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# Global Health Impacts and Costs Due to Mercury Emissions

**Joseph V. Spadaro[1] and Ari Rabl[1]***

Since much of the emission is in the form of metallic Hg whose atmospheric residence time is long enough to cause nearly uniform mixing in the hemisphere, much of the impact is global. This article presents a first estimate of global average neurotoxic impacts and costs by defining a comprehensive transfer factor for ingestion of methyl-Hg as ratio of global average dose rate and global emission rate. For the dose-response function (DRF) we use recent estimates of IQ decrement as function of Hg concentration in blood, as well as correlations between blood concentration and Hg ingestion. The cost of an IQ point is taken as $18,000 in the United States and applied in other countries in proportion to per capita GDP, adjusted for purchase power parity. The mean estimate of the global average of the marginal damage cost per emitted kg of Hg is about $1,500/kg, if one assumes a dose threshold of 6.7 $\mu$g/day of methyl-Hg per person, and $3,400/kg without threshold. The average global lifetime impact and cost per person at current emission levels are 0.02 IQ points lost and $78 with and 0.087 IQ points and $344 without threshold. These results are global averages; for any particular source and emission site the impacts can be quite different. An assessment of the overall uncertainties indicates that the damage cost could be a factor 4 smaller or larger than the median estimate (the uncertainty distribution is approximately log normal and the ratio median/mean is approximately 0.4).

**KEY WORDS:** Damage costs; dose distribution; dose-response function; global modeling; IQ decrement; mercury; methylmercury; transfer factor; uncertainty

## 1. INTRODUCTION

There has been much debate, especially in the United States, about proposed stricter regulations for the emission of Hg (in the United States the Clean Air Mercury Rule went into effect in 2005), and a large number of studies have been performed, for example, Lipfert *et al*. (1996), Trasande *et al*. (2005), Rice and Hammitt (2005), and Griffiths *et al*. (2007). An important consideration in this debate is the comparison of abatement costs and benefits. Estimating the benefits is challenging: the modeling of the environmental pathways is very complex and uncertain, and the dose-response functions (DRF) are poorly known. A further complication arises from the fact that Hg is a global pollutant (Lamborg *et al*., 2002) and therefore the benefits should be evaluated at the global scale, something that has not been attempted before. More precisely, it is Hg(0), the gaseous metallic part of Hg emissions, that is dispersed globally because its effective atmospheric residence time (including resuspension) is about one to two years, long enough for the distribution in the hemisphere to become quite uniform. The other main species are RGM (reactive gaseous mercury $Hg^{++}$) and $Hg_p$ (particulate mercury), and they deposit regionally rather than globally. The speciation of Hg emissions depends on the source, but the contribution of Hg(0) to the total is large.

Even if the United States or the EU were entirely selfish, it would be appropriate for them to consider the emissions from and impacts in other countries, for at least two reasons: first, since the problem is global,

[1] ARMINES/Ecole des Mines de Paris, Paris.
*Address correspondence to Ari Rabl, ARMINES/Ecole des Mines de Paris, 60 boul. St.-Michel, F-75272, Paris; tel: (33)6-3263-0431; ari.rabl@gmail.com.

 

emission reductions need to be negotiated by all concerned parties; second, the most certain contribution to the damage cost (the one evaluated in this article) is a reduction of the IQ, entailing a delay in economic and social development that leaves everybody worse off. Of course, for international agreements on emission reductions one would need an assessment of the contributions of the concerned countries to the impacts in other countries. Such an assessment would be difficult and far beyond the scope of this article, which intends only to highlight the importance of the problem by presenting a first order-of-magnitude estimate of the global average health damage costs of Hg emissions.

We argue that a global estimate is relatively simple because the dominant pathway is the ingestion of fish and seafood, and the transfer factor from emission to ingestion can be estimated by comparing the global average dose and emissions, both of which have been estimated in a number of studies. Such a comprehensive transfer factor is much easier to determine and less uncertain than a detailed calculation of all the individual transfer factors, in particular the fraction transformed to methyl-Hg (MeHg) and the bioconcentration factor in seafood. The most troubling and currently best understood health impacts of Hg are neurotoxic (see Axelrad *et al*., 2007 and references therein), and we evaluate them using as proxy recent DRF data for IQ decrement due to MeHg ingestion. We neglect other forms of Hg because they are far less toxic, despite relatively high doses, for example, from dental amalgam (see, e.g., Bellinger *et al*., 2006).

The main assumptions are the following.

(1) For an assessment of global impacts of Hg emissions to air the dependence on emission site can be neglected because the residence time of metallic Hg in the atmosphere is sufficiently large to imply a fairly uniform hemispherical distribution of the ingestion dose. Even though the actual distribution of ambient total Hg is not very uniform (because of the contribution of RGM and $Hg_p$), the ingestion dose of MeHg becomes far more uniform because of the wide international trading of fish.
(2) A comprehensive transfer factor, defined as incremental average dose due to an incremental emission, can be estimated as ratio of average dose and total (natural + anthropogenic) global emission at steady-state conditions.
(3) The worldwide average dose from fish and seafood is about 2.4 $\mu$g/day-person of MeHg as reported by UNEP (2002).
(4) The worldwide emission rate is about 6,000 tons/year, as estimated by the recent UN study (UNEP, 2002); about one-third of that is from natural sources.
(5) The DRF for IQ points lost can be approximated by a straight line with two possibilities for a threshold, either no threshold or a threshold corresponding to the reference dose RfD of EPA (2001); as slope of the DRF we take the value found by the integrative analysis of Axelrad *et al*. (2007) and we convert it to an ingestion dose using conversion factors from the literature.
(6) The neurotoxic impact on a population can be estimated by applying the DRF of Axelrad *et al*. to women of child-bearing age.
(7) The average ingestion dose of women of child-bearing age is equal to the average dose reported by UNEP (2002); for the threshold case we assume a distribution of worldwide doses similar to that in the United States (NCHS, 2005).
(8) The social cost of an IQ point lost in each country is calculated by modifying the value in the United States (for which we take $\$_{2005}$18,000/IQ point in proportion to the GDP/capita), adjusted for purchase power parity (PPP).

We believe that in view of the evidence these assumptions are the most plausible choice for calculating the global average Hg damage cost and that no better calculation is possible with the limited data currently available. An examination of the uncertainties suggests that the result estimates the global damage cost within a factor of about 4 (around the geometric mean).

## 2. THE COMPREHENSIVE TRANSFER FACTOR

Of course, the pathways of Hg in the environment are extremely complex and at the present state of knowledge a detailed modeling of global impacts would be difficult and very uncertain. The difficulties begin with the speciation during emission. The dominant species emitted from coal-fired power plants are Hg(0), RGM, and $Hg_p$, and typical percentages in the United States are around 58% for Hg(0), 40% for

RGM, and 2% for $Hg_p$, although highly variable from one plant to another (RGM from utility boilers in the United States can vary from 10% to 90%) (Sullivan *et al*., 2003). Pirrone *et al*. (2001) report Hg speciation estimates for all anthropogenic sources as 64% Hg(0), 28.5% RGM, and 7.5% $Hg_p$. Natural emissions are mostly elemental mercury, according to EMEP [http://www.msceast.org].

RGM and $Hg_p$ are water-soluble and deposit by wet and dry deposition (Rea *et al*., 2000, 2001; Vette *et al*., 2002; Landis *et al*., 2002). Several recent articles (EPA, 1997; Rossler, 2002) indicate that in the United States about 60% of the Hg deposition is from local sources, the global reservoir contributing the rest. Hg(0) becomes part of the global mercury cycle. The effective lifetime of Hg(0) in the atmosphere is in the range of one to two years, long enough to cause mixing in the entire hemisphere (Lamborg *et al*., 2002). Note that this life time is much longer than what would be implied by a simple consideration of the wet and dry deposition velocities because much of the deposited Hg(0) evaporates again for another deposition-volatization cycle. If natural emissions are one-third of the total with about 95% Hg(0), while anthropogenic emissions are two-thirds with about 64% Hg(0), it follows that about three-quarters of the total emitted mercury disperses globally—hence the importance of a global assessment of damage costs.

The dose from fish and seafood involves the transformation of Hg into MeHg by aquatic microorganisms. Even though only a small percentage of the Hg is thus transformed, MeHg is much more toxic than the other forms. Furthermore, the bioconcentration factor of MeHg is very large, leading to relatively high concentrations in seafood, especially among predatory fish. For these reasons the ingestion of fish and seafood is by far the most important pathway for human health impacts.

Trying to model the detailed pathways at the global scale would be most problematic because of the almost universal lack of data; the uncertainties would be enormous. As an alternative to detailed pathway modeling we use a comprehensive transfer factor $T_{av}$, defined as the ratio of the global average ingestion dose $D_{av}$ per person and the global emission rate $E$,

$$T_{av} = D_{av}/E, \tag{1}$$

it has units of $(\mu g_{MeHg}/year)/(kg_{Hg}/year)$. (The key symbols of our article and their definitions are summarized in Table I.) Estimates of global emissions and global ingestion dose have been reviewed in a major study by UNEP (2002). Based on this study we take a global emission rate of $E = 6{,}000$ tons/year (uncertainty range 3,000–9,000 tons/year). A large part of that, about one-third, is due to natural emissions; the anthropogenic part has been increasing steadily since the Industrial Revolution (Lamborg *et al*., 2002). One of the difficulties of estimating global emissions lies in the fact that a significant fraction of the deposited Hg is later, possibly much later, reemitted to the atmosphere.

For the global average ingestion dose of MeHg we take $D_{av} = 2.4$ $\mu$g/day per person, based on a report of the WHO (World Health Organization) as cited in Table 4.3 of UNEP (2002). For comparison we note that in the United States the ATSDR dose estimate is 50 ng/$kg_{bw}$/day, implying 3.0 $\mu$g/day for a body weight of 70 $kg_{bw}$ [http://www.atsdr.cdc.gov/toxprofiles/phs46.html], quite similar in view of the uncertainties. Data for the United Kingdom (2002) indicate intakes of about 3 $\mu$g/day for adults. With $D_{av} = 2.4$ $\mu$g/day per person we obtain a transfer factor of

$$T_{av} = 4.0\text{E} - 07(\mu g/day)/(kg/year). \tag{2}$$

**Table I.** Definition of the Most Important Symbols

| Symbol | Definition and Equation Where it is Defined |
|---|---|
| $T_{av}$ | Transfer factor $T_{av}$ = ratio of global average ingestion dose $D_{av}$ per person and global emission rate $E$; Equation (1) |
| $s_{DR}$ | Slope of dose-response function; Equation (5) |
| $I_i$ | Lifetime impact for individual $i$ exposed to dose $D_i$; Equation (6) |
| $\Delta D_{av}(D_{th})$ | Dose above threshold $D_{th}$ [$\mu$g/day per person], averaged over entire population; Equation (8) |
| $D_{av}(D_{th})$ | Total dose of the individuals who are above $D_{th}$, averaged over entire population; Equation (11) |
| $C$ | Marginal damage cost in €/kg emitted; Equation (17) |
| $f_{world}$ | Fraction of world population $p_{world}$ that is affected per year by Hg, weighted by GDP/capita; Equation (21) |

The product of $T_{av}$ and the world population, 6.4 billion (times the ratio of molecular weights Hg/MeHg) is the intake fraction, that is, the fraction of the emitted Hg that passes through a human body as MeHg on its way to the ultimate environmental sink, mostly ocean sediment (Lamborg *et al*., 2002). Our result for intake fraction, 0.9E-03, is in the range of values found for other toxic metals (Spadaro & Rabl, 2004). Despite the low proportion of Hg that is transformed to MeHg we find such an intake fraction plausible because of the large bioconcentration factor in fish and seafood.

## 3. DRF

In this article we are only concerned with impacts at low doses. Among the various possible health impacts of Hg at low doses, the neurotoxic impacts on fetus and infants have been investigated most thoroughly and they appear to be the most worrisome and the least uncertain. The adult brain is far less sensitive to Hg and no significant association with neurotoxic impacts on adults has been found at low doses (Weil *et al*., 2005). Evidence is also accumulating for another impact at low doses, coronary heart disease, see, for example, the review by Virtanen *et al*. (2007), although the case seems less clear and we do not consider it, noting only that Rice and Hammitt (2005) find that it can make a very large contribution to the damage cost. Measuring impacts at the low exposures of typical populations is in fact a great challenge. For instance, the study of neurotoxic effects of PCBs and Hg in the United States by Stewart *et al*. (2003) has not been very conclusive for Hg; however, the focus was on PCBs, Hg being examined only as an effect modifier. The most important studies on neurotoxic impacts have followed cohorts of children among three populations (New Zealand, the Seychelles, and the Faroe Islands) whose diet contains a particularly large portion of seafood; here significant associations between exposure and neurotoxic impacts have been observed.

Based on the findings in New Zealand, the Seychelles, and the Faroe Islands, Trasande *et al*. (2005) have estimated the social cost of the IQ decrement that can be attributed to Hg ingestion in the United States. In view of the large uncertainties, they consider several possible forms of the DRF, both linear and logarithmic, with a no-effect threshold of 8.2 $\mu$g/L$_{\text{cord}}$ in the cord blood of the newborn infant. In a recent update (Trasande *et al*., 2006) they revise their DRFs, the new ones being much lower than in their 2005 article.

In this article we take the DRF of Axelrad *et al*. (2007) because it is derived by an integrative analysis of the New Zealand, the Seychelles, and the Faroe Islands studies, with a method that uses the maximum of information from all three studies. Thus it holds promise to be much more reliable than any single study. They assume a linear DRF and their central estimate of the slope is 0.18 IQ points per ppm increase of maternal hair mercury (95% confidence interval 0.009 to 0.378). Sensitivity analyses produce estimates in the range from 0.13 to 0.25 IQ points per ppm, not very different.

To apply this DRF we need to relate the Hg concentration in maternal hair to the MeHg intake dose $D$ by ingestion. Axelrad *et al*. indicate a concentration ratio hair/cord blood of 200. The concentration in cord blood is higher than in maternal blood, but there is considerable uncertainty about the ratio cord blood concentration/maternal blood concentration. In this article we assume a ratio of 1.65, the mean of the meta-analysis by Stern and Smith (2003). These authors find that the distribution of values for this ratio is lognormal with median (=geometric mean) of 1.45, which implies a geometric *SD* $\sigma_g = 1.66$ since one has (see, e.g., Spadaro & Rabl, 2008)

$$\sigma_g = \text{Exp}[(2\text{Ln}(\text{mean/median}))^{0.5}] \qquad (3)$$

for the lognormal distribution.

To relate blood concentration to dietary intake, we note that according to UNEP (2002) the ratio of the steady-state blood concentration $c$[in $\mu$g/L$_{\text{mat}}$] and the average dietary MeHg intake $D$[in $\mu$g/day] is in the range $c/D = 0.3$ to 0.8. Here, we assume the model of Stern (2005) for the relation between intake dose $D$ of MeHg and concentration $c$, namely,

$$c = 0.61 * D. \qquad (4)$$

The coefficient 0.61 $\mu$g/L$_{\text{mat}}$/($\mu$g/day) accounts for blood volume, absorption, and elimination rate.

Multiplying these factors we obtain a DRF slope of

$$\begin{aligned} s_{\text{DR}} &= 0.18\,\text{IQpoints/ppm}_{\text{hair}} * 0.2\text{ppm}_{\text{hair}}/(\mu\text{g/L}_{\text{cord}}) \\ &\quad * 1.65\,\mu\text{g/L}_{\text{cord}}/(\mu\text{g/L}_{\text{mat}}) \\ &\quad * 0.61\mu\text{g/L}_{\text{mat}}/(\mu\text{g/day}) \\ &= 0.036\,\text{IQpoints}/(\mu\text{g/day}). \end{aligned} \qquad (5)$$

As for a possible threshold, EPA (2001) noted that "*no evidence of a threshold arose for methylmercury-related neurotoxicity within the range of exposures*" Axelrad *et al*. (2007) also argue for linear DRF without threshold. We find the possibility of a straight

line without threshold not only plausible but probable. Hg is a neurotoxicant that damages the developing brain and reduces the IQ, just like Pb. Also, like Pb it is a substance that has only harmful effects, by contrast to other metals (for instance, Cr and Se) that are toxic at high doses but of which the organism needs a certain minimum to survive. Furthermore, whereas in the past the DRF for IQ decrement due to Pb was believed to have a threshold, more recent studies have found that at the lowest doses the DRF for Pb is at least as high as the extrapolation of the high dose points, and quite possibly even higher (Lanphear *et al.*, 2005). Nonetheless, we also evaluate the impacts if there is a no-effect threshold dose $D_{th}$, assuming the same slope $s_{DR}$. As threshold we take the oral reference dose RfD of EPA (2001), noting, however, that it is not a no-effect level but intended as a guideline for protecting the population with a sufficient margin of safety.

## 4. CALCULATION OF LIFETIME IMPACT

To apply the DRF, it might appear necessary to consider the time window during which the brain is affected by Hg. The sensitivity of the brain to Hg is greatest during the early development of the body, but the precise time distribution of the damage is not known. Whereas the damage is incurred only during early development, it is assumed permanent and measurable at the ages reported in the epidemiological studies. Since the DRF of Axelrad *et al.* is based on correlations between the maternal hair concentration and the IQ of the children, it implicitly includes also the effect of diet during early infancy before the IQ of the children was measured. One can assume that the diet of the infants is strongly correlated with that of the mothers. Thus the DRF slope of Equation (5) describes the total lifetime impact on children whose mothers are exposed to a specified steady-state ingestion dose, and the detailed time distribution of the sensitivity to Hg does not matter for the calculation of impacts.

If a particular mother $i$ has had an ingestion dose $D_i$, the lifetime impact $I_i$ on the offspring is an IQ loss of

$$I_i = \begin{cases} 0 & \text{if} \quad D_i < D_{th} \\ s_{DR}(D_i - D_{th}) & \text{if} \quad D_i \geq D_{th} \end{cases}, \tag{6}$$

where $D_{th}$ is the threshold dose. Since everybody alive has or had a mother, the total impact in a population of $p$ individuals is obtained by summing Equation (6) over the doses of the mothers:

$$I_{\text{tot}} = s_{DR} \sum_{i=p_{th}}^{p} (D_i - D_{th}), \tag{7}$$

where $p_{th}$ is the number of individuals with maternal dose below $D_{th}$ and the sum covers only the individuals with maternal dose above $D_{th}$. We find it convenient to express everything as impact per person, averaged over the entire population (men, women, all ages) rather than just the sensitive individuals. Using the notation $\Delta D_{av}(D_{th})$ for the positive differences $D_i - D_{th}$, averaged over the entire population,

$$\Delta D_{av}(D_{th}) = \frac{1}{p} \sum_{i=p_{th}}^{p} (D_i - D_{th}), \tag{8}$$

one can write the average lifetime IQ loss per person $I_{av} = I_{\text{tot}}/p$ as

$$I_{av} = s_{DR} \Delta D_{av}(D_{th}). \tag{9}$$

To calculate this quantity one needs data for the distribution of doses for the world population. For most countries that is unfortunately not available. The best data are the ones of NCHS (2005) (see also Mahaffey, 2005) for the fraction of women of child-bearing age in the United States that has various levels of MeHg concentration (as $\mu$g per L of maternal blood). This distribution is to a good approximation lognormal with geometric mean $\mu_g = 0.7$ $\mu$g/L and geometric $SD$ $\sigma_g = 3.5$ (a distribution that is fairly similar to data of the United Kingdom, 2002). Using this lognormal distribution one can readily calculate the population averaged dose above $D_{th}$ of Equation (8). Since the distribution is not weighted by the number of children, our calculations assume implicitly that the number of children per mother is independent of her dose.

The resulting mean dose in the United States is about 2.5 $\mu$g/day and very close to the UNEP (2002) estimate of the average dose of the entire world, 2.4 $\mu$g/day, the dose that we use as basis for our damage cost estimates. Lacking data for the dose distributions in other countries, we use the distribution of NCHS (2005) for the world and scale it down by a factor 2.4/2.5, assuming that the UNEP data also apply to women of child-bearing age.

Fig. 1 shows the resulting relation between threshold dose $D_{th}$ and $\Delta D_{av}(D_{th})$ of Equation (8). The fraction $f(D_{th})$ of the population that is above threshold is indicated by the dashed line and the right-hand scale. The threshold RfD of EPA is 0.1 $\mu$g/day/$\text{kg}_{\text{bodyweight}}$, implying $D_{th} = 6.7$ $\mu$g/day for an average weight

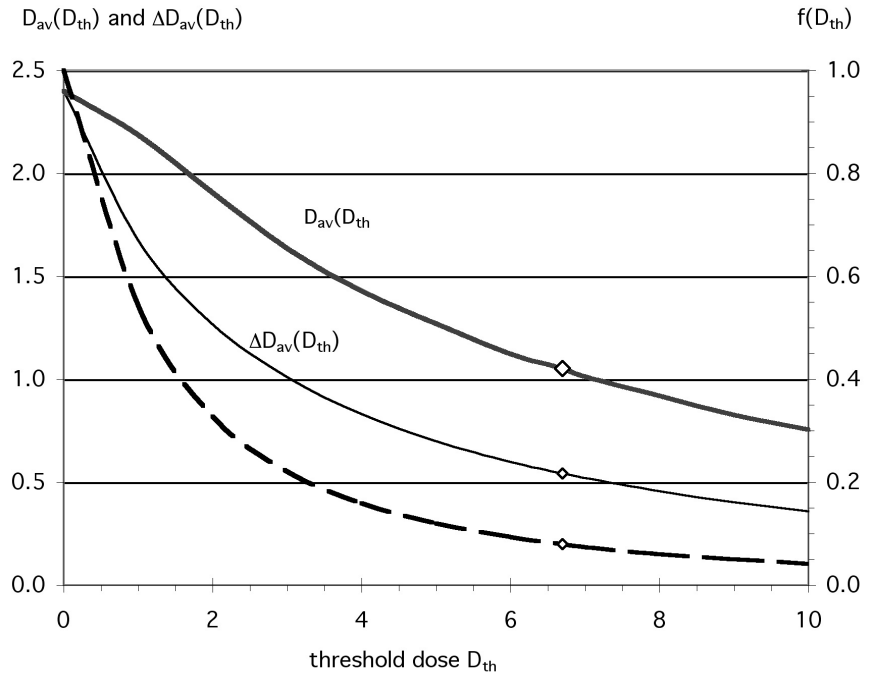


**Fig. 1.** Thin solid line shows relation between threshold dose $D_{th}$ and dose $\Delta D_{av}(D_{th})$ above $D_{th}$ per person averaged over entire population, Equation (8); this is needed for the total lifetime impact. Thick gray line shows the dose $D_{av}(D_{th})$ of Equation (11), needed for the marginal damage cost. Dashed line and right-hand scale shows the fraction $f(D_{th})$ of the population that is above threshold. Diamonds mark the EPA reference dose of 6.7 $\mu$g/day. Based on the distribution of NCHS (2005) and scaled to correspond to the UNEP dose of 2.4 $\mu$g/day.

of 67 $\text{kg}_{\text{body weight}}$; this is indicated by the diamonds. The graph also shows the quantity $D_{av}(D_{th})$ of Equation (11) in the next section, which will be needed for the marginal damage cost. Multiplying the dose $\Delta D_{av}(D_{th})$ by $s_{DR}$ of Equation (5) one finds the corresponding average IQ loss is 0.020 IQ points if $D_{th} = 6.7$ $\mu$g/day. For zero threshold the loss is 0.087 IQ points.

## 5. MARGINAL IMPACTS AND COSTS PER KILOGRAM OF EMITTED HG

By contrast to the lifetime impact, the calculation of impact and cost of an additional kg of emitted Hg (also called marginal or incremental cost) requires consideration of the rate at which new individuals are affected, hence the birth rate $b$ enters into the calculation. For the allocation of an additional impact $\delta I$ to an additional emission $\delta Q$ it may be helpful to assume that emission and impact are simultaneous and occur during a certain time interval for which one can arbitrarily take $\Delta t = 1$ year; in reality the impact occurs later, of course, but the delay does not change the magnitude of the impact. Only the $\Delta p = pb\Delta t$ individuals born during this time interval are affected, and the incremental impact on the total population is

$$\delta I_{\text{tot}} = pb\Delta t s_{DR}\delta\Delta D_{av}(D_{th}), \tag{10}$$

where $\delta\Delta D_{av}(D_{th})$ is the increment of $\Delta D_{av}(D_{th})$ of Equation (8) due to $\delta Q$. To find how an increase of the emission rate $\delta E = \delta Q/\Delta t$ changes the dose $\Delta D_{av}(D_{th})$, note that in the limit of small changes the number $p_{th}$ of individuals above threshold does not change (a change in $p_{th}$ would yield a term of order $\delta E^2$ in the incremental impact and is, by definition, neglected in the calculation of marginal costs). Whereas the doses $D_i$ are proportional to the emission rate $E$, the quantity $\Delta D_{av}(D_{th})$ is not because it contains only the contributions above threshold. To find how $\Delta D_{av}(D_{th})$ changes, let us define the population-average of the total dose of the individuals who are above $D_{th}$ as

$$D_{av}(D_{th}) = \frac{1}{p}\sum_{i=p_{th}}^{p} D_i, \tag{11}$$

and rewrite Equation (8) in the form

$$\Delta D_{av}(D_{th}) = D_{av}(D_{th}) - f(D_{th})D_{th} \text{ with } f(D_{th}) = (1 - p_{th}/p). \tag{12}$$

To relate it to the emission rate $E$, we multiply $D_{av}(D_{th})$ by a factor $E\,T_{av}/D_{av,}$ which is equal to unity by its definition in Equation (1):

$$\Delta D_{av}(D_{th}) = ET_{av}\,D_{av}(D_{th})D_{av} - f(D_{th})\,D_{th}. \tag{13}$$

Since a small change in emission rate changes neither the transfer factor $T_{av}$ nor the shape of the probability distribution of doses, the quantity $T_{av}\, D_{av}(D_{th})/D_{av}$ is independent of $E$. Hence, the increment $\delta\Delta D_{av}(D_{th})$ equals

$$\delta\Delta D_{av}(D_{th}) = \delta E T_{av} D_{av}(D_{th})/D_{av.} \qquad (14)$$

Inserting this into Equation (10) and replacing the incremental emission rate by $\delta E = \delta Q/\Delta t$, we obtain

$$\delta I_{\text{tot}} = pb\Delta t\, s_{DR}(\delta Q/\Delta t)\, T_{av}\, D_{av}(D_{th})/D_{av}. \qquad (15)$$

The time interval $\Delta t$ is indeed arbitrary because it cancels out. The ratio of $\delta I_{\text{tot}}$ and incremental emission $\delta Q$ is the incremental impact per emitted Hg:

$$\text{incremental impact per emitted Hg} = s_{DR}[T_{av}\, D_{av}(D_{th})/D_{av}] \sum_k p_k b_k, \qquad (16)$$

where $D_{av}(D_{th})$ is given by Equation (11), and we have replaced $pb$ by a sum over countries $k$ to allow for the fact that birth rates in different countries are different. Multiplying by the cost $c_k$ per IQ point in country $k$, we obtain the marginal damage cost $C$ in €/kg emitted,

$$C = s_{DR}[T_{av}\, D_{av}(D_{th})/D_{av}] \sum_k p_k\, b_k\, c_k\, f_{\text{disc}}. \qquad (17)$$

We have included a factor $f_{\text{disc}}$ to account for the time lag between a change in emissions and the impact

$$f_{\text{disc}} = (1 + r_{\text{disc}})^{-N}, \qquad (18)$$

where $r_{\text{disc}}$ is the discount rate and $N$ the time lag; following Griffiths *et al.* (2007), we take $r_{\text{disc}} = 3\%$ and $N = 15$ year for the central value, resulting in $f_{\text{disc}} = 0.64$. Note that we apply this discount factor only to the marginal cost, not to the lifetime cost, since the latter corresponds to current exposure.

For the cost associated with the loss of an IQ point there is a range of estimates in the United States, mostly based on lost earnings or remedial education. In particular we cite

- Muir and Zegarac (2001) $\$_{1999}$14,700/IQ point,
- Grosse *et al.* (2002) $\$_{2000}$14,500/IQ point,
- Rice and Hammitt (2005) $\$_{2000}$16,500/IQ point,
- Trasande *et al.* (2005) $\$_{2000}$22,200/IQ point, and
- Griffiths *et al.* (2007) $\$_{2000}$11,245/IQ point.

Adjusting these figures to \$2005 by means of the CPI we obtain a mean of about $\$_{2005}$18,000/IQ point. To apply this cost in different countries we modify the cost in proportion to the $\text{GDP}_{PPP}$/capita, the per capita GDP adjusted for purchase power parity [http://www.cia.gov/cia/publications/factbook/]. So the cost of an IQ point in country $k$ is

$$c_k = c_{\text{USA}} \frac{(GDP_{PPP}/capita)_k}{(GDP_{PPP}/capita)_{\text{USA}}}. \qquad (19)$$

The worldwide average cost is

$$c_{av} = \sum_k c_k \frac{p_k}{p_{\text{world}}} = \$3,890/\text{IQpoint}. \qquad (20)$$

Let us define a fraction $f_{\text{world}}$ of the world's population $p_{\text{world}}$ that is affected per year by Hg, weighted by GDP/capita, as

$$f_{\text{world}} = \sum_k b_k \frac{p_k}{p_{\text{world}}} \frac{(GDP_{PPP}/capita)_k}{(GDP_{PPP}/capita)_{\text{USA}}}, \qquad (21)$$

its numerical value is 0.00315. Thus we can write $C$ in the form

$$C = s_{DR}[T_{av}\, D_{av}(D_{th})/D_{av}] \times f_{\text{world}}\, p_{\text{world}}\, c_{\text{USA}}\, f_{\text{disc}} \text{ in \$/kg emitted}. \qquad (22)$$

As shown in Table II, the marginal damage cost $C$ per kg of emitted Hg is about \$1,500/kg for a threshold dose of 6.7 mg/day and \$3,400/kg for zero threshold. It is also interesting to look at the total average worldwide IQ loss per person due to current emissions (0.02 IQ points/person with and 0.087 IQ point/person without threshold) and the corresponding costs (\$78/person with and \$344/person without threshold). We note

**Table II.** Our Estimates of the Total Worldwide Lifetime Impact and Cost (Equations (8) and (20)), and of the Marginal Damage Cost $C$ (Equation (22)) of Hg Emissions, for Two Assumptions about the Threshold Dose $D_{th}$

| | | Lifetime Impact and Cost | | | Marginal Damage Cost | |
|---|---|---|---|---|---|---|
| $D_{th}$ ($\mu$g/day) | $f(D_{th})$ $= 1 - p_{th}/p$ | $\Delta D_{av}(D_{th})$ ($\mu$g/day) | Lifetime Impact (IQ points/person) | Lifetime Cost (\$/person) | $D_{av}(D_{th})$ ($\mu$g/day) | $C$ (\$/kg) |
| 6.7 | 0.08 | 0.54 | 0.020 | 78 | 1.05 | 1,500 |
| 0 | 1 | 2.40 | 0.087 | 344 | 2.40 | 3,400 |

*Note*: $f(D_{th})$ is the fraction of the total population that is above threshold.

that the marginal damage cost for the case with threshold is appropriate only for small changes: for large changes the fraction of the population above threshold will also change.

## 6. ASSESSMENT OF UNCERTAINTIES

Uncertainty assessments for this kind of problem are usually done via a Monte Carlo analysis; the result is numerical and not very transparent. Instead, we apply the method of Rabl and Spadaro (1999) and Spadaro and Rabl (2008), which is appropriate in this case because the impact and cost are essentially a product of factors (the summation over countries introduces errors that are entirely negligible). This approach has the advantage of being simple and transparent, and its accuracy is sufficient because in any case an assessment of uncertainties involves subjective judgments and is very approximate. The uncertainty of each factor is characterized in terms of its geometric *SD*. Then the geometric *SD* $\sigma_g$ of the product can readily be calculated from the geometric $SD\sigma_{gj}$ of the factors by the equation

$$[\ln(\sigma_g)]^2 = [\ln(\sigma_{g1})]^2 + [\ln(\sigma_{g2})]^2 + \cdots + [\ln(\sigma_{gn})]^2, \tag{23}$$

assuming that the distributions are statistically independent.

The central limit theorem implies that the distribution of the product is approximately lognormal in the limit where the number of factors becomes very large. In practice the distribution is close to lognormal even for a small number of factors, unless the distribution(s) with the largest width is (are) far from lognormal. For a lognormal distribution the geometric *SD* indicates multiplicative confidence intervals, analogous to the additive confidence intervals of the Gaussian distribution. One can show that for the lognormal distribution the geometric mean $\mu_g$ is equal to the median and the ratio of mean/geometric mean is given by

$$\mu/\mu_g = \exp\left(0.5 \ln^2(\sigma_g)\right). \tag{24}$$

If a quantity with a lognormal distribution has been found to have a geometric mean $\mu_g$ and a geometric *SD* $\sigma_g$, the probability is approximately 68% for the true value to be in the interval $[\mu_g/\sigma_g, \mu_g\,\sigma_g]$ and 95% for it to be in the interval $[\mu_g/\sigma_g^2, \mu_g\,\sigma_g^2]$. Our estimates of the uncertainties are listed in Table III for the case with threshold; without threshold the uncertainty is lower. We have taken the geometric $SD\sigma_{gi}$ of the individual factors as 4th root of the ratio's high value/low value, roughly interpreting the low to high range as width of the 95% confidence interval. For the estimation of the average dose above threshold we have considered a wide range of possible dose distributions and their effect on the ratio $D_{av}(D_{th})/D_{av}$; we believe that this accounts for the uncertainty. The uncertainty of $f_{\text{world}}$ includes the GDP/capita adjustment of the cost of an IQ point in different countries. The resulting $\sigma_g$ is 4.2.

**Table III.** Our Estimates of the Uncertainties of the Marginal Damage Cost for the Case with Threshold

| Factor | Unit | Mean | Low | High | $\sigma_{gi}$ | $\ln^2(\sigma_{gi})$ |
|---|---|---|---|---|---|---|
| Emission rate, $E$ | $t$/year | 6000 | 3000 | 9000 | 1.32 | *0.08* |
| Average dose, $D_{av}$ | $\mu$g/day | 2.40 | 0.60 | 4.00 | 1.61 | *0.22* |
| Dose above threshold, $D_{av}(D_{th})/D_{av}$ | – | 0.44 | 0.20 | 1.20 | 1.57 | *0.20* |
| $s_{DR}$ based on Hg in hair | IQ point/ppm | 0.18 | 0.009 | 0.38 | 2.55 | *0.87* |
| Hair conc/cord blood conc | ppm/($\mu$g/$L_{\text{cord}}$) | 0.20 | 0.10 | 0.30 | 1.32 | *0.08* |
| Ratio of cord to maternal blood | ($\mu$g/$L_{\text{cord}}$)/($\mu$g/$L_{\text{mat}}$) | 1.65 | 0.41 | 3.14 | 1.66 | *0.26* |
| Ratio of maternal blood to dose intake | ($\mu$g/$L_{\text{mat}}$)/($\mu$g/day) | 0.61 | 0.40 | 1.00 | 1.26 | *0.05* |
| Cost of IQ point in USA | \$/IQ point | 18,000 | 10,000 | 25,000 | 1.26 | *0.05* |
| $f_{\text{world}}$ [Equation (21)] | 1/year | 0.00315 | 0.0014 | 0.0071 | 1.50 | *0.16* |
| Discount factor, $f_{\text{disc}}$ | – | 0.64 | 0.30 | 0.82 | 1.29 | *0.07* |
| World population, $p_{\text{world}}$ | – | 6.43*E*+09 | | | 1.00 | *0.00* |
| Results for cost $C$ | \$/kg | 1,487 | | | $\Sigma_i \ln^2(\sigma_{gi}) =$ | *2.04* |
| | | | 68% CI | | | |
| | Median | Mean | Low | High | $\sigma_g$ | |
| Results for cost $C$ [\$/kg] | 531 | 1487 | 126 | 2230 | 4.2 | |

*Note*: The geometric *SD* $\sigma_{gi}$ of the individual factors are the 4th root of the ratio's high/low. Some numbers for the results have been rounded in the text.

The lower part of the table lists the 68% confidence interval $[\mu_g/\sigma_g, \mu_g \sigma_g]$ of the result. To place the confidence intervals, one has to note the difference between mean and median. We have calculated the mean as product of the means of the factors. Since the distribution of the product is fairly close to lognormal, the ratio median/mean is 0.36 according to Equation (24) if $\sigma_g = 4.2$, and for the 68% confidence interval the ratio's lower bound/mean and upper bound/mean are 0.06 and 1.5. The placement of the confidence interval is very asymmetric because $\sigma_g$ is so large.

Of course, estimating component uncertainties necessarily involves a certain amount of subjective judgment. Our choice of the $\sigma_{gi}$ is somewhat arbitrary since the probability distributions of most of the parameters may be quite different from lognormal and our high and low values may not be the correct 95% confidence intervals, but even with different and equally plausible choices the $\sigma_g$ of the result would not be very different as the reader can readily verify. Note that because of the quadratic combination of the individual terms (see last column of the table), terms with relatively small uncertainty make negligible contributions to the total. In view of the great uncertainty of the estimation of uncertainties we round the numbers to summarize by saying that $\sigma_g$ is about 4 and the 68% confidence interval extends from about 0.1 to 1.5 times the mean.

## 7. CONCLUSIONS

Using a comprehensive transfer factor, defined as ratio of global Hg emissions and global average ingestion dose of MeHg, we have estimated the global average impact and cost. This includes local and regional impacts, but only as global averages. For a specific source and site the impact can be quite different. The ratio of local + regional/total impact can also be very different for different sources because much of the local and regional impact is due to RGM and $Hg_p$ whereas metallic Hg(0) is dispersed globally.

Some people might argue that regional and local variability could invalidate our global approach. As far as environmental pathways are concerned, such variability arises only from the $Hg_p$ and RGM component of the emissions, since Hg(0) disperses globally. Our model would be exact for Hg(0) emissions if we had exact data for the Hg(0) component of global emissions and for its contribution to the global average dose. Instead, our transfer factor includes the other components and thus our model combines global and regional impacts, the latter as global average. The other source of variability arises from the distributions of ingestion doses in different parts of the world. We believe that our uncertainty analysis of Section 6 adequately accounts for the possible effects of such variability by including a wide range of possible doses and dose distributions, and that therefore our results are valid within the uncertainty bounds we have estimated. Another possible criticism concerns our use of a steady state analysis whereas in reality emissions and doses have been changing. Here, too, our uncertainty analysis covers the ground because the range of emissions and doses is wide enough to account for changes during the past one or two decades (i.e., the time constant for the environmental pathways).

In terms of absolute magnitude the impacts and costs are small: 0.02 IQ points/person with and 0.087 IQ points/person without threshold, and \$78/person with and \$344/person without threshold; also one should note that about two-thirds of the current total emissions are anthropogenic. However, because of the small quantity of Hg that is involved, the marginal damage cost is high compared to other pollutants that have been evaluated, for example, by the ExternE project series of the EC [www.externe.info]; they are approximately \$1,500/kg if the threshold dose is 6.7 $\mu$g/day and about \$3,400/kg without threshold.

An examination of the uncertainties leads us to a geometric *SD* of about 4, in other words, the damage cost could be a factor 4 smaller or larger than the median estimate. However, the uncertainty about a possible threshold is difficult to capture because of the lack of information. As an indication one can consider the range of results between zero threshold and the RfD threshold of EPA, but the true threshold could be even higher: the RfD is a guideline for protecting public health and derived by including a large margin of safety (see also the discussion of thresholds by Lipfert *et al*. (1996)). Thus the impact and costs might even be an order of magnitude smaller than our estimate.

It is interesting to compare our marginal damage cost *C* with a recent study by Rice and Hammitt (2005), who estimate the benefit of the new regulation for Hg emissions by power plants in the United States. Of course, their numbers are not very comparable with ours because they consider only impacts within the United States, whereas our estimate is a simple global average. Due to local and regional variations in Hg speciation, dispersion, population, dietary habits, etc., a regional calculation such as that of Rice and Hammitt can find very different results. In particular, the speciation for the power plants considered

by Rice and Hammitt has a higher fraction of RGM and $Hg_p$ than the global emissions we have assumed; thus they find a higher fraction of Hg deposited in the United States where the cost of an IQ point is higher than the global average. However, the threshold assumptions are the same.

To estimate what our model implies with the scope and assumptions of Rice and Hammitt, we multiply by the ratio of the United States cost and the global cost in our global calculation; that ratio is 0.207, relatively high because of the high cost of an IQ point in the United States compared to the rest of the world (in other words, the result of Rice and Hammitt would be a factor 1/0.207 larger if they had included global impacts and if the damage of U.S. power plant emissions were equal to our very simple global model). We also adjust for different assumptions about cost of IQ point ($16,500/IQ point for Rice and Hammitt vs. $18,000/IQ point for us, and DRF slope $s_{DR}$ (0.12 IQ point/($\mu$g/day) for Rice and Hammitt vs. 0.0362 IQ point/($\mu$g/day) for us).

Finally, about 60% of U.S. emissions are deposited on U.S. soil (EPA, 1997) because of the large fraction of RGM in the speciation of power plant emissions in the United States; to convert from the typical speciation that we assume to power plant emissions in the United States we, therefore, have to multiply by a factor of 3. Combining these adjustment factors we multiply our global cost $C$ (but without discount factor $f_{\text{disc}}$ for consistency with Rice and Hammitt) by $0.207 * 3.0 * (0.12/0.0362) * (16.5/18) = 1.89$ to obtain our estimate for the United States. The result, $4,380/kg with and $9,993/kg without threshold, agrees with the corresponding numbers of Rice and Hammitt, $4,300/kg and $11,200/kg, within 12%, far closer than one could expect in view of the radically different approach.

Abatement of Hg emissions is cost effective if the marginal abatement cost is smaller than the marginal damage cost. Abatement costs are highly variable, depending on technologies and specific conditions. For example, Jones *et al*. (2007) evaluate the cost of Hg removal by activated carbon injection for six power plants, with several variations, and find a range of about $8,400 to $365,000/$kg_{Hg}$; in most cases the abatement would exceed by far the benefits we have calculated. Of course, the benefits may turn out much larger if other impacts, in particular cardiovascular, are included (Rice & Hammitt, 2005; Virtanen *et al*., 2007).

But let us emphasize that for the formulation of new regulations there are other options besides reducing the emissions. In particular, a worldwide educational campaign to prevent pregnant woman and infants from eating fish and seafood with high Hg content may be far more cost effective in many regions and it is all the more relevant in view of the fact that a large part of the emissions is of natural origin. However, careful analysis of all relevant costs and benefits is required before formulating any such advisory because the large health benefits of seafood must not be overlooked (see Cohen *et al*., 2005; Jorgensen *et al*., 2007).

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