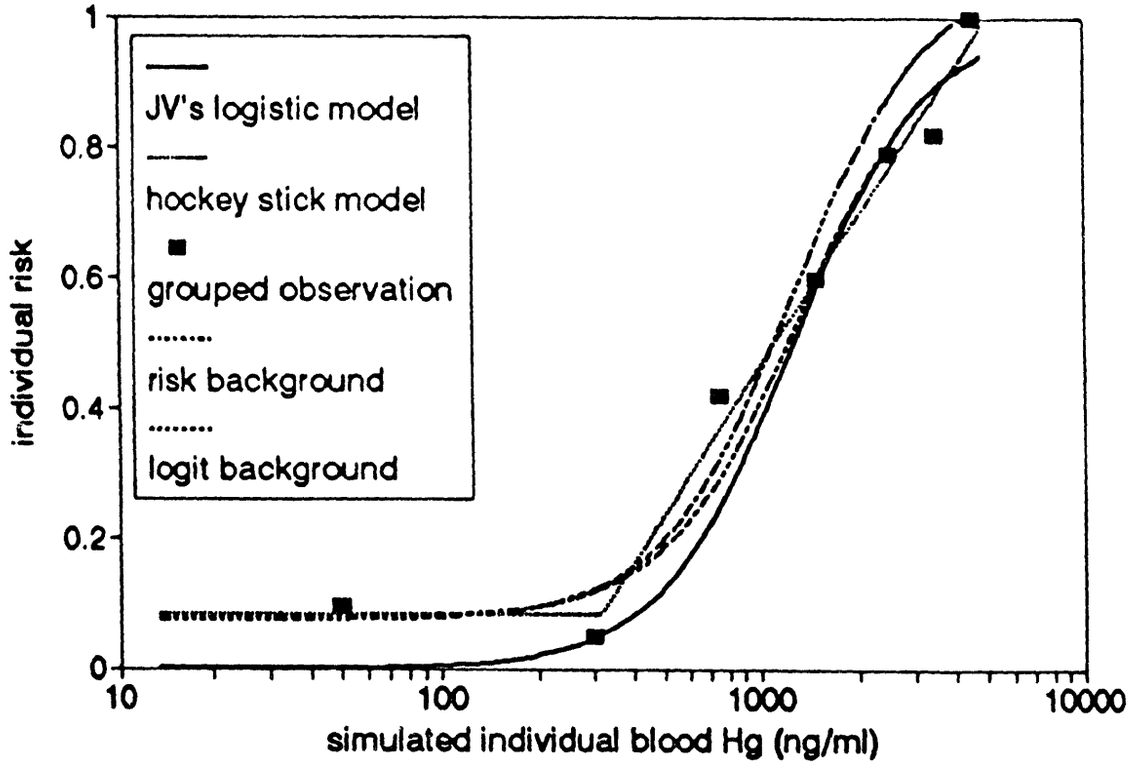

Figure 1. Data on frequency of paresthesia for Iraqi adults who consumed contaminated grain, and alternative dose-response functions fit to that data.

DATE

FILMED

3/11/94

END