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A Systems Approach to Risk Assessment: Application to Methylmercury from Coal Combustion

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INTRODUCTION

Two special studies of mercury (Hg) in the environment are being prepared for Congress in response to the Clean Air Act Amendments of 1990. The National Institute of Environmental Health Sciences has prepared a draft report on the maximum acceptable average daily human intake levels for mercury,[1] and the U.S. Environmental Protection Agency has prepared a draft study of mercury emissions from major anthropogenic sources and their environmental effects.[2] These studies are to be used in an analysis of the need to regulate mercury emissions from utilities, which are currently exempted from the hazardous air pollution regulations in the Amendments. In support of this forthcoming regulatory analysis, The Department of Energy (DOE) asked Brookhaven National Laboratory (BNL) to perform a probabilistic assessment of the health risks associated with Hg from coal-fired power plants.[3-5] The objective of the assessment is to estimate the incremental health risks that might ensue from a typical coal-fired power plant, together with their uncertainties, taking into account existing background levels and the actual adverse health effects that have previously been associated with exposure to various Hg species.

Mercury has a long history of association with adverse neurological effects at high exposure levels. The most important current exposure pathway has been found to be ingestion of fish containing methylmercury (MeHg), which is the end product of bioconcentration moving up the aquatic food chain. Mercury can enter natural waters from either industrial discharges or from atmospheric deposition of various inorganic Hg compounds. Because of the worldwide background and the existence of local emissions sources, Hg deposition must be considered on local, regional and global scales. The regulatory technical challenge presented by methylmercury is to protect public health without foreclosing an appreciable a portion of the food supply or impacting on the lifestyles of North American native populations.

This paper presents an abbreviated account of the DOE/BNL risk assessment, as viewed from a systems perspective. We review the structure of the model, the sources of data used, the assumptions that were made, and the interpretation of the findings. Since publication of the first risk assessment report[3], we have refined our estimates of local atmospheric dispersion and deposition and "calibrated" the pharmacokinetic portion of the model against observations.

THE BNL RISK ASSESSMENT MODEL

The probabilistic risk assessment model combines probability distributions for a number of input parameters that define exposure to a toxic agent, and then compares the resulting distribution of exposures to the levels at which adverse health effects have been observed. The resulting risk is thus defined as the product of the probability of exposure at some level times the probability of experiencing an adverse effect at that exposure level. This assessment comprises an assembly of 3 modular "subsystems":

1. the global Hg background, which defines the Hg intake and status of persons not exposed to current anthropogenic emissions,
2. the impacts of current anthropogenic emissions over and above that background,
3. the information necessary to describe human health effects that might be expected at various exposure levels.

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Because of the recycling and persistence of mercury in the environment, some portion of today's background may have originated as anthropogenic emissions in previous years. We describe the elements of the model in terms of the sequence of calculations from emission source to receptor, with accumulation in freshwater fish as an important intermediate point in the chain.

Emissions and Control Processes

The model powerplant is assumed to be located near a lake in the upper Midwest and to have a capacity factor of 75%. In order to allow for a range of generation capacities and coal Hg contents, we assumed the product of nameplate rating and Hg concentration to be 250 ppm-MW, which corresponds to annual Hg emissions of about 560 kg. This allows for, say, a 1000 MW plant burning coal with 0.25 ppm Hg or a 2500 MW plant burning coal at 0.1 ppm Hg or combinations of values in-between. We note that this assumed emission rate corresponds to the upper end of the range of Hg emissions from U.S. power plants.[2] We assume that the plant is equipped with state-of-the art electrostatic precipitators that remove 10% of the Hg, but we do not consider the environmental effects of Hg in the solid waste stream in this assessment.

Atmospheric Processes

The model tracks the fates of 3 Hg species through the atmosphere: elemental Hg (Hg^0), reactive Hg (Hg^{++}), and particulate Hg (Hg_p). Hg^{++} is the most important species for local impacts, since its high solubility leads to rapid washout and deposition in the near field downwind of the plant (ca. 50 km). Based on early experimental results,[6] we assumed that 75% of the emitted Hg is in the form of Hg^{++}. Results computed by the U.S. Environmental Protection Agency (EPA) for several coal-fired power plants in the range 900-1100 MW were used to estimate the near-field atmospheric dispersion ratios (Figure 1). The peak cumulative average concentrations and thus deposition occur about 2-10 km from the plant. By the time the emissions travel 50 km from the stack, local concentrations are reduced by an order of magnitude or more, such that their impact on the existing background becomes negligible. Approximately 5-8% of the stack Hg emissions are deposited (wet + dry) within a 50 km radius from the stack. We assume that the remainder of the emitted Hg becomes part of the global background.

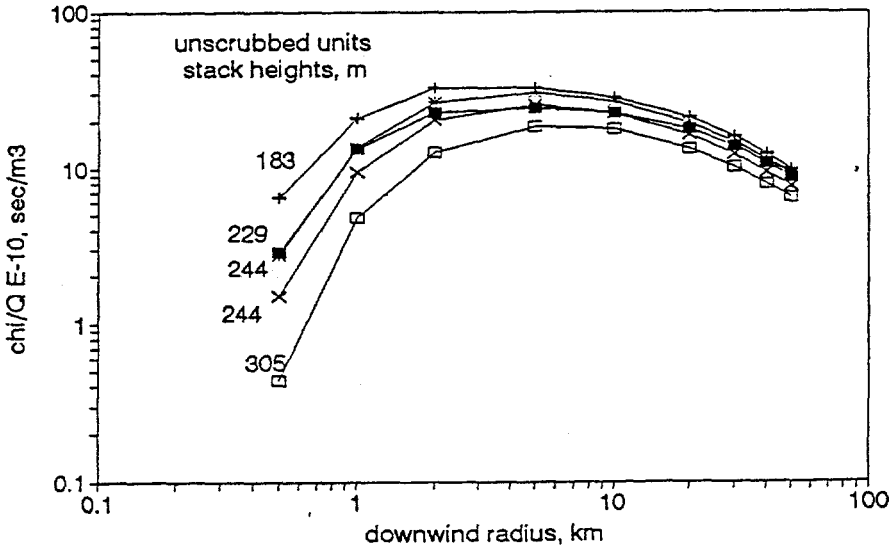

Figure 1. Dispersion factors for annual average concentrations from coal-fired power plants in the Eastern U.S., 900-1100 MW. Values are cumulative average concentrations (g/m^3) out to the specified radius, averaged around the compass, divided by annual emissions (g/s).

Although we found little reliable data on the parameters controlling mercury deposition from power plant plumes, our model compares well with measurements of wet deposition of Hg downwind of a municipal waste incinerator.[7] There is more information on the relevant atmospheric processes for background Hg, but these data may not apply to the near field (i.e., footprint) of a plume.

Probabilistic consideration of all of the vagaries of powerplant design, location, Hg emissions, transport, deposition, and bioaccumulation would entail cumulative uncertainties of several orders of magnitude. To preclude these uncertainties from dominating the risk assessment, we constrained the variability in the net effect of anthropogenic deposition above background through use of an "impact factor", representing the percentage increase in local deposition averaged over a nearby waterbody, with respect to the deposition expected from background Hg sources alone. Based on our dispersion analysis and measured background data, the impact factor was assigned a uniform probability distribution from 1.5 to 3.0 (50% to 200% increase over background). This factor includes the effects of Hg deposited on land in the watershed and subsequently transported into the lake. All other factors remaining equal, the impact factor would be proportionately lower for lower rates of Hg emissions or for a smaller fraction of Hg^{++} in the stack plume.

Baseline Mercury Levels in Seafood

We found substantial variations in the fish mercury concentrations reported in the literature for a given species. Some of this variation 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 appear to be fish size and age. In addition, for freshwater species, the levels of dissolved organic carbon (DOC) and pH of the water body from which the fish were taken can affect Hg levels. We were not able to identify an effect of watershed/lake surface area ratio on Hg content from the available data, which raises questions as to the mobility of Hg deposited in a watershed. There is some recent evidence that the presence of wetlands along the lake shore may be an important factor with respect to the role of DOC.[8] The variations among the average levels of Hg between different fish species were of about the same order as those within a given species, and were probably related to the trophic level of the fish. MeHg from tuna is the largest source of dietary Hg for most Americans; average Hg concentrations are around 0.2 ppm. For other kinds of seafood, there was no difference between average mercury levels in shellfish and finfish; both were about 0.1 ug/g. Mercury levels for freshwater game fish such as pike, walleye or bass averaged about 0.3 ug/g and were more variable. There was the suggestion of a downward trend in fish Hg concentrations over time, especially for canned tuna, but this could not be confirmed statistically. Better data on fish mercury levels are needed.

Effects of Local Hg Deposition on Fish Hg Content

The above considerations apply to the baseline fish mercury concentrations, including the distributed regional effects of existing coal combustion. To estimate the incremental local effects of a hypothetical powerplant, we assume proportionality between local mercury deposition from the atmosphere and the mercury content of local (freshwater) fish. Thus, an impact factor of 1.5 corresponds to a 50% increase in the Hg content of the freshwater fish consumed, with respect to the baseline case.

Fish Consumption Rates

In order to derive fish 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 seafood consumption date from 1973-74, but overall per capita consumption has increased substantially since then. We used the trend data from national overall production statistics to adjust the older distributional statistics upward to more nearly reflect current consumption levels, but 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 per capita fish consumption rate of about 25 g/d, with a 95th percentile level of about 80 g/d. These consumption figures correspond to about 43 and 87 fish meals per year, respectively; the maximum number of fish meals in the simulation was about one per day. Surveys indicate that about 95% of the U.S. population consumes at least some fish over the course of a year.

Meals of tuna fish are assumed to have vary from 26-130 g each; all other fish portions are assumed to be vary between 100-300 g each. However, we also assume a (positive) relationship between body mass and the size of the fish meals consumed, which resulted in a mean intake parameter for the baseline case of 0.032 ug/kg/day and 0.055 ug/kg/day for the impact case. The average correlation between the number of fish meals and body mass in this simulation was 0.66, which is higher than was reported from a survey in Canada.[9] That survey only looked at total food consumption for all ages and found a linear correlation of about 0.33 with body mass. The lower correlation could have resulted in part from the nonlinearity introduced by including teenagers in the sample.

The Pharmacokinetics of Ingested MeHg

In contrast to both carcinogenic and irritant air pollutants, the dose* metric of practical concern for MeHg is neither the maximum rate of acute intake or the total accumulated intake, but the equilibrium body burden that is attained as a balance between quasi-steady rates of intake and excretion. The concept of the "reference dose" (the amount that may be consumed over a lifetime without adverse consequences[2]) would thus appear to be inappropriate here. The overall body burden of MeHg controls health responses and is directly related to measurable levels of MeHg in blood and hair, which serve as biomarkers. Because, as a practical matter, a high body burden of MeHg can only be obtained by eating fish more often (with a normal diet), the averaging process that takes place over time with respect to individual dose increments obtained from eating disparate meals is an important feature of the pharmacokinetics. Monte Carlo simulations were used to develop an empirical model of this process, which is given by

$$\log(GSD) = -1.211 + 1.766 \log(GSD_0) + 0.5\log(f^*k) \qquad [1]$$

where GSD and GSD_0 are the geometric standard deviations of the original and modified fish Hg distributions, f is the frequency of consumption, and k is the elimination constant (k=ln[2]/half-life). The GSDs were reduced by about 40% at the mean according to this relationship, thus narrowing the distribution of body burdens with respect to the distribution of intake rates. Figure 2 shows how the (geometric) standard deviation of body burden decreases as the frequency of consumption increases; eating fish more often increases the mean body burden but reduces its variability. One measure of the suitability of this model may be made by comparing the variability of the simulation results with the variability of observations of blood and hair Hg. The observed data from various sources have GSDs ranging from 1.4 to 2.8; the equivalent values for body burden from our simulations were 1.74 and 2.05, depending on assumptions. If metabolic averaging is not considered, typical MeHg intake GSD values obtained by combining the distributions of Hg concentration and fish consumption can exceed 3.0[10]. Important parameters in this model that are required to predict the equilibrium body burden of MeHg include body mass and the half-life of MeHg in the body. Conversion from body burden to blood and hair concentrations requires other parameters: the fraction of Hg in blood, the volume of blood, and the hair-blood ratio. The nominal blood-body burden ratio was 0.7 with a GSD of 2.0. The distribution of hair/blood ratios was selected to match the 12 studies listed by the U.S. Environmental Protection Agency,[2] which had a weighted mean ratio of 276.

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*Although it is common in the risk assessment literature to refer to the amount ingested as the "dose," strictly speaking, the term should be reserved to indicate the amount received by the for target organ, in this case, the brain.

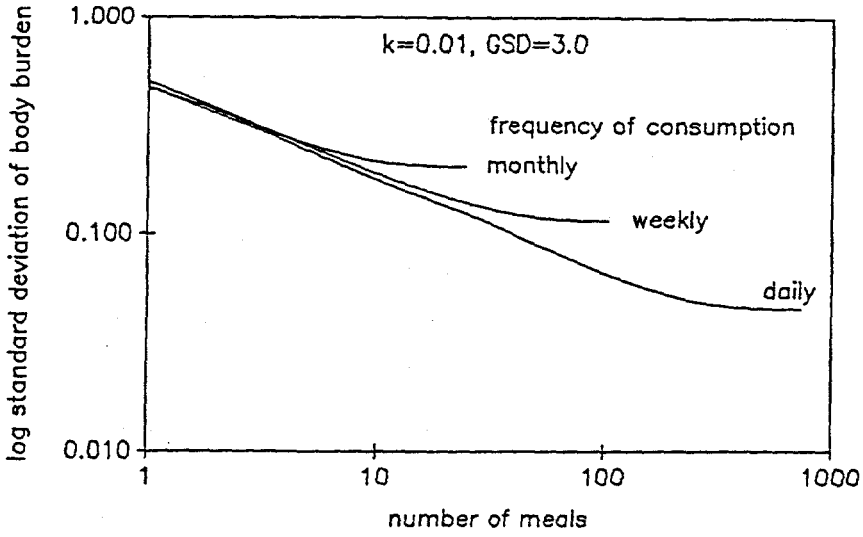


Figure 2. Simulated reduction in the standard deviation of body burden as a function of number of fish meals consumed in the time to reach equilibrium, with an underlying GSD of 3.0. (k = elimination constant)

Results of the Simulations. In order to confirm the suitability of the assumptions made in the model, comparisons were made with a set of hair MeHg concentration data first reported by Smith *et al.*[11] These data were obtained from a "demographically balanced" national sample of women of childbearing age in all 50 states. Hair samples were obtained from 1437 women and were analyzed for MeHg by gas chromatography. Food intake diaries were also completed and the average MeHg intake for the 1009 individuals reporting seafood consumption was about 1 ug/day. The highest recorded MeHg intake was 14.3 ug/d. In order to match this rate of MeHg intake for model "calibration" purposes, we deleted the freshwater fish portion of our dietary intake distribution; this yielded very good correspondence. The relevant statistics of this simulation are compared to the sample data in Table I and the hair Hg frequency distributions are compared in Figure 3. The agreement is seen to be excellent, which validates the model parameter values selected to represent MeHg intake (except for freshwater fish) and the pharmacokinetics. It is also interesting that the observed distributions of MeHg in hair are similar for those who reported eating fish and those who did not. Possible explanations include inaccurate diaries, other sources of MeHg, or confusion between Hg and MeHg peaks in the gas chromatography. Smith (personal communication, December 1994) reported a (log-log) correlation between hair Hg and dietary intake of about 0.59; we obtained a value of 0.53 from the simulation. We thus concluded that the baseline parameters selected for these simulations and their frequency distributions were consistent with observations. Simulations were then performed for the cases of interest, with freshwater fish included in the dietary intake as described above. The statistics derived from these simulations are given in Table II.

Whereas the distribution of body-burden Hg levels comprises a reasonably tight band, plots show that the scatter is greatly increased for hair Hg, varying by 2 orders of magnitude at a given intake level. This variability has important ramifications with regard to the prediction of risk estimates and the ability to develop accurate exposure-response data from observations. These results showed that the highest hair levels should not be expected to coincide with the highest body burdens or consumption rates, and thus are unlikely to relate to the presence of neurological effects on a 1:1 basis. As a further check on the interrelationships in the model,

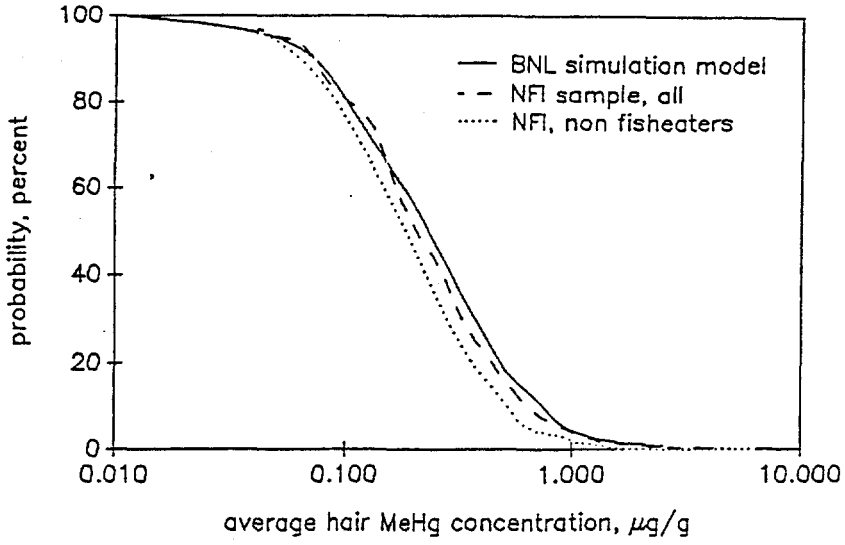


Figure 3. Comparison of simulated and measured[11] distributions of MeHg in hair.

Table I Comparison of Observed and Predicted MeHg Statistics (n=1000)

parameter	Observed[10]	Predicted
mean MeHg intake, ug/d	1.0	1.07
median hair concentrations*		
all subjects	0.26 (2.6)#	0.235 (2.56)
fisheaters	0.27 (2.4)	
non fisheaters	0.25 (2.5)	

* measured as MeHg but reported as Hg (ug/g)
# ( ) = geometric standard deviation

Table II Distribution Statistics Derived from Simulations (n=5000)

	------baseline------			-------impact------		
parameter	mean	median	95%	mean	median	95%
body mass, kg	73	71	104			
half life, days	72	68	118			
# meals in 5 half-lives	43	36	87			
intake, ug/d	2.5	2.0	5.7	4.3	3.5	10.6
intake parameter, ug/kg/d	0.032	0.029	0.061	0.055	0.047	1.22
body burden, mg/kg	0.0033	0.0027	0.0072	0.0057	0.0046	0.0135
blood concentration, ug/L	0.0029	0.0020	0.0086	0.0050	0.0032	0.015
hair concentration, ug/g	0.82	0.54	2.42	1.40	0.86	4.64

we compared correlations of the log of hair concentration with the MeHg intake per unit of body mass. The simulation produced a correlation coefficient of 0.41, which compares with a correlation of 0.32 between log(hair Hg) and the estimated frequency of fish consumption in a New Zealand sample.[12] These comparisons suggest that, even though hair Hg may be a reliable marker for individual MeHg exposure, the relationships between hair Hg and other measures of MeHg tend to be quite variable across a population. Note also that the inability to reliably measure low Hg concentrations (below the detection limit) will inevitably adversely affect comparisons with predictions that are made on a logarithmic scale.

HEALTH EFFECTS OF MeHg

The central nervous system is the principal target for MeHg, with the potential for effects on sensory, visual, and auditory functions. In adults, low doses may create non-specific symptoms such as paresthesia (tingling of the extremities), malaise, or blurred vision. Higher doses may bring deafness, loss of coordination when walking, and speech disorders, and, in extreme cases, coma and death.[13] Effects on fetal development may represent the most critical class of neurological effects, since the brain is thought to be most sensitive during development. Individuals may vary greatly in their responses to mercury exposure.

Adult Health Effects. Transient paresthesia is perhaps the mildest symptom of adult MeHg poisoning and was selected as the endpoint for this portion of the risk assessment. Data were available on 122 Iraqi adults and older children who consumed Hg-contaminated bread in 1971-1972.[14] Fifty-nine cases of paresthesia were observed, among other more severe neurological symptoms. It should be noted that paresthesia is not uncommon even in unexposed populations; thus some residual or background prevalence rate should be expected, perhaps of the order of a few percent.

Two indices of MeHg exposure were available for this cohort, blood concentrations and estimated consumption of bread. Blood Hg levels were determined an average of 65 days (about 1-1.5 half-lives) after the incident and have been used as an index of MeHg exposure in most previous analyses.[13] This suggests that the relevant (peak) blood concentrations may be about a factor of 2 higher than the measurements reported. Further evidence for this hypothesis is derived from exposure-response data based on the numbers of bread loaves consumed. The exposure-response function is plotted in Figure 4, based on the modified body burdens. A continuous fit to these data based on the logit function is also shown and seems to fit the observations moderately well.

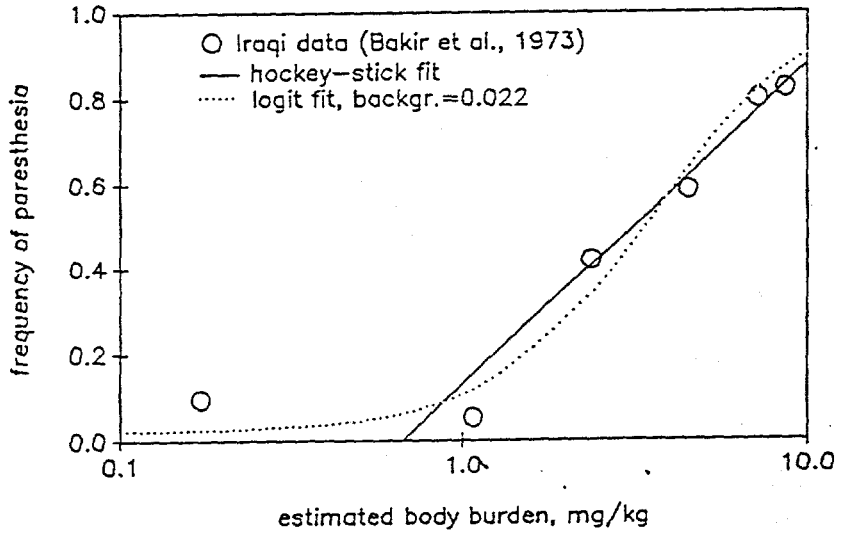


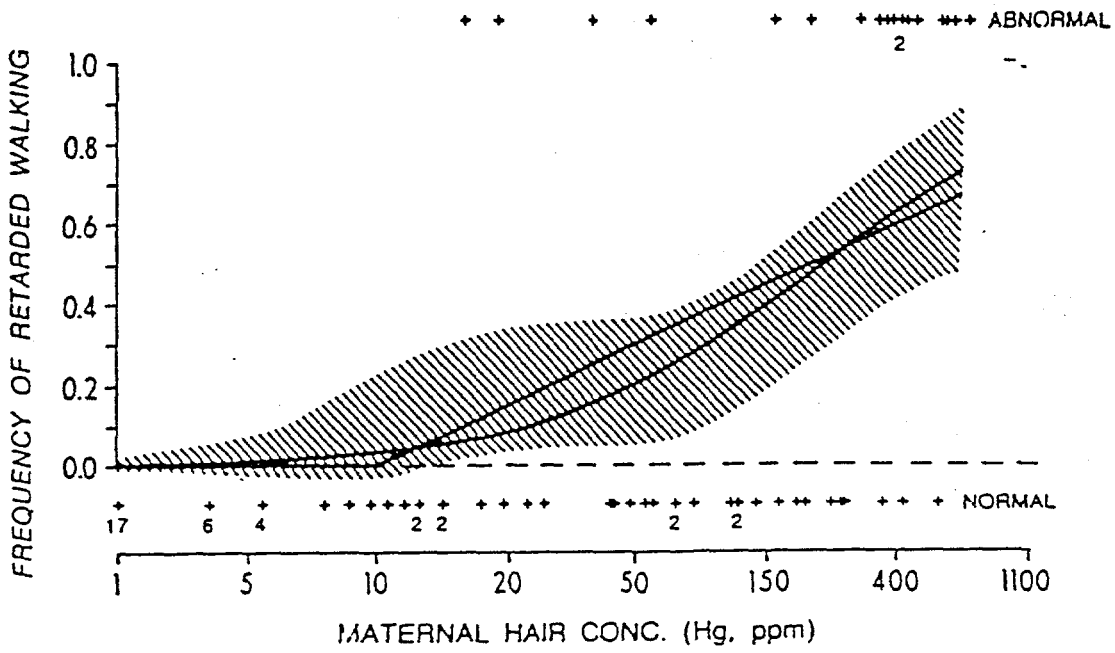
Figure 4. Exposure-response (paresthesia) data from the Iraqi grain poisoning incident. Body burden values have been adjusted to account for the delay in determining peak blood Hg concentrations.

However, Clarkson[15] represented the Iraqi data with threshold or "hockey-stick" models. The top group of data points define a nearly linear relationship quite well, as shown in Figure 4, such that the body-burden threshold depends mainly on the intersection of this diagonal line with the selected background prevalence rate of paresthesia. This methodology leads to an estimated body-burden threshold of onset of adult paresthesia of about 0.75 mg/kg, based on an average body mass of 50 kg for the affected Iraqis.

Congenital Health Effects. Three studies were found in the literature that provided quantitative data suitable for this portion of the assessment. A number of infants were exposed *in utero* during the Iraqi poisoned grain incident of 1971-2. Marsh *et al.*[16] examined 29 Iraqi children, age 4 1/2 to 5, and found that the mothers of the normal children had peak maternal hair Hg levels of 25 ug/g or less (corresponding roughly to average body burdens of 0.14 mg/kg or less). According to the authors, no specific threshold of effects could be identified because of the small sample; the threshold could have been as high as 85 ug/g. Cox *et al.*[17] reported on a much more elaborate analysis of 83 infant-mother pairs from this incident. The mean age of the children was 30 months at the initial interview. They considered frequencies of delayed walking, delayed talking, and of various neurological (CNS) signs. "Increased limb tone and deep tendon reflexes" were the most frequent CNS signs; these symptoms were also noted by Mckeown-Eyssen *et al.*[18] in Cree Indian children at lower maternal hair levels. Note that the definitions of "delayed" walking or talking were somewhat arbitrary and that exact birthdates were sometimes difficult to determine in this population. For this reason, we used the CNS symptom data in our assessment. Cox *et al.* fit logistic and hockey-stick models to these data and developed a data smoothing technique that provided functions of arbitrary shape (Figure 5). The delayed walking and talking exposure-response functions showed a suggestive increase in response around 10 ug/g, with a more definitive rise at around 100 ug/g. The latter point corresponds roughly to the body-burden threshold noted previously for adults. However, the CNS function was more nearly continuous, with the initial rise in response at around 15 ug/g peak maternal hair concentration. The curve-fits to these data suggested risks of CNS signs of 1-2% at peak maternal hair levels of 5 ug/g, for example.[17]

McKeown-Eyssen *et al.*[18] studied a group of Cree Indians in Northern Quebec in 1978. A total of 247 children, ages 12 to 30 months, were identified in four communities known to have experienced high levels of MeHg exposure. The source was fish contaminated by industrial Hg discharges. Hair samples were obtained from the mothers and a medical team examined each child for physical and neurological characteristics. The medical team was blinded to the exposure data. Confounding variables considered included amount of time spent "in the bush," maternal alcohol, tobacco, and caffeine consumption, mother's age, lactation, complications of pregnancy and numbers of previous pregnancies. The most frequent neurological finding was "abnormality of tendon reflex;" 13 boys and 14 girls. When combined with 2 cases of increased muscle tone, 15 "cases" were thus identified among the boys and a statistically significant ($p=0.05$) relationship was seen with prenatal Hg exposure, in the range from about 2-15 ug/g peak maternal hair concentration. There were no differences in the confounding variables between these 15 and the remaining 82 "controls." No other relationships were seen with Hg in boys, and these conditions were not associated with Hg exposure in girls. However, incoordination was negatively associated ($p=0.07$) with Hg exposure in girls. We find that this study offers only minimal support to the hypothesis that maternal hair levels around 10 ug/g are associated with deficiencies in childhood neurological development.

The most extensive data set currently available is that of Kjellstrom *et al.*[12] who selected 73 New Zealand children whose mothers had the highest hair Hg levels during pregnancy, out of a cohort of some 11,000 births. These cases were matched with 164 controls whose mothers had lower hair Hg levels during pregnancy. The analysis then seeks to determine to what extent maternal hair Hg levels may predict the children's IQ at ages 6-7. We performed screening analyses that found 3 low IQ outliers; after removing these points, we found a marginally significant relationship ($p=0.08$) between WISC-R full-scale IQ and the log of average maternal hair Hg during pregnancy:



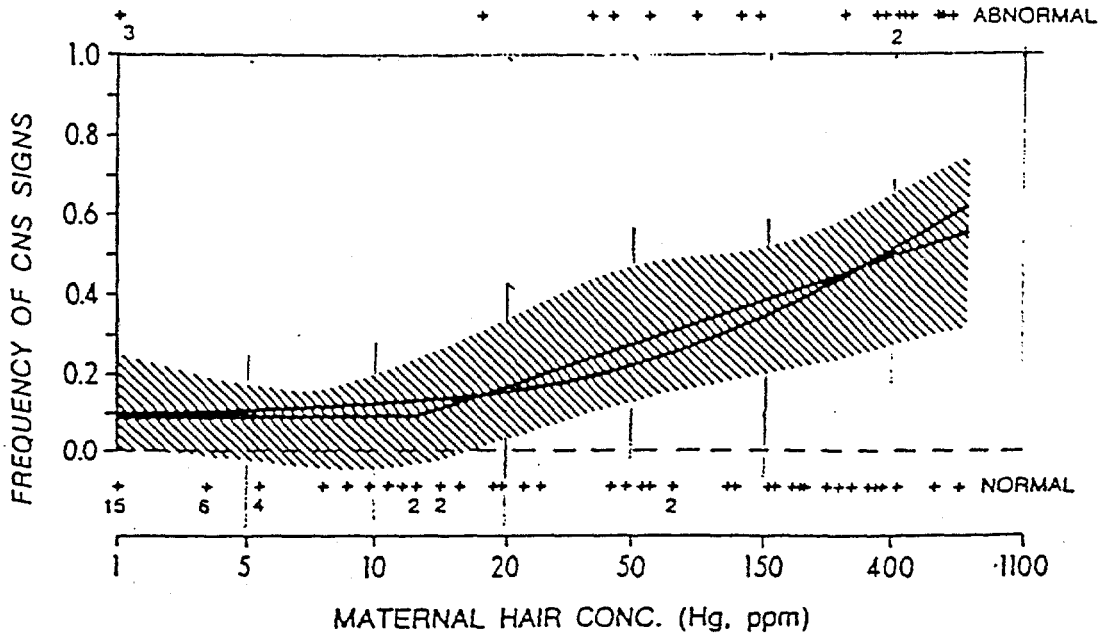


Figure 5. Exposure-response functions for the Iraqi data on prenatal exposures.[13] (a) late walking. (b) central nervous system signs. The solid lines are logit and hockey-stick fits; the shaded area represents 95% confidence limits from nonparametric smoothing analysis (reproduced with permission).

$$IQ = 0.33 \text{ (years in NZ)} + 0.37 \text{ (child's age)} - 4.7 \text{ (log[Hg])} + \text{dummy variables for island of origin.} \quad [2]$$

Similar regressions using the fish consumption index instead of log(Hg) were run; the fish consumption variable never achieved a *p* (significance) level below 0.25. An analysis of possible thresholds in the IQ exposure-response function suggested threshold values anywhere from 4-10 ug/g average maternal Hair Hg. The report of Lipfert[19] provides more details on the reanalysis of the New Zealand data.

Some of the problems with this study include the methods used to select "cases" (exposure rather than outcome), the lack of data on social class, and the possibility of postnatal exposures including exposures to other neurotoxins. Use of only the highest 0.7% of the hair Hg observations to define "cases" may place undue importance on whatever spurious high observations that may have been included. Nevertheless, the New Zealand data imply that average maternal hair Hg levels above about 3-10 ug/g (4.5-15 ug/g as peak hair levels, based on a factor of 1.5 to convert from average hair Hg to peak Hg levels during pregnancy, as suggested by Kjellstrom *et al.*[12]) may carry the risk of a small decrement in the mental development of the offspring, as typically measured by IQ.

Although each of these studies has certain flaws, they all support the onset of children's neurological effects at average maternal hair levels around 10 ug/g. The curve-fits to the Iraqi data by Cox *et al.*[17] seem to provide the best basis for quantitative risk assessment.

ESTIMATED HEALTH RISKS

Adults

The risk analysis simulations were performed for 5000 trials, intended to represent a hypothetical population of 5000 who derive a substantial portion of their fish diet from local waters. We then compare their risks with and without additional Hg deposition into these waters from a nearby coal-fired power plant. We examine the 99th percentiles of these exposure distributions; the statistics would become unreliable for probabilities further out on the "tail." All of the body burden predictions fell well short of the paresthesia threshold of 0.75 mg/kg (Figure 4). The 99th percentile body burden was 0.01 mg/kg for the baseline and was approximately doubled by deposition from the hypothetical power plant. These values are almost two orders of magnitude below the onset of adult paresthesia observed in Iraq.

Prenatal Exposures

The analysis of congenital health risks proceeds from the same simulations used for adults; no changes in diet or body weight were assumed for pregnant females. All of the exposure-response information on congenital effects was keyed to maternal hair concentrations. Fortunately, we have observations on the distributions of hair Hg to judge the acceptability of the simulated hair Hg distributions.

The simulations predicted average MeHg hair concentrations of 4.5 and 8.1 ug/g at the 99% level, for baseline and impact cases, respectively. We also noted that the baseline difference according to whether (predatory) fresh-water fish is consumed is larger than the effect of the hypothetical power plant. Estimates of the probability of neurological effects due to prenatal exposure may be made by combining the probabilities of exposure and the probability of effects. Using the data tabulated by Cox et al.[17] at a peak maternal hair level of 5 ug/g, we estimated overall risk probabilities of 0.0002 to 0.0004 for the baseline case and 0.0008 to 0.0017 for the impact case. These risk estimates depend somewhat on the intersection point between exposure and response that is selected for evaluation, as well as on the type of curve-fit used to extrapolate the original exposure-response data. The predicted congenital risks are about an order of magnitude higher than the predicted adult paresthesia risks based on similar extrapolations.

CONCLUSIONS

We conclude from this assessment that neurological health risks to adults in the United States from eating fish containing methylmercury are trivial, with or without the local effects of coal combustion. The margins of safety for congenital risks are lower, but this portion of the analysis is compromised by uncertainties in the indices of exposure used (maternal hair concentrations). We also conclude that, given the many elements in such risk assessment and the opportunities for error, it is essential to compare predictions with observations as a form of "reality check."

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