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4 Mercury by the Numbers

Michael C. Newman and Kenneth M. Y. Leung

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It is therefore worth while to search out the bounds between opinion and knowledge.

—John Locke, *An Essay Concerning Human Understanding*

INTRODUCTION

HARMFUL AMOUNTS OF MERCURY? BOUNDING OPINION AND KNOWLEDGE

Most personal decisions about how best to act are based on opinions of others, not rigorous examination of facts by oneself. Our world is too complex for each of us to examine exhaustively, assess, and then decide about every important issue. Such a tactic would soon result in dysfunctional indecision. Since all opinions do not emerge from the same amount of thoughtfulness and objectivity, the key to making wise decisions is understanding the nature of each opinion and then gauging how well it reflects practical knowledge. Making decisions about environmental issues is particularly difficult, but nonetheless very important to our well-being. Environmental issues permeate our laws, politics, economics, personal health, and collective ethos.

Mercury contamination is arguably one of the most prominent environmental concerns facing us today. Our collective attention was drawn to mercury in the late 1950s when an outbreak of human poisonings occurred in Minamata, Japan (Smith and Smith, 1975). Mercury has kept our attention since then, accumulating an overburden

of fact and opinion. These facts and opinions are applied to many decisions. Should one avoid having his or her child vaccinated for the H1N1 influenza because the mercury-based thimerosal preservative in some vaccines has been linked to autism? Should one reduce mercury in his or her diet by avoiding seafood that is also an excellent source of healthy omega-3 fatty acids? Does the mercury in the new low-energy lightbulbs obligate boycott of this product? Winnowing fact from opinion is crucial because mercury can present a real risk* to people and valued wildlife under specific circumstances.

Gathering sound facts and using them effectively to gauge risk is difficult without formal training. Without training the layperson resorts to what cognitive psychologists call informational mimicry (Richerson and Boyd, 2005; Vernimmen et al., 2005), that is, taking the position of an expert whom they trust for some reason such as past reliability or homophily.† This approach is sensible because mimicry is the best strategy when learning facts oneself is error prone and decision error carries substantial cost (Richerson and Boyd, 2005). Unfortunately the most accessible sources of environmental information are now Internet sites that differ widely in reliability and television news programs written as much to entertain as to inform. When confronted with such diverse and confounded information the opinion of most people tends to rely heavily on homophily. Information is sought during decision-making from comfortable and "credible" opinion leaders, and then explored with friends and neighbors with the intent of getting validation for one's emerging decision (Kasperon and Kasperon, 1996). To minimize postdecisional stress, the new opinion is then defended ad hoc from any new information. Unfortunately this common strategy is unreliable because the information for making important decisions about mercury varies so widely in quality and the reliance on homophily is excessive.

THE ADVANTAGE OF MEASURING

A precise statement can be more easily refuted than a vague one, and it can be better tested. This consideration also allows us to explain the demand that qualitative statements should if possible be replaced by quantitative ones.

—Popper (1972)

Given the above challenge, what is the best way to gather information about environmental mercury? The more explicitly a fact can be stated, the more easily its mettle is tested and the more unambiguous predictions can be made from it. Since quantitative facts or statements are easier to assess and use than are qualitative ones, the intent here is to delve into quantitative measurement and prediction of mercury's potential effects.

SOME CASES IN WHICH MEASUREMENT WAS UNNECESSARY

An argument could be made that demanding careful attention to measurement is unnecessary in many cases such as judging unacceptable the risk imposed by mercury-

* Risk is the probability or chance of a specific harmful effect or event occurring, for example, a one out of 100 chance of lung cancer if one is a heavy smoker.

† Homophily is the degree to which two or more interacting individuals are similar in relevant features such as education, professional affiliation, political beliefs, or religious upbringing.

tainted fish eaten by Minamata citizens in the 1950s through 1970s (Smith and Smith, 1975). Irreparable neurological damage occurred to young and old, leading to the current resounding conclusion of unacceptable risk. Yet even in this instance twelve years passed between the first recorded case of *in utero* mercury poisoning (1956) and when mercury was officially identified as the cause of the Minamata Disease outbreak (1968) (Smith and Smith, 1975). Minamata residents continued to debate the cause well after publication of the famous Minamata photographic exposé by Smith and Smith (1975). Therefore, even in the retrospectively most obvious of instances, quantification could have accelerated acceptance of the fact and motivated quicker risk reduction.

EVERYDAY CASES IN WHICH MEASUREMENT IS NECESSARY

Many instances exist in which measurement is desirable or needed to decide wisely about pollutant risk. Measurement can even provide an overarching context for discussing pollutant-related dangers. As one example from two decades ago, Figure 4.1 suggests that voluntary risks to life from tobacco use, poor diet, sedentary lifestyle, and alcohol abuse were much higher in the United States than that from all toxic agents

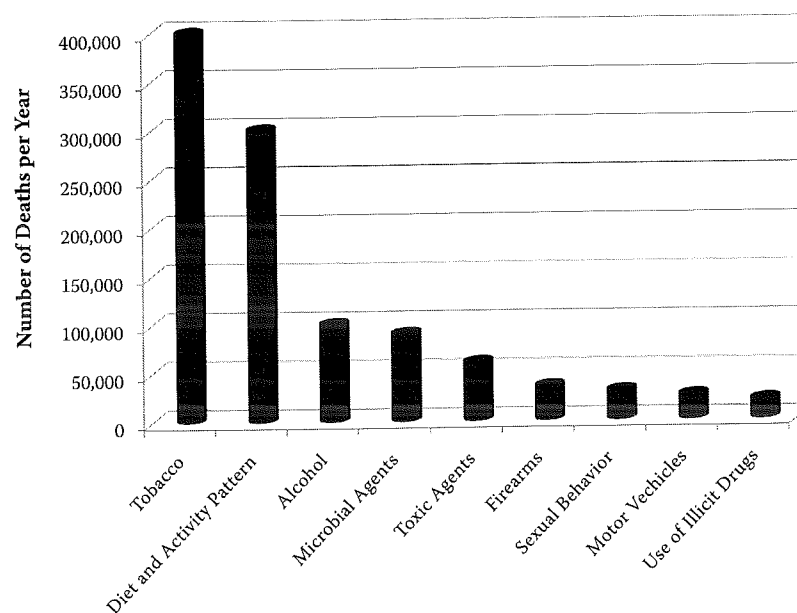


FIGURE 4.1 The most prominent root causes of death in the United States (1990). Deaths are expressed as simple numbers of deaths that year. These contributors account for approximately one-half of all deaths that year, i.e., Tobacco (19%), Diet and Activity Pattern (14%), Alcohol (5%), Microbial Agents (4%), Toxic Agents (3%), Firearms (2%), Sexual Behavior (1%), Motor Vehicles (1%), and Use of Illicit Drugs (<1%). Toxic-agent-related deaths include those associated with occupational exposures and consumer products in addition to deaths from environmental pollutants. Data from McGinnis and Foege (1993).

combined. Yet toxic agent exposure was and still is perceived as much less acceptable by Americans because it is an involuntary risk: One does not choose to take on toxic risk in order to reap the benefits of the technology that produced the toxicants.

Generally risks taken voluntarily by individuals are perceived as more acceptable than involuntary risks of equal seriousness (Gigerenzer, 2002). Acceptability of risk is not determined by objective fact alone: Many social and psychological factors influence a perceived risk's seriousness or acceptability. These include the ease with which the danger is visualized (Piatelli-Palmarini, 1994), past trustworthiness of any related institution (Beamish, 2001), an individual's openness to new technologies (Petrie et al., 2005), an individual's awareness of environmental issues (Gigerenzer, 2002; Winters et al., 2003), and the manner in which a risk situation is presented initially (Gigerenzer, 2002). Clearly the exposure to the toxicant and also any effect resulting from exposure must be measured or predicted quantitatively to minimize muddling misperceptions.

FRAMING THE QUESTION

It is also critical to understand the vantage from which a risk is being judged. Perhaps the best means of illustrating this point is to consider the belief that seat belts save lives. This pervasive belief is sound from the vantage of a person in a serious accident whose risk of death does decrease with seat belt use. But seat belts do not save lives from the vantage of the total number of vehicular fatalities in a country. According to British traffic expert John Adams, people wearing seat belts tend to feel safer and consequently drive less cautiously, resulting in more deaths to individuals in other vehicles or pedestrians struck by the less cautious, belted drivers (Adams, 1995). Thus, seat belts do save the lives from the vantage of those wearing them but do not save lives relative to the entire population of a country. As another example, the importance of vantage was obvious recently when proposed changes in screening for breast, prostate, and colon cancers generated an outburst of confusion in the United States, e.g., Marshall (2010). Epidemiologists had concluded that less intense screening protocols were the most sensible and cost-effective ways to address these cancers in the population. This announcement was met with suspicion based on testimonials from people who, without early detection through screening, would have had a much higher risk of dying. These contrasting decisions were a consequence of clashing vantages.

From the vantage of the health care community, some of the money spent in the current cancer screening regimes would be better spent on other important health risks. Using prostate cancer as an example, Anriole et al. (2009) found no difference in death rates from prostate cancer between groups differing in the diligence of prostate-specific antigen and digital rectal examination screening regimes. So from the vantage of managing the risk of death for the entire population from all diseases, the conventional screening is best replaced by the less intense screening.*

* Complicating the issue further, Schroder et al. (2009) found a 20% lowered death rate with prostate specific antigen (PSA)-based screening for prostate cancer but noted that this improvement came at the elevated risk of overdiagnosis. Overdiagnosis carries consequences such as septicemia or excessive bleeding after biopsy.

Equally valid were the conclusions in testimonies of individuals for whom early detection greatly decreased their personal risk of death from cancer. Risk of death was reduced substantially for the individual who actually had the cancer for which screening was being done. However, the number of saved lives per unit of resources spent to screen was much lower than the number saved if some resources were refocused in other health issues. The costs were worth it from the afflicted individual's vantage but not from that of the population as a whole. Nor was the original screening regime worthwhile for the healthy individual. The risk of diminished health for an individual with a false positive screening result actually increases due to possible complications during follow-up biopsies (Schroder et al., 2009). Finally, the current screening regimes were not advantageous from the vantage of an individual whose life might have been saved by increased screening intensity for another deadly disease using reallocated funds. Hence, although this is seemingly counterintuitive, benefit and risk depend profoundly on vantage.

Understanding the vantage from which risk is framed is also critical for the management of environmental toxicants. The mercury in tainted fish eaten by Minamata victims constituted an unacceptable risk to the Japanese citizens; however, any extremely small increase in risk of autism from vaccination is trivial relative to risks from the diseases (diphtheria, tetanus, pertussis, or H1N1 influenza) for which vaccination is recommended. The benefit in lives saved to the entire population of children is also much higher than the dubious risk of autism to one's vaccinated child given that recent epidemiological evidence has confirmed that low-level mercury poisoning in children is not a cause of autism (Ng et al., 2007). Also, the risk from mercury to a population consuming modest amounts of seafood might be trivial but that to a subpopulation eating large amounts of seafood should warrant careful scrutiny. Likewise, the risk to a particularly vulnerable subpopulation such as unborn children exposed to mercury via food consumption by their mother should warrant much more scrutiny than that to a mature adult. Again, only the clarity associated with quantifying risk makes everything obvious from these different vantages. Without this decisions are merely based on insubstantial opinion and confused vantages.

MEASURING EFFECT LEVELS

What is needed to estimate risk from environmental mercury? The most important mercury source for the general public is seafood so information is needed about mercury in seafood, seafood consumption rates for different groups of people, and the relationship between the amount of mercury to which a person is exposed and the likelihood of it manifesting some adverse effect. For humans this risk might be estimated from the vantage of the entire population (e.g., average US citizen), a sensitive subgroup (e.g., women of childbearing age), or a highly exposed subpopulation (e.g., fishermen or recent Vietnamese residents of the United States who consume more fish than the average citizen). Although human risk assessments require careful consideration of vantage, establishing the vantage for ecological risk estimation requires even more care because exposure might involve many other sources of mercury (i.e., via air, water, soil/sediment, or food chain) and different organisms that vary widely in terms of their mercury exposure route, uptake rate, and sensitivity.

HUMAN HARM

Existing exposure and effects data allow reasonable estimates of mercury risk. Initial efforts to set the limits for amount of mercury that can be ingested (expressed as oral reference doses or RfDs) drew on an accidental poisoning of Iraqis who unknowingly milled methylmercury-fungicide-treated seed into baking flour (Bakir et al., 1973). The limits for daily mercury ingestion in food (0.0001 mg/kg of body weight per day) were calculated after neurological testing of 81 children born to mothers who mistakenly consumed different amounts of this flour while pregnant (Bakir et al., 1973; Crump et al., 2000).*

Reluctance to treat these estimates as definitive was founded on the nature of the exposure. The Iraqi poisonings involved brief and intense exposures but those for which we commonly wish to estimate risk are exposures to low concentrations in seafood for long periods of time. Epidemiologists sought out communities that consumed high amounts of seafood with the intent of quantifying ingestion limits more definitively. Again, the vantage was harm to unborn children from a mother's exposure during pregnancy. Mother-child pairs from the Seychelles Islands in the Indian Ocean (Davidson et al., 1998; Crump et al., 2000; van Wijngaarden et al., 2006) and from the Faroe Islands in the North Atlantic (Grandjean et al., 1997) were meticulously monitored for mother exposure levels and tested for neurological impairment of their children. Inhabitants of the Seychelles consume fish that have mercury concentrations similar to those in the US market, but they consume 10 to 20 times more fish than most US citizens (Davidson et al., 1998). The Nordic inhabitants of the Faroe Islands consume large amounts of fish; however, they also consume pilot whale meat that contains high mercury concentrations in the range of 1.6 µg/g. Both of these epidemiological studies produced a more definitive and directly relevant limit for mercury ingestion (0.0001 mg/kg maternal body weight daily).† Ingestion rates above this limit might carry unacceptable risk to unborn children (Crump et al., 2000; van Wijngaarden et al., 2006).

The United States Environmental Protection Agency (US EPA) maintains an Integrated Risk Information System, or IRIS, that synthesizes these and other study results from the scientific literature. It can be accessed at this time from <http://www.epa.gov/ncea/iris/index.html>. Information for quantitatively estimating the limits for mercury exposure can be found by entering "Methylmercury (MeHg)" into the IRIS front page search tool. A summary document or full report on methylmercury is obtained by simply choosing one or the other on the Advanced Search Results from the methylmercury search. For example (Figure 4.2) the summary provides the daily oral reference dose (RfD) of 0.0001 mg of mercury/kg of person body weight per day.

* This corresponds to a maternal hair mercury concentration in the range of 10 to 20 µg/g (Grandjean et al., 1997), which is in the range of the current health criteria for pregnant women (Fujino, 1994). Hair mercury concentrations were as high as 674 µg/g for poisoned Iraqis, and at that level were linked to seizures, cerebral palsy, and other serious consequences to victims of acute poisoning (Davidson et al., 1998). Adult Japanese displaying neurological or neuropsychiatric disturbances notionally from chronic exposure to mercury in seafood had hair mercury concentrations as high as 37.4 µg/g, and averaging 7.9 µg/g in women and 11.6 µg/g in men (Fujino, 1994).

† This corresponds to maternal hair mercury concentrations of approximately 20 to 25 µg/g of hair.

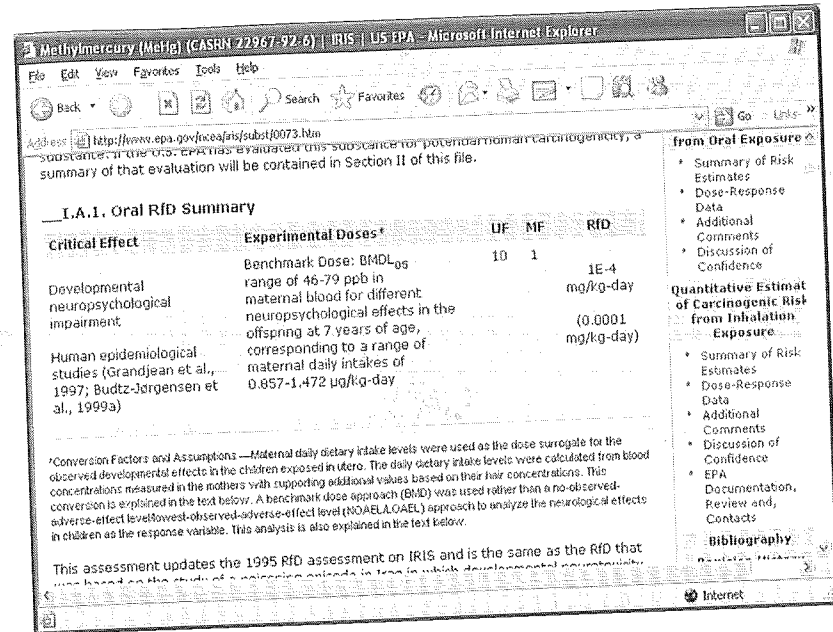


FIGURE 4.2 Screen shot of US EPA's IRIS methylmercury summary for human oral ingestion.

This dose is based on the aforementioned epidemiological study of neuropsychological effects on seven-year-old children born of Faroe Islands mothers whose estimated daily dietary intakes during pregnancy ranged from 0.00086 mg to 0.00147 mg mercury/kg body weight (Budtz-Jørgensen et al., 1999; Grandjean et al., 1997). The mother's dose of concern was denoted the Benchmark Dose (BMDL₀₅), a dose with an excess risk of 5% for animals with abnormal levels. The dose (BMDL₀₅ = 0.00086 mg/kg-day) for this case was divided by a conservative uncertainty factor (UF) of 10 to produce the final reference dose of 0.000086, which is rounded to 0.0001 mg/kg-day. Risk of neurological harm to an unborn child is judged as potentially unacceptable if a mother eats more than that amount of mercury each day while pregnant.

ECOLOGICAL HARM

Risk to valued ecological entities, such as a charismatic species of fish hawk, is much more difficult to quantify although direct laboratory exposures of nonhuman species are possible whereas deliberate laboratory exposures of humans would be unethical. Regardless, there are so many animals and plants to consider that information is often inadequate to accurately estimate ecological risk for any particular species. Consequently, ecological risk assessments draw more from the original primary reports in the literature than from orderly compilations of information such as IRIS. The US EPA does have a data compilation tool called ECOTOX that is accessible from <http://cfpub.epa.gov/ecotox/>. A Quick Database Query specifying "Animals → Chloromethylmercury → Endpoint Reported → All Effect types → Publications from

1980 to 2009 → Viewable Table” will yield a table of effect/concentration data for a series of organisms. (Click on the Key Functions and then Query for Aquatic Biota to begin the search.) The resulting data will be a mixture of various measures of effect at specified mercury concentrations. For aquatic organisms, concentrations will be predominantly for methylmercury dissolved in water. This compromises many ecological risk assessments because most exposure of aquatic animals to methylmercury occurs from ingestion of contaminated food. You can do the search again and specify terrestrial species to get information on land organisms. The results will be biased toward bird species because they tend to be the focus of public attention during ecological risk assessments. Some results will be expressed as the median lethal concentration (LC50), median effect concentration (EC50), no observed effect level (NOEL), or lowest observed effect level (LOEL) values. The LC50 is the concentration calculated to kill half of the individuals exposed to that concentration, and the EC50 is the concentration that causes adverse effect (e.g., growth inhibition or reproductive impairment) to half of the test population. The LOEL is the lowest test treatment level in a toxicity test at which a statistically significant effect was noted and the NOEL is the next lowest test treatment level below the LOEL (Figure 4.3). Like the human health assessment community (Filipsson et al., 2003; Crump, 1984), ecological risk assessors are gradually coming to realize that LOEL or NOEL measures are compromised (Newman, 2008; Warne and van Dam, 2008) and should be replaced by metrics similar to benchmark doses.

But what exactly are these measures of effect? This question can be answered with a single example (Figure 4.3). This figure depicts data from a fictitious study quantifying mercury effect at different concentration or dose treatments. Five treatment groups of ducklings, including a control group, are fed different doses or concentrations of methylmercury for a set amount of time and then some indicator of neurological harm is measured for each of ten ducklings in each of the three replicates for each group. Some expression of the number of afflicted ducklings (mean and standard error) is plotted against the dose. To estimate the benchmark dose (i.e., BMD) for a predetermined effect level such as 5% of ducklings afflicted or 5% decrease in the neurological function, some regression model is fit to these data and a prediction made for the 5% level. Often the prediction is made in a conservative way by using some highest reasonable slope such as the statistical upper limit of the estimated model slope. The intent is to err on the side of safety in predictions of the dose (or concentration) at which that level of effect might be expected. The median lethal dose (LD50) or LC50 is also estimated from the same regression model. It is the concentration or dose predicted to kill 50% of the exposed ducklings. Sometimes, as in this example, the effect might not be lethal and an effective dose or concentration (ED50 or EC50) would be calculated.

Sometimes a model is not assumed and instead the effect noted in each treatment is tested statistically to see if it is different from the effect measured in the control treatment (e.g., using one-way analysis of variance and Dunnett’s post hoc multiple comparison test). Effect levels in some treatments might be significantly different from the control (denoted in Figure 4.3 with an “S” next to the data point) while others might not be. The lowest treatment dose or concentration with a significant difference is called the LOEL and the next lower treatment down is called the NOEL.

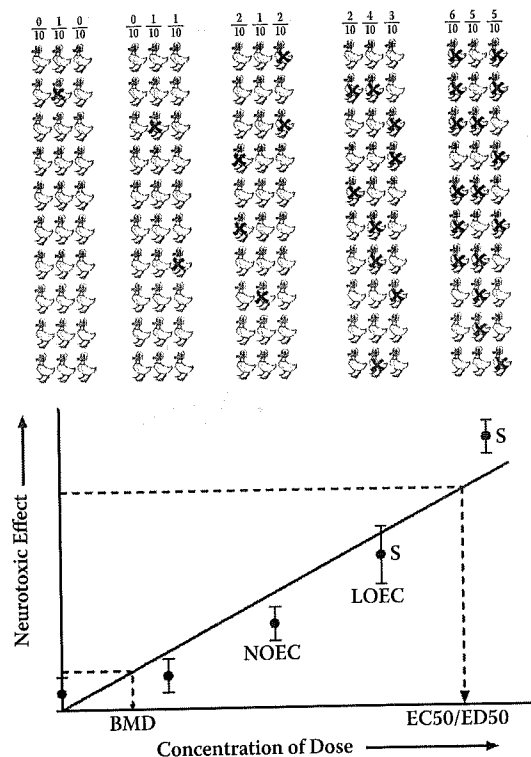


FIGURE 4.3 Analysis of data from a fictitious experiment quantifying neurotoxicity in ducklings fed different doses or concentrations of methylmercury. Each treatment involved three replicates of ten ducklings exposed for a specific time period and then examined for neurological effect. For the sake of clarity, a straight line model is fitted to the mean values (\pm standard error) of the neurological effect response (lower panel), although sigmoid (S-shaped) models are often applied to these types of data. At the top of this figure is a depiction of the ten ducklings per replicate with the numbers adversely affected per ten ducklings shown, e.g., 0/10 1/10, 0/10, etc. With the neurological effect response for each treatment, it is feasible to determine the NOEL and LOEL using analysis of variance and Dunnett’s test and compute the BMDL₀₅ and EC50/ED50 values using the regression model. (See text for more explanation.)

The LOEL and NOEL are thought to define the interval containing the threshold level needed to get an adverse effect. However, there are many difficulties with this approach that are discussed in publications such as Newman (2008) and Warne and van Dam (2008).

A good illustration of currently deficient information for doing risk assessments is the database for birds. Often, but not always, the species for which data are first generated are those easily reared and exposed in the laboratory. Accordingly, feeding trials for the mallard duck (*Anas platyrhynchos*) indicate that ducklings from hens fed 3 $\mu\text{g/g}$ of mercury (as methylmercury) had abnormal avoidance behavior (Heinz,

1976). Ducklings with neurological indications of methylmercury poisoning were associated with eggs having 2 µg/g of mercury due to hen exposure through their feed (Heinz and Hoffman, 2003). Mallard hens receiving 0.5 µg/g as methylmercury in their feed laid fewer eggs and produced fewer ducklings than control hens, and their ducklings were hyper-responsive to alarm stimuli (Heinz, 1979).^{*} These results were similar to those with young captive egrets (*Ardea albus*) fed 0.5 µg/g (as methylmercury) that displayed depressed general activity and prey hunting behavior relative to control egrets (Bouton et al., 1999). This 0.5 µg/g was also similar to those of egret prey species in the Florida Everglades. Surveys similar to the epidemiological studies described for humans are also done to determine concentrations resulting in effects to other species. For example, the survey of common loons (*Gavia immer*) by Meyer et al. (1998) suggested that egg mercury concentrations in lakes with lower reproductive success were similar to egg concentrations in experimentally dosed birds showing an adverse effect (Fimreite, 1971). Comprehensive surveys exist for blood, feather, and egg concentrations in field populations of this fish-eating bird (Evers et al., 1998; Meyer et al., 1998). Laboratory exposures of loons and examination of their chicks suggest mercury concentrations to which loons were exposed might decrease the immunological competency of the chicks (Kenow et al., 2007a).

MEASURING EXPOSURE

The co-occurrence of mercury and an organism does not necessarily mean that that organism is exposed in a way that will cause harm. The form of mercury and the nature of the contact are extremely important in determining the realized exposure.

Mercury can be present in inorganic and organic forms that differ widely in their ability to enter an organism and cause harm. For example, the mercury[†] in silver-mercury amalgam dental fillings is only modestly available to enter the bloodstream (Gundacker et al., 2006), move into nervous tissues, and cause neurological harm. Mercury combined with selenium (i.e., HgSe) is also extremely unavailable. Indeed, toothed whales have evolved a detoxification mechanism that incorporates mercury into inert HgSe granules in tissues (Mackey et al., 2003). At the other extreme, contact with just a minuscule amount of the extremely bioavailable dimethylmercury can kill a person.[‡] Dimethylmercury is very soluble in lipids such as the oils and fats in our cells. It evaporates readily and can be inhaled. Anyone in contact with dimethylmercury will quickly absorb it through the skin or lung surfaces, and the compound will move through the circulatory system to nervous tissue where it can cause lethal

^{*} The mercury concentration of 0.5 µg/g in the feed can be converted to an ingestion rate as described later for humans. These mallard duck hens ate an average of 156 g of feed/kg body mass each day. So 0.5 µg/g × 156 g/kg-day = 78 µg/kg-day or 0.078 mg/kg-day of mercury as methylmercury was the ingestion rate associated with these adverse effects. This also corresponded with approximate mercury concentrations of 10 µg/g of hen primary feathers and 0.8 µg/g wet weight in eggs (Heinz, 1979).

[†] This elemental mercury is in the form, Hg⁰, that is a mercury atom with no charge. The other common form of inorganic mercury in the environment is divalent mercury, Hg²⁺, which can bind to sediments, soils, and molecules in tissues.

[‡] Mercury can combine with one or two methyl- groups to form either monomethylmercury (HgCH₃) or dimethylmercury (H₃CHgCH₃). Both are called methylmercury but the most common form in the environment is monomethylmercury.

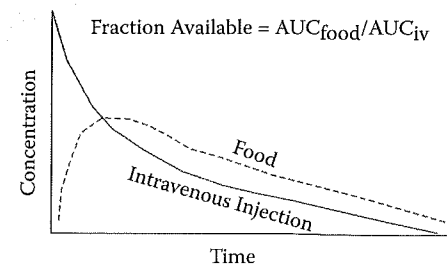


FIGURE 4.4 An example of quantifying bioavailable mercury in food. A dose of mercury (e.g., 0.5 mg of mercury per kg body weight) is injected directly into the bloodstream and the decrease in blood mercury concentration measured over time (solid line). The area under the curve (AUC) is estimated with a model or the simple trapezoid method. The same dose is now administered in the food item of interest and the curve of blood mercury concentration monitored (dashed line). The AUC is estimated for this second curve. By definition the amount of mercury injected into the blood stream is 100% bioavailable so the quotient, $AUC_{\text{food}}/AUC_{\text{iv}}$, is an estimate of the fraction of the ingested dose available to enter the blood stream.

damage. Sadly this was the case in 1996 when brief contact of a toxicology professor's gloved hand with a drop of spilled dimethylmercury led to her death within ten months (Nierenberg et al., 1998).^{*} Intermediate between the bioavailabilities of the mercury in HgSe granules and dimethylmercury is that of monomethylmercury. Monomethylmercury, along with inorganic forms of mercury, comprises most of the mercury found in seafood (Mason et al., 2006). It is bound to sulfur-rich molecules in tissues such as proteins or glutathione (Harris et al., 2003) and is thought to be relatively bioavailable after ingestion.

But descriptors such as “sparingly,” “modestly,” “extremely,” or “relatively” bioavailable are too vague to help in risk assessments; therefore, quantitative measures of bioavailability were created. Usually toxicant bioavailability after ingestion, inhalation, or absorption across the skin is quantified as that proportion of the mercury that gets into the bloodstream (Figure 4.4). This fraction is important to quantify, but a risk assessor might be forced to assume that 100% of the mercury in a source is bioavailable if no bioavailability estimate exists.

Other qualities of the contact with the mercury also need to be understood in order to estimate risk. This is easiest to illustrate with the equation often used for seafood ingestion by regulatory agencies,

$$\text{Intake (mg/kg per day)} = \frac{(\text{CF})(\text{IR})(\text{FI})(\text{EF})(\text{ED})}{(\text{BW})(\text{AT})}$$

where CF = concentration in the food (mg/kg) that might be modified with the bioavailability fraction to the concentration of bioavailable mercury if only a portion of

^{*} Peak hair mercury concentrations (approximately 1100 µg/g) were reached in a little more than a month after the accidental exposure.

the mercury in a food is bioavailable; IR = ingestion rate (kg/meal), e.g., 0.284 kg/meal (upper 95% confidence level for fish) consumption; FI = fraction ingested from the contaminated source (no units to this fraction); EF = exposure frequency (meals/year); ED = exposure duration (years), e.g., 0.75 for a human gestation period; BW = body weight or mass (kg), e.g., 70 kg for an adult; and AT = averaging time (days), e.g., $ED \times 365$ days.

Information needed to estimate intake is generated carefully for the general population or for a particular subpopulation of concern. Conservative values might be entered into the equation if information for the general population is compiled. As an example, the use of a value at the upper 95% confidence limit for the mean seafood ingestion rate for the public can embed another safety factor in the risk assessment process.

HUMAN EXPOSURE

Many research programs generate the information needed to estimate human exposure through fish consumption. Most often the emphasis is on maternal exposure during pregnancy so ED is 0.75 years and AT is 365 (0.75), or 274 days in the above equation. Remaining undefined after this vantage is established are CF, BW, and factors related to consumption habits (IR, FI, EF). These pieces of information are sometimes applied in the form of means or medians; however, increasingly often they are applied as entire distributions using computer simulation methodologies (e.g., Monte Carlo simulation).

Body weight of women is the most obvious item in the equation with which to show the advantage of considering information as distributions. A generic body weight (BW) of 70 kg is often not accurate enough because, as in the case of North American women of child-bearing age, body weights are usually lower (mean: 60.6 kg) and can vary widely (standard deviation: 11.9 kg) (US EPA, 1997). Whether the weight is selected from the center or some other position of the distribution for the population will depend on the consequences of being wrong and the specific group for which risk is being estimated. To be conservative an assessor might pick a smaller woman's weight, such as that associated with a woman in the lower 10% of the population weight distribution.

Mercury concentrations in the ingested food (CF) also vary within and among seafood species. Considerable effort is exerted to survey mercury in potential seafood species. Surveys might emphasize the countrywide seafood market (e.g., Sunderland, 2007), a particular regional market (e.g., Hammerschmidt and Fitzgerald, 2006; Mason et al., 2006; Cheung et al., 2008; Monson, 2009; Peterson et al., 2007), the species eaten in a contaminated region (e.g., Amazon fishes studied by Regine et al., 2006; and Wujiang River fishes surveyed by Li et al., 2009), species high in mercury (e.g., tuna studied by Krapiel et al., 2003), or a species consumed by a particular subpopulation (e.g., small cetaceans eaten by Japanese as noted in Endo et al., 2005). The concentration used in the equation might be a composite of concentrations in different items eaten by the population of interest. A careful assessment might require using distributions of mercury concentrations in many different seafood items (Tang et al., 2009).

Careful surveys of the general population or specific populations (e.g., secondary school students of Hong Kong (Tang et al., 2009)) are used to establish information about ingestion rate (IR), fraction taken from the source of concern (FI), and the exposure frequency (EF). The detail and breadth required in a particular survey depends on the context. An initial estimate of exposure might be generated with information about the average US woman (i.e., 1.8 g/day) (Holloman and Newman, 2010). Ingestion rates for specific seafood items may be used if there are material differences in mercury concentrations in or preference for certain seafood items (US EPA, 2003; Tang et al., 2009). However, results based on generic information could be misleading in some situations. For example, the Japanese noted as having neurological dysfunction (footnote on page 56, this volume) had ingested seafood at a rate of 333.6 g/day (Fujino, 1994). African-American women in Newport News, Virginia, eat 147.8 g/day of seafood (Holloman and Newman, 2010). Relative to EF, Swedish women surveyed by Bjornberg et al. (2005) ate fish 1.6 to 19 times weekly, but women living along the St. Lawrence River ate fish less frequently (0 to 7.9 times weekly) (Morrissette et al., 2004). Similarly, secondary school children of Hong Kong varied materially in their fish consumption rate with estimated dietary methylmercury exposures ranging from 0.4 to 0.5 $\mu\text{g}/\text{kg}$ body weight-weekly (for average consumers) to 1.2 to 1.4 $\mu\text{g}/\text{kg}$ body weight-weekly (for high consumers) (Tang et al., 2009). In such cases the vantage might be refined so as to better assess the health hazard to each subpopulation of concern.

Less detailed exposure assessments are done, but they provide less insight for fully understanding the exposure. This lack of detail can make it difficult to determine what changes in behavior might reduce risk. An important, previously mentioned example was exposure expressed as mercury concentration in hair (Table 4.1). Maternal blood, umbilical cord blood, fingernails, and other samples might be considered as surrogates for exposure concentrations and correlated with effects instead of doing the exhaustive data gathering needed to apply the above ingestion rate equation (e.g., Fok et al., 2007).

ECOLOGICAL EXPOSURE

Exposures of the many valued ecological entities by a variety of pathways are extremely difficult to calculate accurately except in the most fortuitous of situations. The epidemiological surveys, similar to those done to define human exposures, are important, but laboratory experiments are also prominent for nonhuman species' exposures. Because so many species must be considered, information for a surrogate species is commonly employed. For example, exposure information for a fish hawk might be collected with the intention of comparing it to exposure-effect information generated in the laboratory for mallard ducks; marine minks might be used as surrogates for dolphins and seals (Hung et al., 2007). Often an ecological risk assessor is forced into the awkward logic of "it doesn't walk or quack like a duck but, for purposes of this assessment, it's a duck." The mercury in food eaten by mallard ducks could reasonably be assumed to have a different bioavailability when compared with that in the fish eaten by a fish hawk. Obviously, the sound work with mallard ducks is directly relevant to this and similar important duck species but questionable in other

TABLE 4.1
Total Mercury Concentration in Human Hair as a Measure of Exposure

$\mu\text{g Hg/g Hair}$	Study Subject	Reference
0.09	Pregnant women living along St. Lawrence River, GM	Morrisette et al. (2004)
0.12	US children ages 1–5 years (1999–2000), GM	McDowell et al. (2004)
0.20	US women ages 16–49 years (1999–2000), GM	McDowell et al. (2004)
0.38	US women as above but frequent fish consumers, GM	McDowell et al. (2004)
0.38	Vegetarian (vegan) men in Hong Kong, Mean	Dickman et al. (1998)
0.7	Swedish women, Med	Bjornberg et al. (2005)
1.9	Coastal Brazilian population, Mean	Nilson et al. (2001)
1.6	Hong Kong women, Mean	Dickman et al. (1998)
1.6	UK individuals eating fish 1 to 4 times monthly	Airey (1983)
2.4	US individuals eating fish 1 to 4 times monthly	Airey (1983)
2.5	Australian eating fish 1 to 4 times monthly	Airey (1983)
2.6	Fertile Hong Kong men, Mean	Dickman et al. (1998)
4.5	Subfertile Hong Kong men, Mean	Dickman et al. (1998)
4.3	Faroe study, new mothers, GM	Grandjean et al. (1997)
6.8	Maternal hair from Seychelles study	Davidson et al. (1998)
7.9	Men, Katsurajima Island, Japan (1974–79), Mean	Fijino (1994)
10	Maternal hair health upper limit, Faroe study	Grandjean et al. (1997)
11.6	Women, Katsurajima Island, Japan (1974–79), Mean	Fijino (1994)
25	Estimated to correspond with current ingestion limit	Crump et al. (2000)
0.9–28.5	Range in Peruvian fishing village of Mancora	Marsh (1995)
21.5–33.9	Range in Wuchuan mining area of Guizhou, China	Li et al. (2008)
37.4	Highest value, Katsurajima Island, Japan (1974–79)	Fijino (1994)
100–191	Asymptomatic Minamata residents, 1960 survey	Smith and Smith (1975)
0.16–199	Range in Wujiazhan, China (mean = 3.41)	Zhang and Wang (2006)
96.8–705	Symptomatic Minamata residents, 1960 survey	Smith and Smith (1975)
674	Iraqis poisoned by methylmercury-treated grain	Davidson et al. (1998)
1100	Maximum during acute dimethylmercury poisoning	Nierenberg et al. (1998)

Note: Maternal hair mercury concentration in the range of 10 to 25 $\mu\text{g/g}$ is indicative of possible harm to unborn children based on compilations of these types of postexposure data.

GM = geometric mean, Mean = arithmetic mean, Med = median

instances. Much effort is being spent at this time to produce information needed to reduce the inaccuracy of such compromised assessments.

However, information directly useful for relevant species is increasingly being produced for assessing mercury exposure. As an example, the information base for the fish-eating common loon, *Gavia immer*, is growing rapidly. Surveys have been

published that describe the geographic distribution of mercury measured in feathers and blood of North American loon populations (Evers et al., 1998). The exposure measures of feather and blood mercury have been correlated in field surveys with adult loon reproduction and survival (Meyer et al., 1998). Results from these field studies have been integrated with those from laboratory exposures of loon chicks to suggest linkage between mercury exposure and adverse effects (Kenow et al., 2007a, 2007b). Another example is exposure information (often hair concentrations) of wild mammals such as mink (Moore et al., 1999), arctic fox (Fuglei et al., 2007), and polar bear (Dietz et al., 2006).

CONCLUSIONS

Selecting an appropriate vantage and then quantifying exposure and an associated effect level are essential to understanding enough to make a reasonable judgment about risk from environmental mercury. Dependence on unreliable opinions and widely divergent information is the only other option in the absence of these essential steps. Fortunately enough information is slowly emerging to make wise decisions for environmental risk assessment of mercury. A well-informed quantitative risk assessment not only reveals the current hazard of mercury to human populations and ecosystems of concern, but also provides essential information such as recommended safe food items and their allowable intake rates for effective risk management of environmental mercury. This short chapter sketches out the associated information and provides the general quantitative approach. The interested reader is urged to explore the references cited below for more information.

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MERCURY POLLUTION

A Transdisciplinary Treatment

Edited by

Sharon L. Zuber and Michael C. Newman



CRC Press
Taylor & Francis Group
Boca Raton London New York

CRC Press is an imprint of the
Taylor & Francis Group, an **informa** business

CRC Press
Taylor & Francis Group
6000 Broken Sound Parkway NW, Suite 300
Boca Raton, FL 33487-2742

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Printed in the United States of America on acid-free paper
Version Date: 20110711

International Standard Book Number: 978-1-4398-3384-1 (Hardback)

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Library of Congress Cataloging-in-Publication Data

Mercury pollution : a transdisciplinary treatment / editors, Sharon L. Zuber and Michael C. Newman.
p. cm.

Summary: "Using mercury pollution as an example, this book illustrates the interdisciplinary approach required for solving environmental problems. It explains the details of the natural cycling of mercury in and on the earth and discusses how humans have dramatically disrupted its exchange among the earth's soil, air, and water. The chapters discuss history, media, and politics in relation to mercury and contain links to established websites with specific resources for readers. Also included are smaller case studies, such as the Minamata tragedy, fish consumption, and international treaties"-- Provided by publisher.

Summary: "Mercury is the gravest chemical pollutant problem of our time, and this is the first publication that has undertaken holistic coverage of this truly global issue. This book is an outstanding product of the innovative Global Inquiry Groups (GIG) process of interdisciplinary collaboration, which brings together the physical sciences, social sciences, and the humanities including the study of media, visual, and literary arts, uniquely enabling us to look at and understand mercury from the many perspectives needed in order to grasp the problem in its totality. This book clarifies how we are all connected to mercury, how we take it in through the food we eat and the air we breathe, and how we release it as a consequence of our lifestyle. It tells us about the relationship people have had with mercury from ancient to modern times. It tells us how people have given artistic expression to the ravages of mercury, touching our emotions and changing us. It refers to the social injustice of mercury pollution. This book is an attempt to communicate beyond the walls of academia to a larger audience. Therefore, on behalf of the authors of chapters within this book and the mercury group, I invite you to explore metal mercury from the varied perspectives of history, science, sociology, government, writing, and art. Each chapter represents a disciplinary thread from the GIG. Our goal was to weave these interdisciplinary threads into a tapestry that presents a more complete picture of the effects of mercury pollution and to provide new ways to think about the environment and our individual responsibility toward each other and our earth"-- Provided by publisher.

Includes bibliographical references and index.

ISBN 978-1-4398-3384-1 (hardback)

1. Mercury--Environmental aspects. 2. Mercury--Toxicology. 3. Mercury--Health aspects. I. Zuber, Sharon L. II. Newman, Michael C.

TD196.M38M4686 2011
363.7384--dc23

2011012189

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