Risk Analysis, Vol. 24, No. 5, 2004

Pathway Analysis for Population-Total Health Impacts of Toxic Metal Emissions

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

This article describes a simple model for quantifying the health impacts of toxic metal emissions. In contrast to most traditional models it calculates the expectation value of the total damage (summed over the total population and over all time) for typical emission sites, rather than "worst-case" estimates for specific sites or episodes. Such a model is needed for the evaluation of many environmental policy measures, e.g., the optimal level of pollution taxes or emission limits. Based on the methodology that has been developed by USEPA for the assessment of multimedia pathways, the equations and parameters are assembled for the assessment of As, Cd, Cr, Hg, Ni, and Pb, and some typical results are presented (the dose from seafood is not included and for Hg the results are extremely uncertain); the model is freely available on the web. The structure of the model is very simple because, as we show, if the parameters can be approximated by time-independent constants (the case for the USEPA methodology), the total impacts can be calculated with steady-state models even though the environment is never in steady state. The collective ingestion dose is found to be roughly 2 orders of magnitude larger than the collective dose via inhalation. The uncertainties are large, easily an order of magnitude, the main uncertainties arising from the parameter values of the model, in particular the transfer factors. Using linearized dose-response functions, estimates are provided for cancers due to As, Cd, Cr, and Ni as well as IQ loss due to Pb emissions in Europe.

KEY WORDS: Arsenic; atmospheric dispersion; cadmium; chromium; collective dose; dose-response functions; ingestion dose; inhalation dose; intake fraction; IQ decrement; lead; mercury; nickel; thresholds; toxic metals; transfer factors; typical impacts; uncertainty

1. PATHWAYS AND FRAMEWORK

The goal of this article is to calculate expectation values of the total collective dose, damage, and cost per kg of emitted pollutant, for typical sites and conditions, in contrast to most traditional models that are intended to calculate values for a "worst case," a particular site or a particular population at risk, often by choosing upper limits rather than expectation values for the parameters. We consider the most toxic metals: As, Cd, Cr, Hg, Ni, and Pb; the model can readily be extended to other pollutants, including organics. Our model is based on the methodology developed for the assessment of multimedia pathways by EPA (1998). A similar model has been developed by IAEA (1994, 2001) for the assessment of radiological impacts, but its list of parameter values has many gaps. We rely mostly on the EPA model, supplemented in some cases by data of IAEA. Some of our equations are different from those of EPA or IAEA because we include integration over all time (in the case of very long time constants we also show the results for a time horizon of 100 years).

[1] Centre d'Energétique, Ecole des Mines, Paris, Cedex 06, France.
* Address correspondence to Ari Rabl, Centre d'Energétique, Ecole des Mines, 60 boul. St. Michel, F-75272, Paris, Cedex 06, France; ari.rabl@ensmp.fr.

0272-4332/04/0100-1121$22.00/1

The model is needed for the evaluation of many environmental policy measures, e.g., for determining the optimal level of pollution taxes or of limits for the emission of pollutants. The underlying philosophy is totally different from the traditional approach of environmental policy, which was founded on the belief that a pollutant has no effect below a certain threshold dose. If that is the case there is a natural policy goal (although not necessarily the appropriate one): reducing the emission of a pollutant below the level where the highest dose is below the threshold. Standards for ambient air quality were developed, for example, by the World Health Organization, and industry was forced to reduce emissions to reach these standards.

However, the situation is changing. For many pollutants, epidemiologists have not been able to find no-effect thresholds, in any case not at the level of an entire population; in particular for $PM_{2.5}$ and Pb the hypothesis of a linear no-threshold dose-response function (DRF) seems now the most plausible. Linearity is already generally accepted for substances that initiate cancers. Furthermore, even if there is a threshold, it is not its absolute level that matters, but whether it is above or below current background exposures. For substances with substantial background exposures, such as several of the metals considered here, linearity appears very plausible, as emphasized by Wilson and his co-workers (Crawford & Wilson, 1996). Furthermore, even if the DRF is very nonlinear, for the calculation of population-total impacts one can approximate the DRF by a straight line corresponding to the population average, as we show in Section 2.

A related consideration is the geographic range of the impacts. Pollutants emitted into the air are dispersed over hundreds to thousands of kilometers, as highlighted by Fig. 1. In the case of Hg the dispersion covers the entire hemisphere. Any individual who, because of local pollution, diet, or individual sensitivity, is already above a no-effect threshold will suffer an impact, even far from the source. Clearly, the traditional models, limited to populations close to the source, fail to account for such impacts, which can be quite significant when summed over the entire population. If there are individuals above the threshold in the impact region there is no safe threshold as far as population-total impacts are concerned.

In the absence of no-effect thresholds there is no natural criterion for deciding how much to spend for the reduction of environmental risks. Thus one has to deal with a new paradigm, and quantification

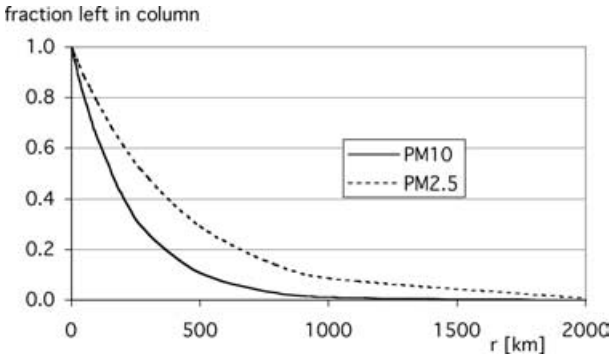


Fig. 1. Fraction of emitted pollutant that remains in the air at a distance r from the source. (Wind speed 5.5 m/s, mixing layer height 800 m, $v_{dep} = 0.0049$ m/s for PM_{10} and 0.0027 m/s for $PM_{2.5}$.)

of environmental benefits is required to make policy decisions consistent with collective preferences. One needs to consider expectation values, rather than upper bounds.

For the impacts quantified in this article, linearity is the most plausible assumption. Linearity of the DRF has important consequences, allowing tremendous simplifications of the analysis. It suffices to calculate the collective dose, regardless of how it is distributed among the population: the total time-integrated impact is proportional to the collective dose. The result is unaffected by people moving as long as the population densities do not change. Furthermore, as shown in Section 3, if the parameters can be approximated by time-independent constants (the case for the USEPA methodology), the collective dose depends only on the total emission, regardless of its distribution over time. Therefore, the total damage per kg of a pollutant can be calculated by a simple steady-state analysis, assuming a constant emission rate and steady-state conditions. The results for the total collective dose should be equal to a calculation with a level IV model in the terminology of the life cycle assessment community (Mackay, 2002), for example, the CalTOX model (McKone & Enoch, 2002); however, there are major differences in the detailed modeling of the pathways.

Even though the present article and our model are concerned only with collective doses and impacts, we emphasize that for policy decisions the impacts on vulnerable subpopulations should also be considered because the perception of an impact can be very nonlinear: a 10^{-2} risk to a subpopulation of 100 is not the same as a 10^{-8} risk to a total population of 100 million.

For the first step of an analysis of emissions to air we greatly simplify the calculation of inhalation doses

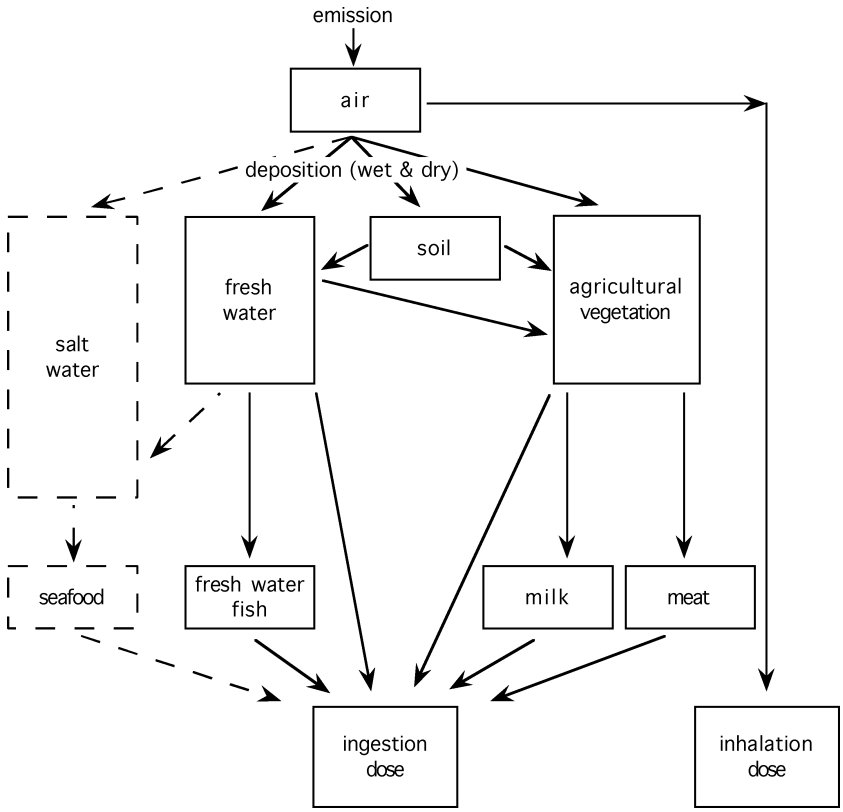


Fig. 2. Pathways taken into account for health impacts of air pollutants. Direct emissions to soil or water are a special case where the analysis begins at the respective "soil" and "water" boxes. In the present version seafood is not yet included.

by using the "uniform world model" (UWM) of Curtiss and Rabl (1996) and Spadaro (1999), which has been found to be a very useful summary of the results of detailed atmospheric dispersion calculations. This model is a simple product of a few factors. If all the parameters are geographically uniform, it is exact, as consequence of the conservation of mass. The UWM has the advantage of providing typical values that are relevant for many environmental policy decisions. The validity of the UWM for this purpose has been demonstrated in the case of air pollution by comparison with a large number of site-specific calculations in many countries of Europe as well as China and Brazil: for typical industrial sources with stack heights above about 50 m UWM agrees with an exact calculation within a factor of about 3. Correction factors can be applied for special sites or conditions. Of course, the UWM is no substitute for a detailed analysis if site-specific values are needed. But we have found that policy makers often do not bother to take site dependence into account, using a site-specific result as if it were typical, thus being precisely wrong rather than approximately right. The UWM is transparent: it is easy to recognize the important parameters, variables, and relationships, and it provides at least an order of magnitude estimate (note that in this field even the best calculation has an uncertainty almost as large (Rabl & Spadaro, 1999)). Here we extend the UWM to multimedia impacts. All equations and parameters are shown in the text or the appendices.

We account for the pathways in Fig. 2. We do not consider dermal contact because that pathway has been found to be entirely negligible for these metals (e.g. EPA, 1998; McKone & Enoch, 2002). Like the underlying EPA model we do not consider groundwater, assuming that, on average, inflow and outflow are equal. We do not yet have all the elements for calculating the dose due to ingestion of seafood, potentially large because of bioconcentration and because most fish come from the ocean rather than freshwater. One would need compartment models of all the oceans, coupled with data on fish production. Even if the concentration increment in the sea is very small, the collective dose from seafood could be significant if the removal processes (sedimentation) are slow and the analysis has no cutoff in time. The problem of long time constants also haunts the assessment of pathways that pass through soil. Neither EPA nor IAEA consider the impacts beyond the lifetime of the emitting installation, typically a few decades. Being concerned

with total impacts, we present two sets of results: one for the totality of the collective dose, and one for the collective dose incurred during the first 100 years. To allow valuation of the costs beyond the first generation with a lower intergenerational discount rate, we also indicate what fraction of the dose is incurred during the first 30 years after an emission.

We have implemented the model in an Excel file called RiskPoll_Multimedia (available at http://www.arirabl.com). We calculate doses from inhalation and from ingestion. Within the uncertainties our results are consistent with data reported by the World Health Organization (WHO, 1988–2001) and with results of CalTOX (McKone & Enoch, 2002). The total dose can be about 2 orders of magnitude larger than the inhalation dose. We also estimate the resulting impacts and damage costs for endpoints where the DRFs are sufficiently well established and linearity is most plausible: cancers due to As, Cd, Cr, and Ni, and IQ decrement due to Pb. The model is fully documented in this article, including all the equations and parameter values.

The novelty of this article is:

- To explain the need for the calculation of expectation values of collective doses and impacts, taking into account regional or global, not just local, dispersion;
- To point out the simplifications that can be made in the case of linear DRFs;
- To develop a simple model for the analysis of typical emission sources by adapting the equations and parameters of EPA;
- To apply this model to the calculation of incremental doses due to incremental emissions of As, Cd, Cr, Hg, Ni, and Pb in Europe, and to provide estimates of impacts and damage costs; and
- To point out gaps in current knowledge that render the results uncertain.

2. LINEARIZATION OF THE DOSE-RESPONSE FUNCTIONS

Our starting point is to approximate the DRF by a straight line in the dose range of interest, because the relevant dose range covers increments above typical background doses rather than above zero dose, an argument presented by Wilson and his co-workers (Crawford & Wilson, 1996). The possibility of a no-effect threshold below typical background doses is of no concern; only linearity in the dose range of interest is needed for our model. Linearity is commonly assumed for carcinogenic agents and justified on theoretical grounds for all agents that initiate the development of cancers. Linearity without threshold has also been found for some other cases where sufficient data are available to trace the DRF, e.g., for the IQ decrement due to Pb (Schwartz, 1994; Lanphear *et al.*, 2000) and for mortality due to particulate air pollution.

Of course, the precise form of a DRF is difficult to determine at low doses, and for most substances the available information indicates only a NOAEL (no observed adverse effects level) or a LOAEL (lowest observed adverse effects level). NOAEL or LOAEL data are not sufficient to calculate damages. Note that even a NOAEL does not preclude the possibility that more precise measurements in the future will detect a harmful effect at lower doses in a large population with a wide range of individual thresholds. For example, whereas it used to be conventional wisdom that particulate air pollution had a NOAEL, now the most plausible form is linear without threshold (see, e.g., Daniels *et al.*, 2000; Rabl, 2003).

If there is a no-effect threshold, the DRF probably departs from zero as a smooth function rather than with a sharp corner as a hockey stick. Furthermore, it is unlikely that such a threshold would happen to be precisely at current background doses, and even if that is the case, background exposures and individual sensitivities not being uniform, the population-averaged DRF is a smooth function as a result of averaging over zero and nonzero slopes. However, determining the appropriate average may be difficult in practice. The approximation of a DRF for incremental doses by a straight line is illustrated in Fig. 3. If the increment is large, the integration of the incremental impacts over the dose range is equivalent to averaging the slopes over this range.

Even if the slopes are different for different parts of the population, for the estimation of population-total impacts a calculation with a single average slope is adequate because differences average out when summing over the total population. This has in effect been demonstrated by the calculations that Curtiss and Rabl (1996) and Spadaro (1999) carried out to verify the relevance of the UWM; the variations of population density considered by these authors have the same effect as variations in the DRF slopes. The reliability of the UWM breaks down only if the variables of the impact calculation are strongly correlated, for instance, in the case of the high local inhalation doses from automotive emissions in large cities. As

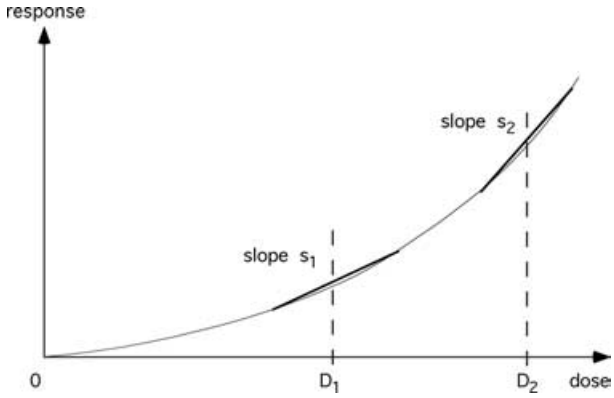


Fig. 3. DRF at two values of the background dose, D_1 and D_2. For small variations around the background the DRF can be approximated by straight lines with slopes s_1 and s_2. The slopes can be different between groups with different backgrounds or different sensitivities. For the calculation of population-total impacts one can take the population-weighted average of the slopes. Doses below a NOAEL are a special case with slope zero.

we argue in Section 5, for ingestion doses the UWM is expected to be excellent because most food is transported over large distances, thus making the distribution of doses very uniform.

3. JUSTIFICATION FOR STEADY-STATE MODELS

If the concentration of a pollutant in a compartment is not uniform, one can subdivide it into smaller compartments, for example, a parcel of air above a city or a layer of soil in a cornfield. In any compartment with first-order processes, the mass m_j in compartment j of the environment can be described by a first-order differential equation in time,

$$\kappa_j m_j + \frac{dm_j}{dt} = \dot{m}_{j,in}, \tag{1}$$

where κ_j = rate parameter, $\dot{m}_{j,in}$ = inflow of pollutant into the compartment (e.g., the emission by a smoke stack into the surrounding air column), and $\kappa_j m_j$ is the outflow. m_j and $\dot{m}_{j,in}$ are functions of time t. Henceforth we assume that the rate parameters can be approximated by time-independent constants, i.e., κ_j = rate constant (=1/time constant).

Without loss of generality one can assume that $\dot{m}_{j,in}(t)$ and $m_j(t)$ are nonzero only for $t > 0$. Then the solution is

$$m_j(t) = \exp(-\kappa_j t) \int_0^t \exp(\kappa_j t')\, \dot{m}_{j,in}(t')\, dt'. \tag{2}$$

Since the dose obtained in this compartment at time t is proportional to the pollutant mass $m_j(t)$ inside, the total (collective) dose D_j, integrated over all time, in this compartment is proportional to the integral

$$D_j = K \int_0^\infty dt\, m_j(t), \tag{3}$$

where K is the proportionality constant between mass and dose. Inserting Equation (2) and changing the order of integration, one can show that the total dose is the integral of the total net flow into the compartment

$$D_j = (K/\kappa) \int_0^\infty dt\, \dot{m}_{j,in}(t)\ . \tag{4}$$

Since with linear dose-response functions only the collective dose matters for the total impact (irrespective of how it is distributed in time or among individuals), a dynamic model consisting of compartments with first-order processes and constant rate parameters yields exactly the same result as a steady-state model with the same compartments, regardless of any detail of the time history of the inflow $\dot{m}_{j,in}(t)$. Therefore, a steady-state model is sufficient for calculating the total dose, even though the real environment is never in steady state. It is easy to see why the argument breaks down if the rate parameters vary with time. Suppose, for example, that the deposition from the atmosphere is nonzero only outside the growing season; then there is no direct deposition on crops even though a calculation with time-averaged parameters would yield such a term.

Under steady-state conditions $\dot{m}_{j,in}$ = constant and the time derivative in Equation (1) is zero; thus one finds that the mass m_j inside the compartment is

$$m_j = \dot{m}_{j,in}/\kappa_j. \tag{5}$$

In passing we note that if there is no removal process the time constant and the total dose are infinite even if the total inflow is finite.

For practical calculations we find it convenient to work in terms of rates by allocating emissions and impacts on an annual basis; in other words we take the emission rate equal to the average during the year and calculate the dose corresponding to an emission pulse of 1 year at this rate. This dose divided by 1 year is the dose rate resulting from the assumed emission rate. The actual time distribution of the dose does not matter for the total. Thus the typical periodicity of emissions and of agricultural production is automatically taken into account.

For soil and for oceans the time constants of some of the removal processes can be very long, much

longer than the time horizon of policy decisions. Thus certain doses, even from small emissions, can be very large if one integrates over all future generations that are affected, and the question arises whether such results should be taken seriously. The weight of future generations is greatly reduced if one applies typical discount rates. There is no universally accepted value for an appropriate intergenerational discount rate, although a consensus seems to be emerging that a lower rate is appropriate for the distant future than for the present generation (for a justification, see Rabl, 1996). The subject of discounting remains controversial and in this article we present undiscounted results. But to enable discounting with different rates for the short and long term, we indicate what fraction of the collective dose is incurred during the first 30 years and the first 100 years.

Actually, the problem involves not only the discount rate: before one discounts a future cost, one must predict how costs will evolve. Taking an extreme illustration, if a perfect cure for cancer is found, all cancer impacts beyond that date can be neglected. As another example consider the prospect that in the future an increasing fraction of the world's seafood will no longer be harvested from the open sea but grown in marine farms, perhaps with techniques to remove pollutants from the water. In view of the rapid progress of medicine and other technologies it is likely that in the more distant future means will be available to mitigate some, if not most, of the impacts of toxic metals. It would be absurd for the present generation to spend scarce resources trying to avoid an impact that in any case would not happen.

But since one cannot count on such progress, the dilemma remains. The models of EPA and IAEA, being conceived in the spirit of calculating peak impacts rather than total impacts, do not even address this problem; their equations in effect limit the time horizon over which impacts are counted to the duration of the emissions, in the order of 20 to 40 years for most installations (we modify the EPA equations so they correspond to steady-state conditions). For the calculation of total impacts one cannot escape this problem, and so we propose two estimates of such impacts: an upper limit corresponding to true steady state, and a lower limit corresponding to the doses that will be reached at the end of a cutoff for which we choose $t_{\text{cut}} = 100$ years as an arbitrary but reasonable number; for the purpose of discounting we also show results for a 30-year cutoff. For a cutoff this means replacing the time constant $1/\kappa$ of a slow process by

$$1/\kappa \to \min[1/\kappa, (1 - \exp(-\kappa t_{\text{cut}}))/\kappa], \qquad (6)$$

since the dose rate from a compartment is proportional to the time constant, and the dose rate at time t is $(1 - \exp(-\kappa t))$ times its asymptotic steady-state limit.

4. DOSE AND IMPACTS FROM INHALATION

The collective inhalation dose rate $\dot{D}_{\text{inhal}}(\dot{m})$ (kg/yr or g/s) can be written as integral over all points $\mathbf{x} = (x, y)$ where people are affected by the pollutant under consideration,

$$\dot{D}_{\text{inhal}}(\dot{m}) = \dot{V}_{\text{inhal}} \int dx\, dy\, \rho(x)\, c_{\text{air}}(x, \dot{m}), \qquad (7)$$

where $\dot{m}$ = rate at which pollutant is emitted to the air (kg/yr), $\dot{V}_{\text{inhal}}$ = population-averaged inhalation rate for which we take 20.6 m^3/(pers/day), $\rho(\mathbf{x})$ = population density (pers/km^2), and $c_{\text{air}}(\mathbf{x}, \dot{m})$ = concentration increment (μg/m^3) at $\mathbf{x}$ due to emission rate $\dot{m}$.

For many impacts one does not need to calculate explicitly a dose for inhalation because the DRFs for inhalation are usually stated in terms of the concentration to which an individual is exposed rather than in terms of the inhaled dose; the units are different and we call such DRFs concentration response functions (CRF). Thus the impact rate due to inhalation can be written as

$$\dot{I}_{\text{inhal}}(\dot{m}) = \int dx \int dy\, s_{\text{CR}}(\mathbf{x})\rho(\mathbf{x})c_{\text{air}}(\mathbf{x}, \dot{m}), \qquad (8)$$

where $\dot{I}_{\text{inhal}}(\dot{m})$ = impact rate (cases/yr) due to emission rate $\dot{m}$, and $s_{\text{CR}}(\mathbf{x})$ = CRF slope at $\mathbf{x}$ ((cases/yr)/(pers $\cdot$ (μg/m^3))).

It is convenient to express the concentration in terms of the flux $F_{\text{dep}}(\mathbf{x}, \dot{m})$, defined as the rate ($\mu$g/($m^2$/s)) at which the pollutant is removed from the atmosphere by dry and/or wet deposition,

$$F_{\text{dep}}(\mathbf{x}, \dot{m}) = c_{\text{air}}(\mathbf{x}, \dot{m})v_{\text{dep}}(\mathbf{x}), \qquad (9)$$

where $v_{\text{dep}}(\mathbf{x})$ = total deposition velocity (dry plus wet) (m/s). The resulting expression,

$$\dot{I}_{\text{inhal}}(\dot{m}) = \int dx\, dy\, s_{\text{CR}}(\mathbf{x})\rho(\mathbf{x})F_{\text{dep}}(\mathbf{x}, \dot{m})/v_{\text{dep}}(\mathbf{x}), \qquad (10)$$

can be evaluated in closed form if $s_{\text{CR}}(\mathbf{x})$, $v_{\text{dep}}(\mathbf{x})$, and $\rho(\mathbf{x})$ are independent of $\mathbf{x}$, because the integral of the deposition flux is equal to the emission rate $\dot{m}$ by virtue of conservation of mass,

$$\dot{m} = \int dx \int dy F_{\text{dep}}(\mathbf{x}, \dot{m}). \qquad (11)$$

In a uniform world where $s_{\text{CR}}(\mathbf{x})$, $v_{\text{dep}}(\mathbf{x})$, and $\rho(\mathbf{x})$ can be replaced by constants s_{CR}, v_{dep}, and ρ, one obtains

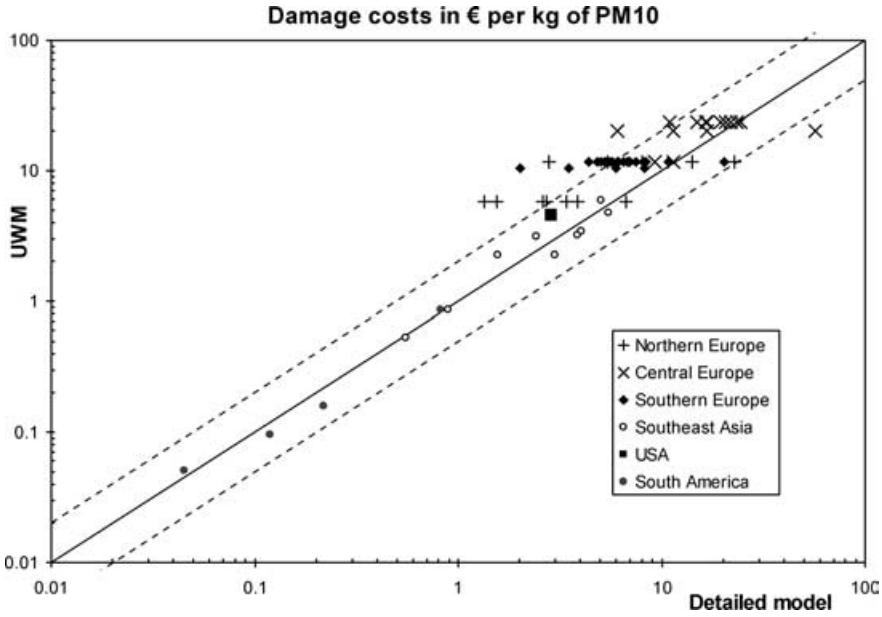


Fig. 4. Comparison of UWM with detailed dispersion models. (The costs cannot be compared between the United States, Asia, and Europe because of different assumptions about DRFs and unit costs.)

the simple expression

$$\dot{I}_{\text{inhal}}(\dot{m}) = s_{\text{CR}}\rho\dot{m}/v_{\text{dep}} \tag{12}$$

called "uniform world model" (UWM) for air dispersion (see Curtiss & Rabl, 1996; Spadaro, 1999). The analogous result for the inhalation dose is

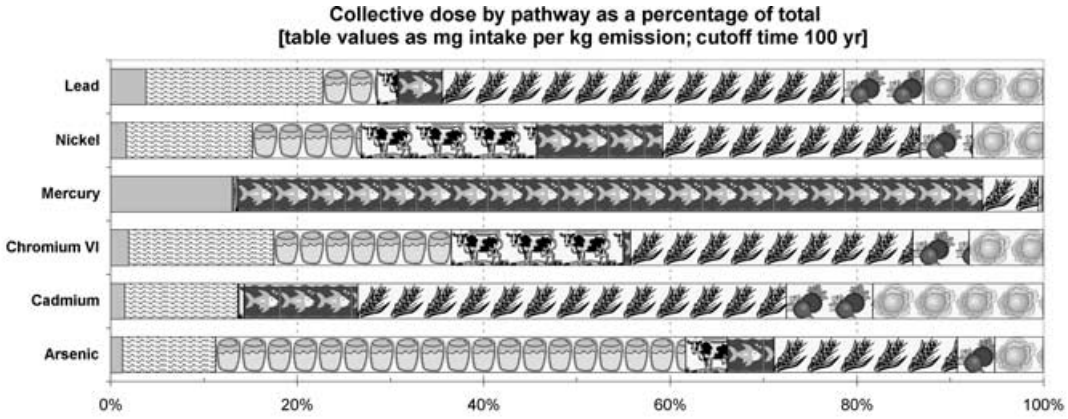
$$\dot{D}_{\text{inhal}}(\dot{m}) = \dot{V}_{\text{inhal}}\rho\dot{m}/v_{\text{dep}}. \tag{13}$$

In Fig. 4 we compare Equation (12) with the results of detailed site-specific calculations for about 100 installations in many countries of Europe, as well as Southeast Asia and America (Spadaro, 1999; Spadaro & Rabl, 2002, and additional calculations by Spadaro). Most of the detailed calculations were done with the EcoSense software (Krewitt *et al.*, 1995) of the ExternE Project series (1998). UWM is so close to the average that it can be recommended for the calculation of typical values for emissions from tall stacks, more than about 50 m; for specific sites the agreement is usually within a factor of 2 to 3. For ground-level emissions in cities the impact can be much larger than UWM because of the combination of high receptor densities with the high concentrations near ground-level sources, but simple estimates can still be obtained by applying correction factors to UWM (see the caption of Fig. 5). The reason why the UWM is such a good representation of typical results is that the latter correspond to an average over many emission sites and that averaging over sites is equivalent to averaging over different distributions of population, thus rendering the distribution more uniform.

The UWM involves the replacement of the average of a product by the product of the averages, an approximation that is justified to the extent that the factors are not correlated with each other and do not vary too much. In practice $F_{\text{dep}}(\mathbf{x}, \dot{m})$ varies the most, being high near the source and decreasing with distance. For sources in large cities this variation is correlated with the population density and so UWM underestimates the impact. For large cities far from the source the strong variation of $\rho(\mathbf{x})$ occurs in a region where the variation of $F_{\text{dep}}(\mathbf{x}, \dot{m})$ is slow, so their contribution is adequately taken into account in the average population density ρ and the acceptability of UWM is not affected. This argument also completes the demonstration in Section 2 why the $s_{\text{CR}}(\mathbf{x})$ can be replaced by its average, as if the CRF were linear.

In the present article we do not consider the possibility of chemical reactions in the atmosphere, but they could readily be included because the UWM can be generalized to secondary pollutants (Curtiss & Rabl, 1996).

The deposition velocity is derived by regression of dispersion data, and the population density ρ is the average (land and water) within a radius of about 500 to 1,000 km. The appropriate radius depends on the deposition velocity: a radius of 1,000 km is appropriate for European conditions, but for regions with higher precipitation and hence more rapid deposition a smaller radius should be used. For example, in Amazonia the deposition velocities are about 3 times



Pathway	Arsenic [a]	Cadmium [a]	Chromium VI [a]	Mercury [c]	Nickel [a]	Lead [b]
Inhalation [d]	3.9	3.9	3.9	22.0	3.9	7.1
Water [e]	31.1	31.7	31.0	0.3	31.5	35.5
Cattle milk	156.2	0.3	38.0	0.4	27.2	10.8
Cattle meat	13.8	1.4	36.8	0.3	43.9	4.1
Freshwater fish	15.6	31.7	1.6	134.9	31.6	9.0
Grains	60.8	119.5	60.4	10.1	64.4	80.4
Root vegetables	12.4	24.1	12.0	0.7	13.1	16.0
Green vegetables	16.2	47.5	15.9	0.3	17.7	24.0
TOTAL	**310**	**260**	**199**	**169**	**233**	**187**

[a] As, Cd, Cr and Ni are modeled as PM10.
[b] Pb is modeled as PM2.5 emitted by cars in typical cities; for power plant emissions as PM10 the ingestion dose would be the same but the inhalation dose would be smaller by a factor of about (1/20)((0.0049/0.0027) (footnote d and Eq.14).
[c] Hg is emitted as metallic Hg, inhalation dose includes only metallic Hg, ingestion dose only methyl Hg.
[d] The inhalation doses are for typical power plant emissions (stack height around 100 m); for As, Cd, Cr, Ni and Pb they should be multiplied by about 3 for typical industrial emissions (near cities, stack height 0 to 40 m), and by about 20 for typical automotive emissions in cities, but for Hg the power plant numbers can also be taken for industrial emissions because global impacts are so much more important than local ones.
[e] Doses from drinking water are lower if the water utilities remove toxic metals.

Fig. 5. Collective doses for central European conditions, by exposure pathway as a percentage of the total (figure) and as intake fraction in mg per emitted kg (table), for the default values of the parameters. Doses from seafood are not included, and results for Hg are tentative and very uncertain. These doses are the intakes, not the absorbed doses.

larger than in Europe and a radius of 500 km is recommended. For central Europe the average population density is $\rho = 80$ pers/km^2 (land and water).

Least squares fits to EcoSense dispersion data for PM emitted in Europe yield deposition velocities of

$$v_{\text{dep}} = 0.0049 \text{ m/s} \quad \text{for PM}_{10}, \quad \text{and}$$
$$v_{\text{dep}} = 0.0027 \text{ m/s} \quad \text{for PM}_{2.5}. \qquad (14)$$

We assume that half of these numbers are due to dry deposition and the other half due to wet deposition at an average precipitation rate of 0.75 m/yr; for other precipitation rates we scale the wet deposition velocity in proportion to the precipitation. Assuming As, Cd, Cr, and Ni to be emitted as part of industrial particles, we use the PM_{10} value; for Pb we consider automotive sources and take the $PM_{2.5}$ value. It is easy to see how the results change with the deposition velocity: only the inhalation dose changes while the ingestion doses remain unaffected. Mathematically, this can be seen from Equation (25). The health impact from inhalation is obviously proportional to $1/v_{\text{dep}}$. The impact from the ingestion doses is independent of v_{dep} because the factor $X_{\text{food},p}$ (defined as ratio of concentration in food product p and concentration in air) is proportional to v_{dep} as can be seen explicitly from the equations in the Appendix. For an intuitive explanation we note that under steady-state conditions the total mass flow from the air to the food

compartments is equal to the emission rate from the source to the air, regardless of how long the pollutant stays in the air.

Hg is emitted almost entirely as metallic vapor, Hg(0). The deposition velocity to use in the UWM is much lower than for $PM_{2.5}$, because even after deposition a large fraction reenters the atmosphere, by volatilization or resuspension. About 1–3% of the Hg in the atmosphere is in the form of reactive gaseous mercury (RGM), apparently due to continual conversion from Hg(0). RGM is soluble and its deposition velocity is much larger, by more than an order of magnitude, than that of Hg(0). After entering the terrestrial and aquatic environment, the mercury is gradually transformed by aquatic sulfate-reducing microorganisms into the very toxic form of methylmercury (MeHg). Approximately 5% (range 1–10%) of the mercury in water is MeHg. The MeHg enters the food chain, especially via fish where it accumulates, the bioaccumulation factor (BAF) being in the order of 10^6. (This paragraph is based on WHO (1991), Sullivan *et al.* (2003), Sullivan, personal communication (2003), and comments by one of the reviewers.)

In view of this we have taken the following approach for the model in this article. For the inhalation dose we consider only Hg(0). Based on Boudala *et al.* (2000) we assume that the time constant for the removal of Hg from the atmosphere is approximately $\tau = 1$–2 years (of which we take the geometric mean to set $\tau = 1.4$ years). The atmospheric time constant is sufficiently long in relation to atmospheric mixing processes (Seinfeld & Pandis, 1998) that as a first approximation one can assume uniform mixing throughout the troposphere over the entire hemisphere. To estimate an appropriate deposition velocity for the UWM, we take the average height of the troposphere as roughly $h = 10$ km. Thus the mass and concentration of Hg in the atmosphere are related by

$$m = Ahc_{\text{air}} \quad \text{for Hg(0)}, \tag{15}$$

where A is the surface area of the hemisphere. Relating the mass to the emission rate $\dot{m}$ according to Equation (5), with rate constant $1/\tau$, one finds

$$\dot{m} = Ahc_{\text{air}}/\tau. \tag{16}$$

Since $\dot{m}$ is also equal to the product of A and the deposition flux $F_{\text{dep}} = c_{\text{air}} v_{\text{dep}}$, one obtains

$$v_{\text{dep}} = h/\tau = 0.000226 \text{ m/s} \quad \text{for Hg}. \tag{17}$$

There are, of course, local variations of the dry and wet deposition rates, but for the purpose of calculating the collective dose we take Equation (17) as average, half of it being dry, half wet. We use the average population density of the northern hemisphere, 21 pers/km^2.

For terrestrial and aquatic pathways and the resulting ingestion doses we consider only MeHg. Not being sure how much of the mercury deposited in soil and in plants gets converted to MeHg, we assume that deposition in plants makes no contribution to the dose but that 5% of the mercury deposited in the soil is converted to MeHg.

5. DOSE AND IMPACTS FROM INGESTION

Let $\dot{d}_{\text{food},p}(\mathbf{x}, \dot{m})$ designate the individual dose rate (kg/(pers/yr)) due to ingestion of a food product p for a person living at $\mathbf{x} = (x, y)$,

$$\dot{d}_{\text{food},p}(\mathbf{x}, \dot{m}) = c_{\text{food},p}(\mathbf{x}, \dot{m}) Q_{\text{food},p}, \tag{18}$$

where $c_{\text{food},p}(\mathbf{x}, \dot{m})$ = pollutant concentration in food product p (kg/kg$_{\text{food}}$) at $\mathbf{x} = (x, y)$ due to emission $\dot{m}$, and $Q_{\text{food},p}$ = quantity of food product p consumed per person (kg$_{\text{food}}$/(pers/yr)).

Data for $Q_{\text{food},p}$ are shown in Table I. Here we take a single value for all of Europe; a more detailed analysis with smaller compartments and an explicit atmospheric dispersion model could distinguish different consumption rates in different regions.

The impact rate $\dot{I}_{\text{food},p}$ (cases/yr) due to this dose is analogous to Equation (8),

$$\dot{I}_{\text{food},p}(\dot{m}) = \int dx' \int dy' s_{\text{DR}}(\mathbf{x}') \times \rho(\mathbf{x}') c_{\text{food},p}(\mathbf{x}', \dot{m}) Q_{\text{food},p}(\mathbf{x}'), \tag{19}$$

where $s_{\text{DR}}(\mathbf{x}')$ = DRF slope at $\mathbf{x}'$ (cases/kg).

The food consumed at $\mathbf{x}'$ is actually grown at a variety of sites $\mathbf{x}$ and transported to $\mathbf{x}'$. The effect of food transport can be described in terms of a transfer

Table I. Default Annual Consumption Rates Q_{food}

Water	0.600	m^3_{wat}/(pers/yr)
Milk + milk products	250	kg$_{\text{milk}}$/(pers/yr)
Meat	100	kg$_{\text{FW}}$/(pers/yr)
Aboveground fruit & vegetables (moisture 86%)	26.5	kg$_{\text{DW}}$/(pers/yr)
Belowground vegetables (moisture 80%)	20.0	kg$_{\text{DW}}$/(pers/yr)
Cereals (moisture 14%)	100.6	kg$_{\text{DW}}$/(pers/yr)
Freshwater fish	3	kg$_{\text{FW}}$/(pers/yr)
Marine fish	6	kg$_{\text{FW}}$/(pers/yr)
Shellfish	1	kg$_{\text{FW}}$/(pers/yr)

Notes: DW = dry weight, FW = fresh weight.
Adapted from Eurobarometer data.

function $X_{\text{trans},p}(\mathbf{x}', \mathbf{x})$ for transport, $X_{\text{trans},p}(\mathbf{x}',\mathbf{x})$ being the fraction of the food product p consumed at $\mathbf{x}'$ that is grown at $\mathbf{x}$,

$$c_{\text{food},p}(\mathbf{x}', \dot{m}) = \int dx \int dy\, X_{\text{trans},p}(\mathbf{x}', \mathbf{x}) c_{\text{food},p}(\mathbf{x}, \dot{m}). \tag{20}$$

Inserting this into Equation (19) and changing the order of integration one can write

$$\dot{I}_{\text{food},p}(\dot{m}) = \int dx \int dy c_{\text{food},p}(\mathbf{x}, \dot{m}) \int dx' \int dy' s_{\text{DR}}(\mathbf{x}') \times \rho(\mathbf{x}') X_{\text{trans},p}(\mathbf{x}', \mathbf{x})\, Q_{\text{food},p}(\mathbf{x}'). \tag{21}$$

The integration over receptor sites $\mathbf{x}'$ has the effect of averaging the distribution of receptors and their characteristics concerning DRFs and food consumption, thus rendering it much more uniform and allowing us to replace $s_{\text{DR}}(\mathbf{x}')$, $\rho(\mathbf{x}')$, and $Q_{\text{food},p}(\mathbf{x}')$ by average values $s_{\text{DR,av}}(\mathbf{x})$, $\rho_{\text{av}}(\mathbf{x})$, and $Q_{\text{food},p,\text{av}}(\mathbf{x})$ at the points $\mathbf{x}$ where the food is grown, the product of these averages being

$$s_{\text{DR,av}}(\mathrm{x})\rho_{\text{av}}(\mathbf{x}) Q_{\text{food},p,\text{av}}(\mathbf{x}) = \int dx' \int dy' s_{\text{DR}}(\mathbf{x}')\rho(\mathbf{x}') X_{\text{trans},p}(\mathbf{x}', \mathbf{x}) Q_{\text{food},p}(\mathbf{x}'). \tag{22}$$

Because most food is transported over large distances, tens to thousands of kilometers, the conditions for the validity of the UWM are even better satisfied for ingestion than for inhalation. In fact, precise modeling of all the respective transport details would be difficult or impossible because of lack of data. In particular, the total ingestion dose is very insensitive to variations in the site where the pollutant is emitted into the air.

Now we define a quantity $X_{\text{food},p}(\mathbf{x})$ as the ratio of the concentration in food product p and the concentration in air, at a point $\mathbf{x} = (x, y)$,

$$X_{\text{food},p}(\mathbf{x}) = c_{\text{food},p}(\mathbf{x}, \dot{m})/c_{\text{air}}(\mathbf{x}, \dot{m}); \tag{23}$$

it is in effect a transfer factor from air to food, in units of $(\text{kg/kg}_{\text{food}})/(\mu\text{g/m}^3)$. $X_{\text{food},p}(\mathbf{x})$ does not depend on $\dot{m}$ because the incremental concentrations are proportional to $\dot{m}$. The equations for $X_{\text{food},p}(\mathbf{x})$ are developed in the following section and in the Appendix. For pathways that pass through the soil, the contribution to the concentration in the food produced at $\mathbf{x}$ is proportional to the local deposition and hence to $c_{\text{air}}(\mathbf{x}, \dot{m})$. Using the default parameters of EPA for the soil pathways the ratio $X_{\text{food},p}(\mathbf{x})$ is the same for all points $\mathbf{x}$ in a watershed. Since for the present version of the model we use the same default values for all of Europe, we can assume $X_{\text{food},p}$ to be independent of $\mathbf{x}$.

Replacing the $X_{\text{food},p}(\mathbf{x})$ by site-independent constants $X_{\text{food},p}$, we obtain, analogous to the derivation of the UWM for inhalation impacts, the UWM for the collective ingestion dose rate

$$\dot{D}_{\text{food},p}(\dot{m}) = X_{\text{food},p} Q_{\text{food},\rho}\, \dot{m}/v_{\text{dep}}. \tag{24}$$

Finally, the total impact rate, $\dot{I}(\dot{m})$(cases/yr), of a pollutant is the sum over inhalation and food products

$$\dot{I}(\dot{m}) = \left(s_{\text{CR}} + s_{\text{DR}} \sum_p X_{\text{food},p} Q_{\text{food},p}\right) \rho\dot{m}/v_{\text{dep}}. \tag{25}$$

This separation of inhalation and ingestion impacts corresponds to the frequent practice of defining the dose-response functions s_{CR} for inhalation in terms of ambient concentrations, whereas the dose-response functions s_{DR} for ingestion are defined in terms of the ingested quantity. Therefore, the units of s_{CR} and s_{DR} are different. Note that all s_{CR} and s_{DR} in this article are defined relative to the average population; if only a certain group, e.g., people over 60 years of age are affected, we include the respective fraction of the population in s_{CR} and s_{DR}.

The ratio of the rates $\dot{I}(\dot{m})$and $\dot{m}$is the unit impact I_u (cases/kg), i.e., the number of cases attributable to the emission of a kilogram of the pollutant

$$I_u = \dot{I}(\dot{m})/\dot{m}. \tag{26}$$

6. CONCENTRATION IN FOOD

The calculation of pollutant concentrations in food begins with an analysis of the concentrations in soil and water. In the EPA model the following pathways are taken into account for pollutants that enter the soil:

- On cropland the pollutants in the top 20 cm (typical root depth) can enter the food grown;
- On pasture the pollutants in the top 10 cm (typical root depth) can enter the feed for farm animals;
- Soil from the top 1 cm can be ingested by farm animals or children.

Like EPA (1998) we do not consider pollutant exchange with deeper soil layers or with ground water (except for leaching, which in our model is an attenuation term, implying a one-way transport to deeper soil layers or ground water for part of the pollutant mass whose impacts we do not consider). In other words,

we assume steady-state conditions in the sense that, averaged over time, the pollutant flow to deep soil or ground water is equal to the outflow from deep soil or ground water to the top 20 cm.

Consider an area A where the deposition flux is $F_{\text{dep}} = v_{\text{dep}} c_{\text{air}}$. According to Equation (5) for steady-state conditions, the pollutant mass $A d_{\text{soil}} \rho_{\text{soil}} c_{\text{soil}}$ in a layer of depth d_{soil} with density ρ_{soil} and average soil concentration c_{soil} ($\text{kg/kg}_{\text{soil}}$) equals the ratio of inflow AF_{dep} and decay rate (here called loss constant) κ_{soil}, which yields the concentration

$$c_{\text{soil}} = \frac{F_{\text{dep}}}{d_{\text{soil}} \rho_{\text{soil}} \kappa_{\text{soil}}}. \tag{27}$$

We calculate values for c_{soil} for three soil depths, 1 cm, 10 cm, and 20 cm. The ratio

$$\frac{c_{\text{soil}}}{c_{\text{air}}} = \frac{v_{\text{dep}}}{d_{\text{soil}} \rho_{\text{soil}} \kappa_{\text{soil}}} \tag{28}$$

is uniform in the entire region if, as in this article, a single set of parameter values is chosen. On irrigated cropland there is an additional deposition term for the pollutant influx from irrigation water. Since this term involves both soil and water concentration, the equations for c_{soil} and c_{water} would have to be combined to solve for the resulting concentrations. But in Europe the fraction of cropland that is irrigated is rather small, only 12% (WRI, 1994, p. 295), and so we neglect this complication as does EPA (1998).

For the soil loss constant κ_{soil} (yr^{-1}) we take into account leaching, runoff, and erosion:

$$\kappa_{\text{soil}} = \kappa_{\text{soil,leach}} + \kappa_{\text{soil,ro}} + \kappa_{\text{soil,er}}. \tag{29}$$

In general, there can also be biotic and abiotic degradation, and volatilization, but for metals their contributions can be neglected. The equations for the soil loss constants are listed in Table A1 of the Appendix, including the default parameter values used in this article.

The calculation of the water concentration involves the flow rate of the pollutant through the rivers and lakes of a watershed. It has four terms: direct deposition into rivers and lakes, pollutant in the runoff from impervious surfaces, pollutant in the runoff from pervious surfaces, and pollutant in the contribution from erosion. The equations are described in Table A2 of the Appendix. For agricultural uses we suppose that the water is not filtered, and so we take the concentration $c_{\text{wc,tot}}$ in the water column (which includes suspended sediments). For drinking water we take the dissolved phase water concentration $c_{\text{wat},d}$ (without suspended sediments). Note that some water utilities employ special treatment for the removal of toxic metals, although lacking general data we do not take this into account in our calculations.

EPA also gives a formula for the concentration in the benthic sediment, but since the consumption of food from this compartment for freshwater is negligible we do not use it (for seafood, by contrast, the benthic compartment would be important because of the large consumption of shellfish).

Substances are assimilated into vegetation due to foliar absorption of external deposits (dry and wet) and uptake from the soil via roots. The contribution of pollutants in irrigation water is easy to include in foliar absorption (unlike root absorption for which the equations for c_{soil} and c_{wat} would have to be coupled), and so we modify Equations (5)–(14) of EPA (1998) by adding to the wet deposition flux on plants the term $c_{\text{wc,tot}} v_{\text{irr}}$ where v_{irr} is irrigation rates, averaged over time and over all cropland.

The total plant concentration is the sum of the contributions from foliar absorption and from uptake through the roots. The equations and default parameter values are listed in Table A3 of the Appendix.

Accumulation in milk and meat occurs when cattle ingest soil, contaminated water, and feed. The intake depends on a number of factors, including animal species, mass, age and growth rate, feed digestibility, and the milk yield for lactating animals. For simplicity we consider only cattle as if all meat consumed were equivalent to beef. The concentrations in meat and in milk are obtained by multiplying the intake of soil, water, and feed by appropriate biotransfer factors B_m. For fish the dissolved phase concentration in water is multiplied by the bioaccumulation factor B_{fish}. The equations and default parameter values are listed in Table A4 of the Appendix.

7. DIRECT EMISSIONS TO SOIL OR WATER

If a pollutant is emitted directly into a river, lake, or the sea, the resulting concentration near the source (first few kilometers) can be calculated with a Gaussian plume model. Beyond a few kilometers of flow in a river one can assume that the pollutant is uniformly mixed; that is the case of interest here. By conservation of mass, the emission rate $\dot{m}$ (kg/s) equals the rate at which the substance flows through a cross-section A_{cross} (m^2) of the river,

$$\dot{m} = c_{\text{river}} A_{\text{cross}} v, \tag{30}$$

where v is flow velocity of river (m/s), and c_{river} is concentration of pollutant in river.

This implies that the concentration is

$$c_{\text{river}} = \dot{m}/(A_{\text{cross}} v) \text{ after a few km from the source.} \quad (31)$$

If this water is used for drinking or irrigation, the resulting health impacts can now be calculated with the method described in the previous section.

For direct emissions to soil one omits the term for deposition on the vegetation, and one replaces the deposition flux to the soil F_{dep} by F_{soil}, the emission rate per unit soil surface over which the pollutant is emitted,

$$F_{\text{soil}} = \dot{m}/A_{\text{soil}}. \quad (32)$$

8. RESULTS FOR DOSES

Fig. 5 shows the collective dose in milligrams due to the atmospheric emission of 1 kg of the respective metals under typical central European conditions. The average population density is 80 pers/km^2 (land and water). Taken as dimensionless ratios, the numbers in the table under Fig. 5, multiplied by 10^{-6}, are the fraction of the emitted pollutant that passes through human bodies; this is sometimes called the intake fraction.

The doses shown are the total ingested or inhaled quantities, without regard to the fraction that is actually absorbed. If the absorption rates are less than 100%, they have to be included before applying DRFs that are based on absorbed dose. Inhalation and ingestion can be associated with very different DRFs. For example, As is more carcinogenic, per mass, if inhaled than if ingested.

The total dose can be much larger than the inhalation dose, by about 2 orders of magnitude. A simple back-of-the-envelope calculation may be instructive in explaining why ingestion can be so much more important than inhalation. Consider an average person exposed to air with concentration c_{air}. The annual inhalation dose is $D_{\text{inhal}} = c_{\text{air}} V_{\text{inhal}}$ with an annual inhalation volume $V_{\text{inhal}} = 7520$ m^3/(pers/yr) (we take a period of a year but that choice has no effect on the result since the argument involves time-averaged values). If aboveground food crops are exposed to the same concentration, the ingestion dose due to direct deposition on the plants is $D_{\text{ing}} = c_{\text{air}} v_{\text{dep}} A_{\text{crop}} \times 1$ yr where A_{crop} is the horizontal area of the crops intercepting the deposition flux. The plant yield of 2.24 kg$_{\text{DW}}$/m^2 in Table A3 of the Appendix together with the consumption rate of 127 kg$_{\text{DW}}$/(pers/yr) for aboveground crops implies an area of $127/2.24 = 56.7$ m^2 per person; however, this number has to be reduced by the fraction of the year the plants are grown (say 2 months/yr) and by the ratio intercepting area/ground area (say 0.2). Thus we take $A_{\text{crop}} = 56.7\,\text{m}^2 \times (2/12) \times 0.2 = 1.9$ m^2. The resulting ratio of ingestion and inhalation doses is $D_{\text{ing}}/D_{\text{inhal}} = v_{\text{dep}} A_{\text{crop}} \times 1\,\text{yr}/V_{\text{inhal}} = 40$, for a typical v_{dep} of 0.005 m/s $= 1.58 \times 10$ m/yr. The ratio is reduced to the extent that the pollutant is not absorbed by the edible portions of the plant, but it increases by the contributions of belowground crops, milk, and meat. In any case, for pollutants that are absorbed by plants, ingestion can indeed be much more important than inhalation.

We have performed a sensitivity analysis to evaluate how much the intake fractions vary with changes in the input parameters; the results are shown in Table A5 of the Appendix. The most critical parameters are the yield per planted area, and in some cases the soil-plant bioconcentration factors and the biotransfer factors for meat and milk. The choice of the deposition velocities is not very critical because of the relative smallness of inhalation. The last lines of Table A5 show how the doses change if the cutoff time t_{cut} in Equation (6) is changed to 30 years and to infinity: extending the time horizon has an appreciable effect for Pb because of the long soil loss time constants.

The results for the ratio ingestion/inhalation are consistent with data reported by WHO (1988–2001). Among models that should give comparable results we have found the CalTOX model of McKone and Enoch (2002), a level IV model in the terminology of MacKay (2002). It analyzes essentially the same pathways as our model; in particular ingestion of seafood and exchanges with ground water are not considered. We have run CalTOX for the most comparable scenario, i.e., in the continuous emission mode with the settings landscape = United States, start of exposure = 30 years, exposure duration = 70 years, and exposure factors = LCIA, to calculate the intake fractions for inhalation and for ingestion. Our inhalation doses tend to be higher than those of CalTOX, our ingestion doses lower. Table II shows the ratios of the total doses calculated by our model and by CalTOX, after multiplying the CalTOX results by the ratio 80/29 of population densities in the EU and the United States. For As, Cr, and Ni the results are fairly close, considering the uncertainties; for Cd and Pb our numbers are 14 to 20 times lower. We do not show a comparison for Hg because the differences in

Table II. Ratio of Total Doses Calculated by Our Model (UWM) and by CalTOX, After Multiplying the CalTOX Results by the Ratio 80/29 of Population Densities in Central Europe and the United States

Ratio of Doses	As	Cd	Cr	Ni	Pb
UWM/CalTOX	0.61	0.07	0.39	0.60	0.05

modeling are too large: we calculate the total dose in the entire hemisphere (inhalation in metallic form, ingestion as methyl Hg), whereas CalTOX considers only the dose in the United States (for Hg or for methyl Hg, without transformation from Hg to methyl Hg).

There are two principal differences between CalTOX and our model: one lies in the modeling of atmospheric dispersion, and the other in the transfer between compartments. Whereas we use empirically determined transfer factors, mostly of EPA, CalTOX calculates the transfer by means of fugacity data. For the dispersion in the atmosphere we assume that As, Cd, Cr, and Ni are emitted as part of PM_{10} (for industrial and power plant emissions) and Pb as part of $PM_{2.5}$ (for automotive emissions), being dispersed and deposited on the ground like other particulate matter of its size. CalTOX, by contrast, assumes emission in pure metallic form, the metals then attaching themselves to other particulate matter in the atmosphere according to the fugacity between the metal under consideration and the particles that are already in the atmosphere (their concentration is one of the input parameters). Since the transfer from metallic phase to particles occurs at different rates for different metals, the atmospheric residence time and hence the inhalation dose are different for different metals. The atmosphere of CalTOX is modeled as a homogeneous perfectly mixed compartment with volume equal to the height of the atmosphere times the impact area under consideration (land area of the United States for the setting landscape = U.S.). We believe that our treatment of the atmosphere is more realistic because it has been explicitly validated by numerous comparisons with detailed atmospheric models. For the transfers between the other compartments we do not know which approach is more reliable.

9. RESULTS FOR IMPACTS AND SOCIAL COSTS

Obviously, impacts can be quantified only to the extent that the slopes of the CRFs or DRFs in the relevant dose range are known. Unfortunately, there is a dearth of information. For most substances the only available data indicate an LOAEL (lowest observed adverse effects level) or NOAEL (no observed adverse effects level), usually from animal tests. Recently, Pennington *et al.* (2002) have proposed a promising method of using LOAEL or NOAEL data for estimating DRFs, but among toxic metals their only result so far is for Hg.

For CRFs determined by epidemiological studies, the question arises whether the effect of the ingestion dose should be added to that of inhalation. This depends on what exactly was measured in the epidemiological study. Typically, the study population was exposed simultaneously via inhalation and ingestion. Even if the result of a study is stated as CRF, i.e., in terms of ambient air concentration, it may in fact reflect the total dose. But if the ratio of inhalation and ingestion for the general population is different from that of the study population, one does not know how to apply the CRF unless one can make reasonable assumptions about the separate inhalation and ingestion doses of the study population and the relative effectiveness of these two dose routes.

For the carcinogenic metals, As, Cd, Cr (in oxidation state 6), and Ni, the CRFs for inhalation given by EPA are stated as unit risk, shown here in the third line of Table III; they are the probability, per $\mu g/m^3$ of ambient concentration, of getting a cancer due to a lifetime exposure, taken as 70 years. With our definition of the CRF as impact for a 1-year exposure, the slope s_{CR} is the unit risk divided by 70. The fifth row shows the cancers per kg of emitted pollutant, based on the inhalation dose only.

At the present time the evidence for cancers due to the ingestion of Cd, Cr, and Ni is not sufficiently convincing for EPA to indicate a DRF. As ingestion is considered carcinogenic, with slope factor 1.5 per mg/(kg/day). Since the slope factor indicates the lifetime risk due to ingesting the same dose every day for 70 years, we need to divide by 70×365 days and the average weight of 55 kg/pers (adults and children) to obtain the DRF in our units. Multiplying the resulting s_{DR} by the total ingestion dose in Fig. 5, we find 3.32×10^{-4} cancers per kg due to ingestion of As, much more than due to inhalation although not in proportion to the dose ratio; ingestion seems to cause fewer cancers per dose than inhalation. But that may be a serious overestimate because it assumes the same toxicity for organic and inorganic As. At the present time EPA and the International Agency for Research

	As	Cd	Cr-VI [a]	Ni
Inhalation				
Unit risk (cancers/(pers/70/yr/μg/m^3))	4.30×10^{-3}	1.80×10^{-3}	1.20×10^{-2}	2.40×10^{-4}
s_{CR} (cancers/(pers/yr/kg/m^3))	6.14×10^{4}	2.57×10^{4}	1.71×10^{4}	3.43×10^{3}
Cancers/kg, inhalation, UWM	3.18×10^{-5}	1.33×10^{-5}	8.85×10^{-5}	1.78×10^{-6}
Ingestion				
Slope factor (cancers/(mg/(kg$_{body}$/day)))	1.50×10			
s_{DR} (cancers/kg)	1.07×10			
Cancers/kg, ingestion	3.32×10^{-5}[b]			
Total cancers/kg	6.5×10^{-5}	1.33×10^{-5}	8.85×10^{-5}	1.78×10^{-6}
Cost/kg (€/kg) at 2 M€/cancer	130	27	177	3.6

Table III. CRFs, DRFs and Impacts, per kg Emitted, for the Carcinogenic Metals

Unit risk and slope factor from IRIS http://www.epa.gov/iris.
[a]If only total Cr emission is known, one must estimate the fraction in the VI oxidation state; typical numbers are 11% for coal-fired and 18% for oil-fired power plants, according to EPA (www.epa.gov/ttncaaa1/t3/meta/m28497.html and www.epa.gov/ttncaaa1/t3/meta/m27812.html).
[b]Dose from drinking water only.

on Cancer do not provide any information on the carcinogenicity of organic As. Most of the ingestion dose is organic, with the exception of drinking water, which is inorganic. Taking only the dose from drinking water, the cancers from ingestion are only 1/10th that of 3.32×10^{-5} cancers per kg of As, as shown in Table III. Even that is an overestimate if some of the As is removed by water treatment.

For the social cost of cancers we cite the value of about 2 M€ per cancer used by the ExternE project series; it is an average over fatal and nonfatal cancers and takes into account the shortening of life expectancy. If other endpoints were included, the cost would be higher, but we do not know how much.

Finally, we calculate the impact and damage cost of IQ decrement due to Pb, a cost that can be quantified with present knowledge and probably the dominant part of the total damage cost of Pb. The dose-response function is quite well determined, thanks to a meta-analysis by Schwartz (1994) who found a decrement of 0.026 IQ points for a 1 μg/L increase of Pb in blood, a relation that appears to be linear without threshold. More recently, a study designed to identify effects at the lowest doses found an even larger effect, 0.055 IQ points per 1 μg/L, without any threshold (Lanphear *et al.*, 2000). Here we continue to use 0.026 IQ points per 1 μg/L, being based on meta-analysis rather than a single study.

To relate blood level to exposure and dose we have found two options, and so we present two calculations. The first is a relation recommended by a recent U.K. review (EPAQS, 1998), which finds that a 1.0 μg/m^3 incremental exposure to Pb in ambient air increases the blood level by 50 μg/L, not very different from values in an earlier review by Brunekreef (1984). Combined with 0.026 IQ points per 1 μg/L, this implies a loss of 1.3 IQ points per child per μg/m^3.

We also need to consider the time window during which an exposure causes damage. The sensitivity of the brain to Pb is greatest during the first 2 years of life, although the precise time distribution of the damage is not known. However, this does not matter since the result of Schwartz expresses the total impact in a population due to a constant exposure. Furthermore, the half-life of Pb in blood and other soft tissues is relatively short, about 28–36 days (although much longer in bones) (WHO, 1995). Thus, for the purpose of damage calculations, one can equally well assume that the damage is incurred during a 1-year exposure by infants between the ages of 0 and 1 year only, or during a 3-year exposure between the ages of 0 and 3 years. To see that the effect is the same, note that the percentage of the population between 0 and 3 years is essentially 3 times the percentage between 0 and 1 year, the latter being 1.1% in the EU. If the sensitive period is only 1 year, the loss due to a 1-year exposure is 1.3 IQ points/(μg/m^3) $\times$ 1.1% of population of EU. If the sensitive period is 3 years, the affected cohort is essentially 3 times as large but the damage rate is 3 times smaller, so the loss due to a 1-year exposure is (1.3 IQ points/(μg/m^3))/3 $\times$ (3 $\times$ 1.1% of population of EU), essentially the same. To express the CRF slope in a form consistent with this article, i.e., relative to the entire population, we therefore multiply the 1.3 IQ points/(μg/m^3) by the fraction of the population that is affected (1.1% per year), to obtain

$$s_{CR} = 1.43 \times 10^{-2} \text{IQ points/(pers/yr}(\mu\text{g/m}^3)). \quad (33)$$

We use this function without adding further contribution from ingestion because the above relation between ambient concentration and blood level has been observed in populations that also received a dose from ingestion; thus the ingestion dose is implicitly taken into account.

Combining Equation (13) of the UWM, multiplied by a factor 20 for automotive emissions in cities, with the CRF of Equation (33) we find a loss of 0.268 IQ points per kg of Pb. For the cost associated with the loss of an IQ point there is a range of estimates, mostly based on lost earnings or remedial education. Lutter (2000) indicates 3,000 €/IQ point, whereas Grosse *et al.* (2002) estimate $14,500 per IQ point and Muir and Zegarac (2001) $15,000 per IQ point. In the EU the GREENSENSE project (2004) offers an estimate of 8,600 €/IQ point. Here we take 10,000 €/IQ point as intermediate estimate. Thus we find a damage cost per kg of Pb emitted in Europe of 10,000 €/IQ point × 0.268 IQ points/kg of Pb = 2,680 €/kg, based on the relation between blood level and concentration in air.

The second option is a relation between blood-level Pb and ingestion dose, published by WHO (1995). Surprisingly, the blood level per ingested quantity is higher at low doses, perhaps because of increased excretion at higher dose or storage in bones. Here we use the level found at the lower dose, 72 μg/L, for infants who ingest 17 μg/day, or 4.2 μg/L per ingested μg/day. Together with the above-mentioned 0.026 IQ points per 1 μg/L increase of blood Pb this implies a loss of 0.026 IQ points × 4.2 (μg/L)/(μg/day) × (1 yr/365 days) = 3.02 $\times 10^{-4}$ IQ points/(μg/yr) per child. As in the argument leading to Equation (33) we multiply this number by 1.1%, the fraction of the total population below 1 year of age and sensitive to Pb, to obtain a DRF slope of

$$s_{DR} = 3.30 \times 10^3 \text{ IQ points/(kg}_{\text{absorbed}}). \quad (34)$$

Again, the duration of the sensitive period during infancy does not matter. Multiplied by the collective ingestion dose of 1.87×10^{-4} kg per kg emitted (with 100-yr cutoff), this yields an impact of 0.59 IQ points per emitted kg. At 10,000 €/IQ point the cost is 5,900 €/kg. The variation with source site is weak because the contribution of the inhalation dose in this calculation is very small. We have more confidence in the relation of blood level with ingestion than with inhalation. The relation with inhalation appears less reliable, as the inhalation/ingestion ratio is likely to be quite variable with site, and over time as well, especially with the phasing out of leaded gasoline.

The motivation for this calculation is the fact that even the so-called unleaded gasoline can contain Pb: the regulatory limit for unleaded gasoline in the EU after 2000 is 5 mg/L (EC, 1998). At this level, and with 5,900 €/kg, the associated damage cost would be 3 Eurocent/L, small but not negligible compared to the price of gasoline. However, the real level is lower, in the range of 1–2 mg/L during 2001–2002, and is expected to decline further as residual contamination is eliminated from the system (CONCAWE, Gasoline Quality Survey 2001/2002, October 2003).

10. CONCLUSIONS

We have presented a simple multimedia model for the calculation of the expectation value of collective doses and impacts due to the atmospheric emission of toxic metals. It is totally different in purpose and methodology from conventional models such as EUSES (1997) that analyze a particular population at risk or a particular episode, usually under "worst-case" assumptions. The results indicate that the ingestion dose can be about 2 orders of magnitude larger than the dose from inhalation alone. We have also calculated several impacts for which DRFs appear to be reasonably well established: cancers due to As, Cd, Cr, and Ni, as well as IQ decrement due to Pb. The model could readily be extended to organic pollutants.

The uncertainties are large, easily an order of magnitude, the main uncertainties arising from the parameter values of the model, in particular the transfer factors. For the parameters a wide range of values can be found in the literature, sometimes even spanning several orders of magnitude. However, one should note that in many cases the range is a reflection of local or temporal variability rather than uncertainty (although such variability causes uncertainty when the local or temporal conditions are not sufficiently well known). Since we are interested in collective doses and impacts due to typical sources of the pollutants, we have taken typical parameter values for average conditions. For the collective doses the uncertainties due to local or temporal variability may be relatively small because of averaging over different local conditions.

An important pathway not included in the current model is the ingestion of seafood. Another shortcoming of the present formulation of the model is

the lack of attention to the chemical form in which the metals are ingested. The chemistry can play an important role because the toxicity can vary greatly between different chemical compounds of the same metal; for example, Cr is carcinogenic only in the VI oxidation state. If sufficient data were available, this point could be taken into account by distinguishing the different compositions typically found in the different food products and then using the respective dose-response functions for each composition. Other than that the chemical form of the pollutant is implicitly taken into account because the transfer factors are stated in terms of the mass of the respective metals for typical conditions, whatever their chemical forms may be in a specific medium.

As far as the application to the quantification of impacts and damage costs is concerned, the principal difficulty lies in the limitations of our knowledge about the DRFs. Even for the endpoints that we have quantified, large question marks remain about the relative contributions of inhalation and ingestion and about the chemical form. In the case of CRFs that have been determined by epidemiological studies and that are stated in terms of ambient concentrations, i.e., inhalation alone, it is not clear to what extent part of the impact may have been due to a simultaneous ingestion dose. The lack of reliable DRFs or CRFs at low doses is especially frustrating in the case of Hg and its compounds because this is a highly neurotoxic element that is emitted in large quantities.

ACKNOWLEDGMENTS

Part of this work has been performed within the ExternE Project series of the European Commission DG Research. We are grateful for helpful discussions with Leo de Nocker, Luc Int Panis, and Rudi Torfs, and especially with Till Bachmann and Terry Sullivan; the comments of the reviewers are also acknowledged with thanks.

APPENDIX

Table A1. Equations and Input Data for Toxic Metal Concentrations in Soil

Parameters	Definitions and Units	Equations (Reference in EPA, 1998)	Default Values for This Article
c_{soil}	Average soil concentration (kg/kg_{soil})	$\frac{c_{soil}}{c_{air}} = \frac{v_{dep}}{d_{soil}\rho_{soil}\kappa_{soil}}$ (for Hg it is multiplied by fraction that is converted to MeHg)	
c_{air}	Ambient air concentration (kg/m^3_{air})		
v_{dep}	Total (dry + wet) deposition velocity (m/s)		As, Cd, Cr, and Ni: 0.0049 Pb: 0.0027
d_{soil}	Soil depth (m)		Ingestion: 0.01 Animal feed: 0.10 Food crops: 0.20
ρ_{soil}	Soil bulk density (kg_{soil}/m^3_{soil})		1500
κ_{soil}	Total soil loss constant (1/s)	$\kappa_{soil} = \kappa_{soil,leach} + \kappa_{soil,ro} + \kappa_{soil,er} = \frac{1}{\tau_{soil}}$ τ_{soil} = soil time constant (s)	
$\kappa_{soil,leach}$	Loss constant for leaching (1/s)	$\kappa_{soil,leach} = \frac{v_{prec} + v_{irr} - v_{ro} - v_{evap}}{d_{soil}} \frac{1}{\theta_{sw} + K_{sw}\rho_{soil}}$ (Equaion 5-5A)	
v_{prec}	Mean precipitation rate (mm/yr)		750
v_{irr}	Mean irrigation rate (mm/yr)		110
v_{ro}	Mean surface runoff from pervious area (mm/yr)		100
v_{evap}	Mean evaporation rate (mm/yr)		300
K_{sw}	Soil-water partition coefficient ($(kg/kg_{soil})/(kg/m^3_{wat})$)	$K_{sw} = \frac{c_{soil}}{c_{wat}}$ c_{wat} = water concentration (kg/m^3_{wat})	At pH 6.8(default) *4.9* *8.0* As 0.029 *0.025* *0.031* Cd 0.075 *0.015* *4.3* Cr 0.019 *0.031* *0.014* Hg 7.0 Ni 0.065 *0.016* *1.9* Pb 0.9
$\theta_{s,wat}$	Soil volumetric water content (m^3_{wat}/m^3_{soil})		0.2
$\kappa_{soil,ro}$	Soil loss constant for surface runoff (1/s)	$\kappa_{soil,ro} = \frac{v_{ro}}{d_{soil}} \frac{1}{\theta_{sw} + K_{sw}\rho_{soil}}$ (Equation 5-4)	
$\kappa_{soil,er}$	Soil loss constant for erosion (1/s)	$\kappa_{soil,er} = \frac{L_{soil} R_{sed} R_{en}}{d_{soil}} \frac{K_{sw}}{\theta_{sw} + K_{sw}\rho_{soil}}$ (Equation 5-3)	
L_{soil}	Unit soil loss ($kg_{soil}/(m^2/s)$)	$L_{soil} = RF \times K \times LS \times C \times PF$ (Equation 5-33A) $\times \frac{907.18\,kg/ton}{4047\,m^2/acre}$ (Equation 5-33A)	RF = Erosivity factor = 122.5 (1/yr) K = Erodibility factor = 0.39 ton/acre LS = Length slope factor = 1.5 C = Cover management factor = 0.1 PF = Supporting practice factor = 1.0 L_{soil} = 1.61 ($kg_{soil}/(m^2/s)$)
R_{sed}	Sedimentary delivery ratio (–)	(Equation 5-34 for 100 square miles)	0.1065
R_{en}	Soil enrichment ratio (–)		1.0
$X_{soil,ing}$	Air-to-soil transfer factor for ingestion ($(kg/kg_{soil})/(kg/m^3_{air})$)	$X_{soil,ing} = \frac{c_{soil,ing}}{c_{air}}$	

Table A2. Equations and Input Data for Toxic Metal Concentrations in Water

Parameters	Definitions and Units	Equations (Reference in EPA, 1998)	Default Values for This Article
$c_{wat,tot}$	Total water body concentration (kg/m^3_{wat}) (water column + upper benthic sediment layer)	$c_{wat,tot} = \frac{\dot{m}_{wat}}{\dot{V}_{wat} f_{wc} + \kappa_{wat} A_{wat} (d_{wc} + d_{bs})}$	
$\dot{m}_{wat}$	Total pollutant flux to water (kg/yr)	$\dot{m}_{wat} = A_{wat} c_{air} v_{dep} + \dot{m}_{ro,i} + \dot{m}_{ro,p} + \dot{m}_{er}$ $A_{wat} c_{air} v_{dep}$= direct deposition to water area A_{wat} (for Hg A_{wat} c_{air} v_{dep} is multiplied by fraction that is converted to MeHg)	$A_{wat} = 2\%$ of A_{land} A_{land} = watershed surface area
$\dot{m}_{ro,i}$	Runoff from impervious area to water (kg/yr)	$\dot{m}_{ro,i} = A_{imp} c_{air} v_{dep}$; A_{imp}= impervious area of watershed	$A_{imp} = 10\%$ of A_{land}
$\dot{m}_{ro,p}$	Runoff from pervious area to water (kg/yr)	$\dot{m}_{ro,p} = v_{ro}(A_{land} - A_{wat} - A_{imp}) \frac{c_{soil} \rho_{soil}}{\theta_{s,wat} + K_{sw} \rho_{soil}}$ (Equation 5-32)	
$\dot{m}_{er}$	Erosion from pervious area to water (kg/yr)	$\dot{m}_{er} = L_{soil}(A_{land} - A_{imp}) R_{sed} R_{en} \frac{c_{soil} K_{sw} \rho_{soil}}{\theta_{s,wat} + K_{sw} \rho_{soil}}$ (Equation 5-33)	
$\dot{V}_{wat}$	Water flow rate through watershed (m^3_{wat}/yr)	$\dot{V}_{wat} = A_{land}(v_{prec} - v_{evap})$	
f_{wc}	Fraction of $c_{wat,tot}$ that is in water column (−)	$f_{wc} = \frac{(1 + K_{sw} c_{sussol}) d_{wc}}{(1 + K_{sw} c_{sussol}) d_{wc} + (\theta_{bs} + K_{sw} c_{bs}) d_{bs}}$ (Equation 5-36)	
c_{sussol}	Total suspended solids concentration (kg_{sed}/m^3_{wat})		0.01
d_{wc}	Water column depth (m)		1.0
θ_{bs}	Bed sediment porosity (m^3_{wat}/m^3_{sed})		0.6
c_{bs}	Bed sediment concentration (kg_{sed}/m^3_{wat})		100
d_{bs}	Upper benthic sediment layer (m)		0.03
κ_{wat}	Overall water body loss constant (1/s)	$\kappa_{wat} = f_{bs} \kappa_b$; $f_{bs} = 1 - f_{wc}$; $\kappa_b = \frac{L_{soil} A_{land} R_{sed} - \dot{V}_{wat} c_{sussol}}{A_{wat} c_{bs} d_{bs}}$; κ_b = Benthic rate burial constant (Equation 5-38)	
$c_{wc,tot}$	Water column concentration (kg/m^3_{wat}) (dissolved phase + suspended sediment)	$c_{wc,tot} = f_{cw} c_{wat,tot} \frac{(d_{wc} + d_{bs})}{d_{wc}}$ (Equation 5-45)	
$c_{wat,d}$	Dissolved phase water concentration (kg/m^3_{wat})	$c_{wat,d} = \frac{c_{wc,tot}}{1 + K_{sw} c_{sussol}}$ (Equation 5-46)	
X_{wat}	Air-to-water transfer factor (m^3_{air}/m^3_{wat})	$X_{wat} = \frac{c_{wat,d}}{c_{air}}$	

Table A3. Equations and Input Data for Toxic Metal Concentrations in Vegetation

Parameters	Definitions and Units	Equations (Reference in EPA, 1998)	Default Values for This Article
$c_{plant,foliar}$	Plant concentration from foliar absorption (kg/kg_{DW})	$c_{plant,foliar} = \frac{[v_{dep,dry}c_{air} + f_{wet}(v_{dep,wet}c_{air} + f_{irr}v_{irr}c_{wc,tot})]f_{int}[1-\exp(-\kappa_p t_p)]}{Y_p \kappa_p}$ (based on Equation 5-14) (for Hg c_{air} is set to 0 and $c_{wc,tot}$ multiplied by fraction that is converted to MeHg)	
$v_{dep,dry}$	Dry deposition velocity (m/s)	$v_{dep,dry} = v_{dep} - v_{dep,wet}$ with $v_{dep,wet}$ = wet deposition velocity (m/s) = $\frac{v_{dep}}{2} \times (\frac{v_{prec}}{750\ mm/yr})$	
f_{wet}	Fraction of wet deposition that adheres to plant (−)		60%
f_{irr}	Fraction of irrigated cropland (−)		12%
f_{int}	Fraction of deposition intercepted by edible portion of plant (−)		Food crops: 39% Forage: 50% Silage: 46%
v_{irr}	Irrigation rate, averaged over time and all cropland (mm/yr)	Typically, around 10 $kg_{wat}/(m^2 \cdot day)$ during irrigation days; 12% of cropland in EU is irrigated 3 months of the year	110
κ_p	Plant surface loss constant (yr^{-1})		18
t_p	Exposure time of edible portion of plant per harvest (yr)		Food crops: 0.16 Forage: 0.12 Silage: 0.16
Y_p	Yield per planted area (kg_{DW}/m^2)		Food crops: 2.24 Forage: 0.24 Silage: 0.8
$c_{plant,root}$	Plant concentration from root uptake (kg/kg_{DW})	$c_{plant,root} = c_{soil} \times B_{root}$	
B_{root}	Plant-soil bioconcentration factor [$(kg/kg_{DW})/(kg/kg_{soil})$]		**Green vegetables** / **Root vegetables** As: 0.00633 / As: 0.008 Cd: 0.125 / Cd: 0.064 Cr : 0.00488 / Cr: 0.0045 Hg: 0.0294 / Hg: 0.099 Ni: 0.00931 / Ni: 0.008 Pb: 0.0136 / Pb: 0.009 **Grains** / **Animal feed** As: 0.004 / As: 0.036 Cd: 0.062 / Cd: 0.364 Cr: 0.0045 / Cr: 0.0075 Hg: 0.3 / Hg: 0.3 Ni: 0.006 / Ni: 0.032 Pb: 0.009 / Pb: 0.045
$c_{plant,tot}$	Total pollutant concentration in plant (kg/kg_{DW})	$c_{plant,tot} = c_{plant,foliar} + c_{plant,root}$	
X_{veg}	Air-to-vegetables transfer factors (m^3_{air}/kg_{DW})	Green : $X_{gv} = \frac{c_{plant,tot,gv}}{c_{air}}$; Root: $X_{rv} = \frac{c_{plant,tot,rv}}{c_{air}}$	
X_{grains}	Air-to-grains transfer factor (m^3_{air}/kg_{DW})	$X_{grains} = \frac{c_{plant,tot,grains}}{c_{air}}$	
X_{feed}	Air-to-animal feed transfer factors (m^3_{air}/kg_{DW})	Forage : $X_{forage} = \frac{c_{plant,tot,forage}}{c_{air}}$; Silage : $X_{silage} = \frac{c_{plant,tot,silage}}{c_{air}}$	

Table A4. Equations and Input Data for Toxic Metal Concentrations in Meat, Milk, and Fish

Parameters	Definitions and Units	Equations (Reference in EPA, 1998)	Default Values for This Article
c_m	Pollutant concentration in milk or meat from cattle (kg/kg_{milk} or $kg/kg_{beef,FW}$)	$c_m = B_m \Sigma_i\ Q_{feed,i} \times c_{feed,i}$ (Equation 5-22); $Q_{feed,i}$ = quantity of feed intake of type i, including water, forage, silage, grains, and ingestion of surface soil; $c_{feed,i}$ = concentration in feed of type i (for water $c_{wc,tot}$); X_m = transfer factor air to milk or meat (m^3_{air}/kg_{milk} or $m^3_{air}/kg_{beef,FW}$) = c_m/c_{air}	$Q_{feed,i}$ Beef cattle Milk cattle water [m^3_{wat}/day]: 0.04 0.06 forage [kg_{DW}/day]: 8.8 13.2 silage [kg_{DW}/day]: 2.5 4.1 grains [kg_{DW}/day]: 0.47 3 soil [kg_{soil}/day]: 0.5 0.4
B_m	Biotransfer factor for milk or meat from cattle (day/kg_{milk} or $day/kg_{meat,FW}$)		For meat / For milk As: 0.002 / 0.006 Cd: 0.00012 / 0.0000065 Cr: 0.0055 / 0.0015 Hg: 0.00078 / 0.000338 Ni: 0.006 / 0.001 Pb: 0.0003 / 0.00025
c_{fish}	Pollutant concentration in fish ($kg/kg_{fish,FW}$)	$c_{fish} = c_{wc,tot} \times B_{fish}$	
B_{fish}	Bioconcentration factor for freshwater fish ($m^3_{wat}/kg_{fish,FW}$)		As: 0.1 Cd: 0.2 Cr: 0.01 Hg: 100 Ni: 0.2 Pb: 0.05
X_{fish}	Air-to-freshwater fish transfer factor ($m^3_{air}/kg_{fish,FW}$)	$X_{fish} = \frac{c_{fish}}{c_{air}}$	

Table A5. Sensitivity of Intake Fractions to Changes of Input Parameters (Normalized by Base Case with $t_{cut} = 100$ yr)

Parameters Changed in Base Case	As	Cd	Cr	Ni	Pb
Base case (values in paper)	1.000	1.000	1.000	1.000	1.000
$v_{dry} = v_{wet} = 0.335$ cm/s (As, Cd, Cr, Ni, Pb); $v_{dry} = v_{wet} = 0.0053$ cm/s (Hg)	0.996	0.995	0.994	0.995	0.974
$v_{dry} = v_{wet} = 1$ cm/s (As, Cd, Cr, Ni, Pb); $v_{dry} = v_{wet} = 0.016$ cm/s (Hg)	0.990	0.987	0.984	0.985	0.963
$v_{dry} = 3 \times v_{we}$	1.107	1.048	1.108	1.092	1.071
$v_{dry} = 0.333 \times v_{we}$	0.893	0.952	0.892	0.908	0.929
$d_{soil,ingestion} = 3$ cm (3 × Base case)	1.000	1.000	1.000	1.000	0.979
$v_{irr} = 33$ cm/s (3 × Base case)	0.979	0.868	0.984	0.956	0.966
Soil-water partition factors for pH = 4.9 (no values for Hg and Pb, use pH = 6.8)	0.995	0.638	1.012	0.939	1.000
Soil-water partition factors for pH = 8.0 (no values for Hg and Pb, use pH = 6.8)	1.003	1.734	0.995	1.466	1.000
$d_{wc} = 3$ m (3 × base case)	0.998	0.997	0.998	0.997	0.998
$c_{bs} = 1$ kg/L (10 times base case)	1.000	1.000	1.000	1.000	1.000
$c_{sussol} = 0.1\ kg_{soil}/m^3_{wat}$ (10 times base case)	1.000	1.000	1.000	1.000	1.000
Yield per planted area $Y_p = 3 \times$ base case	0.427	0.745	0.424	0.507	0.618
Soil-plant bioconcentration factors = 0.333 × base case	0.983	0.692	0.992	0.960	0.852
Soil-plant bioconcentration factors = 3 × base case	1.051	1.923	1.023	1.120	1.445
Biotransfer factors for meat = 0.333 × base case	0.968	0.996	0.868	0.856	0.983
Biotransfer factors for meat = 3 × base case	1.096	1.012	1.397	1.431	1.050
Biotransfer factors for milk = 0.333 × base case	0.639	0.999	0.863	0.911	0.957
Biotransfer factors for milk = 3 × base case	2.083	1.002	1.411	1.267	1.130
Bioconcentration factors for fish = 0.333 × base case	0.982	0.954	0.997	0.947	0.982
Bioconcentration factors for fish = 3 × base case	1.055	1.139	1.009	1.158	1.054
$t_{cut} = 30$ yr	0.998	0.806	0.999	0.980	0.826
$t_{cut} = \infty$	1.000	1.039	1.000	1.003	1.706

REFERENCES

Boudala, F. S., Folkins, I., Beauchamp, S., Tordon, R., Neima, J., & Johnson, B. (2000). Mercury flux measurements over air and water in Kejimkujik National Park, Nova Scotia. *Water, Air and Soil Pollution*, *122*, 183–202.

Brunekreef, B. (1984). The relationship between air lead and blood lead in children: A critical review. *Science of the Total Environment*, *38*, 79–123.

Crawford, M., & Wilson, R. (1996). Low-dose linearity: The rule or the exception? *Human and Ecological Risk Assessment*, *2*, 305–330.

Curtiss, P. S., & Rabl, A. (1996). Impacts of air pollution: General relationships and site dependence. *Atmospheric Environment*, *30*, 3331–3347.

Daniels, M. J., Dominici, F., Samet, J. M., & Zeger, S. L. (2000). Estimating particulate matter-mortality dose-response curves and threshold levels: An analysis of daily time-series for the 20 largest US cities. *American Journal of Epidemiology*, *152*(5), 397–406. See also Comment in: *American Journal of Epidemiology*, *152*(5), 407–412.

EC. (1998). Directive 98/70/EC of the European Parliament and of the Council of 13 October 1998 relating to the Quality of Petrol and Diesel Fuels and Amending Council Directive 93/12/EEC.

EPA. (1998). *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Support Materials*. U.S. EPA Region 6 U.S. EPA Multimedia Planning and Permitting Division Office of Solid Waste, Center for Combustion Science and Engineering. Available at http://www.epa.gov/epaoswer/hazwaste/combust/riskvol.htm#volume1

EPAQS. (1998). *Expert Panel on Air Quality Standards; Lead*. Department of Transport, Environment and the Regions. HMSO; London.

EUSES. (1997). *EUSES the European Union System for the Evaluation of Substances*. Version 1.0. Joint Research Center, Ispra. European Commission.

ExternE. (1998). *ExternE: Externalities of Energy*. Vol. 7: *Methodology 1998 Update* (EUR, 19083); Vol. 8: *Global Warming* (EUR, 18836); Vol. 9: *Fuel Cycles for Emerging and End-Use Technologies, Transport and Waste* (EUR, 18887); Vol. 10: *National Implementation* (EUR, 18528). Published by European Commission, Directorate-General XII, Science Research and Development. Office for Official Publications of the European Communities, L-2920 Luxembourg. Results are also available at http://ExternE.jrc.es/publica.html.

GREENSENSE. (2004). European Commission, report in preparation.

Grosse, S. D., Matte, T. D., Schwartz, J., & Jackson, R. (2002). Economic gains resulting from the reduction in children's exposure to lead in the United States. *Environmental Health Perspectives*, *110*(6), 563–569.

IAEA. (1994). *Handbook of Parameter Values for the Prediction of Radionuclide Transfer in Temperate Environments*. Technical Report Series No. 364, produced in collaboration with the International Union of Union of Radioecologists, International Atomic Energy Agency, Vienna, Austria.

IAEA. (2001). *Generic Models for Use in Assessing the Impact of Discharges of Radioactive Substances to the Environment*. Safety Reports Series No. 19, International Atomic Energy Agency, Vienna, Austria.

Krewitt, W., Trukenmueller, A., Mayerhofer, P., & Friedrich, R. (1995). EcoSense—An Integrated Tool for Environmental Impact Analysis. In H. Kremers & W. Pillmann (Eds.), *Space and Time in Environmental Information Systems*. Marburg: Umwelt-Informatik aktuell, Band 7. Metropolis-Verlag.

Lanphear, B. P., Dietrich, K., Auinger, P., & Cox, C. (2000). Cognitive deficits associated with blood lead concentrations <10 μg/dL in US children and adolescents. *Public Health Reports*, *115*(6), 521–529.

Lutter, R. (2000). Getting the lead out cheaply: A review of EPA's proposed residential lead hazards standards. *Environmental Science and Policy*, *4*, 13–23.

Mackay, D. (2001). *Multimedia Environmental Models: The Fugacity Approach*, 2nd ed. Boca Raton, FL: Lewis Publishers.

McKone, T. E., & Enoch, K. G. (2002). *CalTOX^{TM}, A Multimedia Total Exposure Model*. Report LBNL-47399. Lawrence Berkeley National Laboratory, Berkeley, CA. Available at http://eetd.lbl.gov/ied/ERA.

Muir, T., & Zegarac, M. (2001). Societal costs of exposure to toxic substances: Economic and health costs of four case studies that are candidates for environmental causation. *Environmental Health Perspectives*, *109* (Supplement 6), 885–903.

Pennington, D., Crettaz, P., Tauxe, A., Rhomberg, L., Brand, K., & Jolliet, O. (2002). Assessing human health response in life cycle assessment using ED10s and DALYs: Part 2—Noncancer effects. *Risk Analysis*, *22* (5), 947–963.

Rabl, A. (1996). Discounting of long term costs: What would future generations prefer us to do? *Ecological Economics*, *17*, 137–145.

Rabl, A. (2003). *The Plausibility of Linear Dose-Response Functions for Air Pollution*. Ecole des Mines de Paris, 60 boul. St.-Michel, F-75272 Paris.

Rabl, A., & Spadaro, J. V. (1999). Environmental damages and costs: An analysis of uncertainties. *Environment International*, *25*, 29–46 (1999)

Schwartz, J. (1994). Low-level lead exposure and children's IQ: A meta-analysis and search for a threshold. *Environmental Research*, *65*, 42–55.

Seinfeld, J. H., & Pandis, S. N. (1998). *Atmospheric Chemistry and Physics: From Air Pollution to Climate Change*. New York: Wiley.

Spadaro, J. V. (1999). Quantifying the effects of airborne pollution: Impact models, sensitivity analyses and applications. Doctoral thesis, Ecole des Mines, Paris, France.

Spadaro, J. V., & Rabl, A. (2002). Assessing the health impacts due to airborne emissions: The airpacts model. Probability Safety Assessment and Management Conference, San Juan, Puerto Rico, June 23–28.

Sullivan, T. M., Lipfert, F. W., Morris, S. C., & Moskowitz, P. D. 2003. *Potential Health Risk Reduction Arising from Reduced Mercury Emissions from Coal-Fired Power Plants*. Report for United States Department of Energy, September 2001. Energy, Environment and National Security Directorate. Brookhaven National Laboratory, Upton, New York 11973–5000.

WHO 1988–2001. WHO (1988). *Chromium*. Environmental Health Criteria 61.

WHO. (1990). *Methyl Mercury*. Environmental Health Criteria 101.

WHO. (1991). *Inorganic Mercury*. Environmental Health Criteria 118.

WHO. (1991). *Nickel*. Environmental Health Criteria 108.

WHO. (1992). *Cadmium*. Environmental Health Criteria 134.

WHO. (1995). *Inorganic Lead*. Environmental Health Criteria 165.

WHO. (2001). *Arsenic and Arsenic Compounds*. Environmental Health Criteria 224. World Health Organization, Geneva, Switzerland.

WRI. (1994). *World Resources: A Guide to the Global Environment*. Oxford: World Resources Institute, Oxford University Press.