Costs of Health Damage from Atmospheric Emissions of Toxic Metals. Part 1: Methods and Results

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Abstract

Significant quantities of toxic metals are emitted to the air by the incineration of waste, as well as by the combustion of coal and oil. To optimize the regulations for their emissions one needs to know the cost of their damage. For that one needs to carry out an impact pathway analysis, with realistic dispersion models, exposure-response functions and monetary values. In this paper we explain the method and assumptions and present results for arsenic, cadmium, mercury and lead, the most important toxic metals in terms of damage cost. We also estimate their contribution to the damage cost of waste incineration and electric power from coal for typical situations in Europe. The damage costs of these metals are much higher than previous estimates by the ExternE projects, because in recent years a large number of epidemiological studies have been published for these metals and they imply more and more serious health effects than what had been known before. New cost-benefit studies for the abatement of toxic metal emissions are advisable. The discussion of the epidemiological studies and the choice of exposure-response functions are presented in two companion papers, one for As and Cd, the other for Hg and Pb.

Key words: toxic metals, arsenic, cadmium, lead, mercury, damage cost, external cost, exposure-response functions

1. Introduction

Toxic metals are emitted to the air by several industrial sources, especially the incineration of waste and the combustion of coal and oil. Even though stringent new regulations for waste management in the EU have brought about impressive improvements, the remaining emissions are still significant in terms of damage costs, as this series of papers shows.

To optimize the regulations for their emissions one needs to know both the cost of their damage, also known as external costs, and the cost of actions to reduce the emissions. Here we provide estimates of the damage cost per kg of emitted pollutant, for typical conditions in

Europe. Because of the length of the documentation and calculations we split the presentation in three parts. In the first we explain the method and assumptions and summarize the results; as illustration we also look at implications for waste incineration and for electricity production from coal. Detailed derivation of the results can be found in parts 2 and 3, respectively for As and Cd, and for Hg and Pb. The focus on these metals has been motivated by the prioritization study of Nedellec, Lapkoff and Rabl [2012], who ranked Arsenic (As), mercury (Hg), cadmium (Cd), nickel (Ni), manganese (Mn), chromium (Cr) and lead (Pb) among the 12 most worrisome pollutants emitted by waste treatment facilities, taking into account both emitted quantities and general toxicological criteria. Whereas for As, Cd, Hg and Pb there are sufficient epidemiological studies, for Cr and Ni only toxicological studies are available for the most worrisome health endpoints and the methodology is a bit different, to be addressed in another publication. The spreadsheet with the calculations can be downloaded from http://www.arirabl.org/software/ [1].

The rationale for damage costs is very different from the current practice of much risk assessment which looks at upper bounds of the 95% confidence intervals rather than expectation values, in order to determine exposure limits that entail negligible risk for the population. Damage costs have to be calculated as expectation value (together with an estimate of the confidence intervals) rather than as upper limit of the confidence intervals.

It is crucial to carry out an impact pathway analysis (IPA), with realistic dispersion models, exposure-response functions (ERF) [2] and monetary values. The principal steps of an IPA can be grouped as follows:

- Emission: specification of the pollution source (e. g. emission of As by an incinerator with such and such stack height at such and such location);
- Dispersion: calculation of increased pollutant exposure in all affected regions;
- Impact: calculation of the impacts (damage in physical units) due to the increased exposure, using ERFs (e. g. cases of cancer due to ingestion of As);
- Cost: monetary valuation of these impacts (e. g. multiplication by the cost of a case of cancer).

The methodology for IPA has been developed by the ExternE project series in the EU [ExternE 2005, 2008] and by analogous projects by EPA [Abt 2004] and National Research Council in the USA [NRC 2010]. For a general review and summary of results see Rabl, Spadaro and Holland [2014]. The focus of these projects has been on greenhouse gases and the classical air pollutants (PM, NO_x, SO_2, etc) that are emitted in large quantities, with little attention given to toxic metals, some of which can impose large damage costs despite the small emitted quantities.

Conventional LCA (life cycle assessment) methods do not use an IPA; most of their impact indicators have little relation with real impacts. This has been shown by Vad [2008] who compared the health impacts between two LCIA methods (Impact2002+ and CML–IA) and the IPA of ExternE, in terms of DALY per kg of pollutant: for most pollutants the results differ by several orders of magnitude between each of these three methods.

[1] Because of rounding, the multiplication of the some of the numbers in this paper differs slightly from the stated results.

[2] The term dose-response function is also widely used, but we prefer ERF because exposure is a more general term that can refer equally well to ambient concentrations and to intake or absorbed dose.

We estimate the incremental exposures due to incremental emissions by using the intake fractions (IF) calculated by Spadaro and Rabl [2004], i.e. the fraction of the emitted pollutant that ends up entering a human body.

We focus on health impacts because experience of external cost assessments in the USA and Europe has found health impacts of pollutants (other than greenhouse gases) to impose much higher costs than other impacts such as ecosystem impacts. The reader may wonder if this is merely due to the lack of understanding of ecosystems compared to health impacts. But we find the smallness of ecosystem damage costs entirely plausible because of the monetary valuation is based on people's willingness-to-pay to avoid a loss [3]. Human health impacts are valued at the level of individuals, ecosystem impacts at the level of species or entire ecosystems. For example, even a slight increase in cancer incidence for humans is of great concern, but few would pay to avoid an analogous increase among animals (which anyway has negligible impact on species survival as it occurs mostly after reproduction).

The ERF is a central ingredient in the assessment of health impacts. The regulatory approach developed by agencies such as EPA and WHO distinguishes cancers due to genotoxic carcinogens from other endpoints. Whereas the ERFs for genotoxic carcinogens are assumed to be linear without threshold, for other endpoints only thresholds are considered in order to answer the question whether or not there is a significant risk for the population (i.e. whether the exposure is above or below the threshold) [4]. As discussed in a report by the National Research Council [NRC 2009], such dichotomy poses problems because thresholds are not sufficient for quantifying the impacts if there is exposure above the threshold. That report recommends developing explicit ERFs for non-cancer endpoints. That is what we do for toxic metals in the present series of papers.

Much progress has been made with regard to the epidemiology of toxic metals, especially in recent years. In the past most studies of toxic metals were based on relatively small cohorts of industrial workers whose exposures were extremely uncertain, with results that may not be representative of the general population because industries tend to recruit workers in better than average shape. More recently improvements of epidemiological studies have, among other topics, focused on exposure characterization, yielding results that are far more reliable. Moreover, numerous national biomonitoring surveys, such as NHANES (National Health and Nutrition Examination Survey), provide detailed data of biomarkers of pollutants for thousands of individuals. Here we use such data for determining the fraction of the incremental collective exposure that is above threshold.

An analysis of this type involves numerous choices for the interpretation of studies and the selection of parameters. Whereas readers may well disagree with our choices, we offer a framework where it is easy to modify the parameters for alternative calculations, and we have put the spreadsheet with the calculations on the website www.arirabl.org.

[3] thus it includes the cost of pain and suffering.

[4] But the practice is evolving, for instance the EU's REACH Regulation goes beyond only considering thresholds for non-genotoxic endpoints.

2. Material and Methods

2.1. Intake fractions

The damage depends on the site of the emitting source (especially the population density of the surrounding population, but also the meteorological and geographic conditions) and the height of the stack. Even though this variation of the damage is very strong for the inhalation impacts of primary air pollutants, one can present typical results for power plants and incinerators because they have tall stacks (which reduces the importance of the local impacts) and they tend to be in zones that do not have the highest population densities. Note that for most policy applications one needs typical rather than site-specific results.

Furthermore, for atmospheric emission of toxic metals the dose due to ingestion is about two orders of magnitude larger than the inhalation dose. Since the ingestion dose involves the production and transport of food over hundreds of km, the variation with emission site is reduced so much that one can calculate the exposure based on regional average values of population density, food intake and environmental parameters (such as meteorology, soil characteristics and types of agricultural production).

Here we use the intake fractions (IF) of Spadaro and Rabl for typical conditions in Europe, listed in Table 1. Their calculations are based on the multimedia model of EPA [1998] and can be downloaded from www.arirabl.org. For other regions they have to be scaled in proportion to the regional population density (within a radius of about one thousand km). These intake fractions are the collective dose in mg per emitted kg, without regard to the fraction that is actually absorbed by the body. They are based on an average respiration rate of 20.6 m^3/day, a water intake of 0.6 m^3/yr and a typical European diet. The IF from water should be reduced to the extent that water treatment plants filter out part of the metals. In Table 1 we do not show Hg because it requires a very different pathway analysis, with dispersion over the entire hemisphere, by far the most serious impacts being due to methyl-Hg in fish, see our companion paper on Hg and Pb.

Table 1. Intake fractions (IF) for typical conditions in Europe, in mg per emitted kg. From Spadaro and Rabl [2004].

Pathway	**As**	**Cd**	**Cr VI**	**Ni**	**Pb** [a]
Inhalation	3.9	3.9	3.9	3.9	3.9
Water	31.1	31.7	31.0	31.5	35.5
Cattle milk	156.2	0.3	38.0	27.2	10.8
Cattle meat	13.8	1.4	36.8	43.9	4.1
Freshwater fish	15.6	31.7	1.6	31.6	9.0
Grains	60.8	119.5	60.4	64.4	80.4
Root vegetables	12.4	24.1	12.0	13.1	16.0
Green vegetables	16.2	47.5	15.9	17.7	24.0
TOTAL	**310**	**260**	**200**	**233**	**184**

[a] values for Pb have been recalculated assuming the same deposition velocity of PM_{10} as for the other metals (instead of the one for $PM_{2.5}$ in the original paper).

2.2. Exposure-response functions (ERF)

To identify the relevant ERFs we have carried out an extensive literature review, beginning with general toxicological profiles by World Health Organization (WHO), EPA and Agency for Toxic Substances and Disease Registry (ATSDR). Then we searched Medline PubMed with the criteria:

(effect OR effects) AND health AND "name of metal",

going back to 1990. We decided to use mainly epidemiological studies for the determination of ERFs, relying on toxicological studies for supplemental information about mechanisms of action and the question of thresholds. The reason for this choice is that for the most important impacts there is now a sufficient body of epidemiological studies and they allow a more reliable impact assessment than the extrapolation of toxicological studies to humans.

In selecting the studies for deriving an ERF, we give preference to: prospective cohort studies, general population studies, individual exposure measurement, and low exposure levels. Rationales for the selected studies are detailed in the companion papers. And of course a key consideration is whether there is sufficient evidence from the totality of the literature for the causality of a specific end point due to a specific metal.

Most epidemiological studies of pollution provide their results in the form of relative risk (RR) for the occurrence of an endpoint as a function of an exposure indicator, relative to a reference level. To construct an ERF one also needs to know the reference incidence rate in the population. We find it convenient to state incidence rates in units of cases per year per average person. Thus we obtain the ERF slope (s_{ERF}) as

$$s_{ERF} = \text{incidence rate} * \Delta RR/\Delta E, \text{ with } \Delta RR = RR - 1 \quad (1)$$

where ΔRR is the RR increase for an increase ΔE of the exposure. Since we state the exposure E as intake rate in units of (mg/yr), the units of s_{ERF} are (cases/yr)/(mg/yr) or simply cases/mg.

Typically the epidemiological studies report the RR for effects observed after exposure during a specific period in time. The exact relation between time and duration of exposure and the resulting effect is difficult to establish: usually the data are insufficient for firm conclusions. For damage cost calculations it is most reasonable to assume stationary conditions, by interpreting the effect rate measured in a study as being due to constant exposure at the measured level. The damage cost is based on lifetime impact due to lifetime exposure. For the calculations in our spreadsheet we find it convenient to prorate exposures and impacts to one year.

For some endpoints additional data are required. For studies that are based on biomarkers (urine or blood concentration of the pollutant or their metabolites) rather than exposure or intake of the pollutant one has to estimate a conversion factor from biomarker to exposure or intake.

For deaths due to specific identifiable causes, such as cancer deaths, it is sufficient to use the corresponding reference mortality rate. But many studies of mortality due to pollution consider a change in all-cause mortality, without identifying specific causes. Typically such studies report RR as change in the age-specific mortality rate $\mu(x)$, defined as the fraction of a cohort between the age x and x + 1 year who have died during the year. As explained by Rabl [2003] it makes no sense to calculate a corresponding number of deaths for all-cause

mortality [5]; only loss of life expectancy (LE) can be calculated unambiguously. For such mortality we use life table data of France to calculate the loss of life expectancy (LE) corresponding to the RR observed in an epidemiological study. The French data for 2005 are representative of much of the EU and have the additional advantage of including detailed annual data for all ages up to age 104, unlike most other databases that stop around age 85. We find the following relation between LE loss and increase in RR, per year of exposure

$$\Delta LE/\Delta RR = 0.148 \text{ YOLL/yr per } \Delta RR, \qquad (2)$$

where YOLL (years of life lost) is used as unit to designate the LE loss. For the calculation of this number we have assumed an increase $\Delta RR = 0.1$ that affects all ages above 10 (the latter chosen to account for a lag between exposure and effect), and we have divided the lifetime change by an effective exposure duration of 70 yr. The number varies somewhat with ΔRR, from 0.141 at $\Delta RR = 0.20$ to 0.151 at $\Delta RR = 0.05$, but Eq.2 is a good compromise because a ΔRR of 0.1 is fairly representative of policy choices.

A crucial and controversial issue is the form of the ERF at low exposures, in particular whether there is a no-effect threshold. The simplest form is a straight line through the origin. Such linearity without threshold is also by far the most convenient for the calculation of damage costs. With linearity the calculations are conceptually clear and numerically simple. It does not matter how the intake fraction or collective dose is distributed among the population and the total damage is equal to the damage of an average person receiving the entire intake fraction.

Linearity without threshold is the appropriate form on theoretical grounds when the effect of a pollutant occurs through a mechanism that is also active without the pollutant. In that case there is, so-to-speak, an underlying ERF associated with that mechanism for which the entire population is above threshold. The effect of an incremental exposure is additive to the background occurrence, and continuity implies proportionality between a small incremental exposure and its effect. Such is the case for genotoxic carcinogens since DNA damage is an ever present background process. In view of the available evidence linearity without threshold is also most plausible for particulate air pollution and for the neurotoxic effects of Hg and Pb. But it is not a general rule, as shown by the "mega-mouse experiment" of Frith, Littlefield and Umholtz [1981].

For the present paper we find it most appropriate to begin with the assumption of linearity without threshold, as an upper bound for the impacts. Then we calculate how the numbers would change in the presence of a threshold, assuming a hockey stick for the form of the ERF (even though in reality population-level ERFs do not have a sharp threshold because of individual differences in sensitivity to pollution). We base the thresholds on limit values recommended by organizations such as EPA for maximum safe exposure.

[5] Multiplying ΔRR $\mu(x)$ by the cohort size seems natural but it is wrong because it does not account for induced changes in the size of age cohorts in subsequent years: everybody dies exactly once, regardless of any change in RR; see Rabl [2003].

2.3. Calculation of damage costs

With the assumption of stationary conditions one can calculate a constant impact rate ΔI, e.g. the rate of the health endpoint per year, corresponding to a constant emission rate ΔQ, e.g. 1 kg/yr, and a constant collective intake rate ΔE in mg/yr. In the absence of a threshold the impact rate is

$$\Delta I = s_{ERF}\, \Delta E \qquad (3)$$

and has units of cases/yr. Dividing the impact rate by the emission rate one obtains the impact per emitted quantity, for instance the cancer deaths per kg. Since the intake fraction IF is the collective intake per kg of emitted pollutant, in units of mg/kg, the intake rate ΔE can be replaced by

$$\Delta E = IF\, \Delta Q\ . \qquad (4)$$

Multiplication of the impact rate by the cost P per case for the endpoint in question yields the damage cost rate, in units of €/yr,

$$\text{damage cost rate} = P\, s_{ERF}\, IF\, \Delta Q\ . \qquad (5)$$

Dividing by the emission rate we obtain the damage cost D in € per kg as

$$D = P\, s_{ERF}\, IF \qquad (6)$$

where

s_{ERF} = ERF slope, in units of cases/mg, and
P = unit cost ("price"), in units of €/case,
IF = intake fraction = collective dose per kg of emitted pollutant, in units of mg/kg.

Because the time base for our calculations is 1 yr, we find it convenient to multiply the customary intake rates by 0.36525 to convert them from μg/day to mg/yr.

Some ERFs are stated in terms of concentrations in ambient air or drinking water rather than intake. In particular, the ERFs of the IRIS website of EPA for cancers due to inhalation (called unit risk factors) are stated as lifetime cancer risk per concentration in the ambient air and based on a lifetime exposure of 70 years. The inhalation unit risk factors can be expressed as equivalent s_{ERF} with units of (cases/mg) for use in Eq.4 with the inhalation IF, if one takes the inhalation rate of 20.6 m^3/day assumed by Spadaro and Rabl [2004] to obtain the inhalation dose in (mg/yr)/(μg/m^3). However, we also note that the unit risk factors are the upper bound of the 95% confidence intervals rather than expectation values; therefore one also has to multiply by the ratio central value/upper bound. Thus we obtain the ERF slope in units of (cancers/yr)/(mg/yr), i.e. cases per mg of intake,

$$\begin{aligned} s_{ERF} &= (1/70) * \text{Unit Risk}/(20.6*365.25/1000\ (\text{mg/yr})/(\mu\text{g/m}^3)) \\ &\quad * (\text{central value/upper bound}) \\ &= (\text{Unit Risk/yr}) * 0.0019 * (\text{central value/upper bound})\ (\mu\text{g/m}^3)/(\text{mg/yr}) \end{aligned} \qquad (7)$$

where Unit Risk = cancers per lifetime per (μg/m^3). Unfortunately the IRIS websites do not provide any information for the ratios of central value/upper bound.

If there is a lag of n years between exposure and impact, we further multiply by a factor $(1 + r_{dis})^{-n}$ where r_{dis} is the social discount rate (for which we take 4% [6]). For the development of cancers we assume a lag of 20 yr; that reduces their cost by a discount factor of 0.46. Even though lags are different for different cancers, 20 yr is a reasonable choice for the cancers in these papers, in particular the dominant type, lung cancers. For general mortality, chronic bronchitis, diabetes, osteoporosis and anemia we assume a lag of 10 yr. We assume that discounting is negligible for neurotoxic impacts because most of the damage occurs during the very first years of life and the assumed cost per IQ point has been calculated as discounted total loss of lifetime earnings at the time of birth. The total cost is the sum over all endpoints.

The monetary valuation is based on the willingness-to-pay (WTP) to avoid a loss; thus it includes not only market costs but most importantly the cost of pain and suffering. We use the unit costs listed in Table 2, as explained in the following (with slight rounding of the numbers). The most important values are for mortality, in particular the value of a prevented fatality (VPF) [7],for which ExternE [2008] takes 1.5 million €$_{2008}$. However, that appears far too low in view of OECD [2011, section 4.1] which recommends a value of $\$_{2005}$ 3.5 million for applications in the EU27, based on the meta-analysis by Lindhjem et al [2011] of all available stated preference surveys [8]. Converting at an exchange rate of 1.30 \$/€ and adjusting for inflation we obtain 3.0 million €$_{2010}$. VPF is based mostly on fatal accidents and here we use it as basis for identifiable causes of death with major loss of life expectancy, namely infant mortality and cancers. For fatal cancers we take 3.30 million €$_{2010}$. For infant deaths we multiply VPF by 1.75 to obtain 5.25 million €$_{2010}$, because parents care so much about their children and values in the range of 1.5 to 2 * VPF have been recommended [OECD 2011, Section 5.2].

Many epidemiological studies report increases in age-specific mortality without being able to identify causes of death. For such studies it is appropriate to evaluate the loss of life expectancy (LE), using the value of a life year lost (VOLY) rather than VPF. As explained by Rabl [2003] and Rabl, Spadaro and Holland [2014], there are several reasons for this choice, especially that only LE loss can be calculated without ambiguity, but not a number of deaths, and that the LE loss per death is much less than for typical accidents. Unlike VPF, only few studies have tried to measure VOLY directly. An indirect approach is to assume that VPF is the sum of discounted VOLY over the years that are lost in typical accidents, implying a VPF/VOLY ratio in the range of 20 to 40. ExternE [2008] used a VOLY of 40,000 €$_{2008}$/YOLL, based on a contingent valuation study that was finally published by Desaigues et al [2011]. That is at the low end, compared to Chanel and Luchini [2014] who find 140,000 €. In view of that situation we keep the ratio VPF/VOLY = 37.5 of ExternE [2008] and obtain, after slight adjustment for inflation, a VOLY of 84,000 €$_{2010}$/YOLL.

For neurotoxic effects we take as proxy a value of $\$_{2008}$18,000 per IQ point lost, following Spadaro and Rabl [2008a] who reviewed studies of the relation between IQ and lifetime

[6] See Table 9.1 of Rabl, Spadaro and Holland (2014) which summarizes a review of social discount rates. There has been a tendency to reduce the discount rates used by governments; now 2 to 3% by USEPA, 6% by EU, 4% by France.

[7] often designated by the unfortunate term "Value of Statistical Life" (VSL) which tends to evoke negative reactions among non-economists. It is not the value of life, whatever that may be, but the willingness-to-pay to avoid an anonymous premature death.

[8] The alternative valuation method of wage-risk studies (favored in the US) tends to yield somewhat higher VPF numbers.

earnings in the US (there are no analogous studies in the EU where IQ is not routinely measured). Converting at an exchange rate of 1.30 $/€ and adjusting for inflation this yields 14,500 €$_{2010}$ per IQ point. One of the endpoints for arsenic is chronic bronchitis for which ExternE [2008] uses 200,000 €$_{2008}$ per case; that is based on two contingent valuation studies in the US as well as a valuation using QALY weights [Desaigues et al. 2007]. For non-fatal cancers we use INC [2007]. The numbers in this study imply that on average the treatment cost is 38,879 €$_{2007}$ and the productivity loss 60,432 €$_{2007}$. The total market cost amounts to 106,511 €$_{2010}$. Following ExternE we multiply by 1.5 to account for pain and suffering to obtain the unit cost of non-fatal cansers as 159,767 €$_{2010}$.

For additional endpoints we use DALY or QALY weights if available [Salomon et al 2012], assuming that the value of a DALY or QALY is equal to a VOLY.

Table 2. The unit costs P for health endpoints assumed in this paper.

End point	**This paper, €$_{2010}$**
VOLY (value of a life year), €/YOLL	84,000
VPF (value of prevented fatality)	3,000,000
Infant death = VPF * 1.75	5,250,000
Fatal cancer = VPF	3,000,000
Non-fatal cancer	159,767
Chronic bronchitis (CB)	210,000
Value of IQpoint	14,500
Value of a DALY or QALY = VOLY	84,000

2.4. Accounting for thresholds

For cancers due to genotoxic carcinogens we follow the standard assumption that the ERF is linear without threshold at low doses. We also assume that there is no threshold for the impacts of Pb, a view that seems generally accepted in view of the fact that the ERF for neurotoxic effects has been measured down to very low exposures and found to be without threshold, and probably even above a straight line.

To find the threshold for all other endpoints, we use guideline values for maximum safe intake that have been established by organizations such as WHO or EPA for the protection of human health. However, we emphasize that such values have been established to ensure that nobody is exposed to significant risk, and they are not necessarily real thresholds. Even if the ERF were a hockey stick, the threshold would be difficult to determine because of all the uncertainties, especially for thresholds based on toxicology and the extrapolation from animals to humans. The hockey stick with maximum safe intake as threshold is an approximation but it is difficult to do better in view of the available information.

To account for a threshold it may seem natural to multiply the no-threshold result by the fraction of the population whose current exposure is above threshold. However, even with the assumption of a hockey stick, the fraction of the population above threshold is not what matters; rather, one has to determine the fraction of the total incremental exposure that is above threshold. That requires an analysis of the distribution of incremental exposures, as shown by Spadaro and Rabl [2008a] who carried out such a calculation for the damage cost of Hg. The fraction f_{thr} of the total incremental exposure that is above threshold is significantly

larger than the fraction of the population above threshold. In the case of Hg we follow Spadaro and Rabl who take a threshold = 6.7E-03 mg_{Hg}/kg_{body}/day [EPA 2001] and find that f_{thr} is 0.44, about 5.5 times larger than the fraction of the population. The following lines explain the calculation of f_{thr}.

Without a threshold the total impact rate due to a total collective exposure E is

$$I = s_{ERF}\, E \tag{8}$$

The collective exposure E is the sum of the individual exposures e_i over the entire population. If there is a threshold e_{thr} the total impact rate is

$$I_{thr} = s_{ERF} \sum_{i=p_{thr}}^{p} (e_i - e_{thr}) = s_{ERF} \sum_{i=p_{thr}}^{p} e_i - s_{ERF}\,(p - p_{thr})\, e_{thr} \tag{9}$$

where the individual exposures e_i are in increasing order and p_{thr}/p is the fraction of the affected population that is above threshold. If an incremental emission rate ΔQ increases the exposures by Δe_i the impact rate increases by

$$\Delta I_{thr} = s_{ERF} \sum_{i=p_{thr}}^{p} \Delta e_i \tag{10}$$

Let us assume, like Spadaro and Rabl, that the distribution of the incremental exposures Δe_i due to atmospheric emissions is like that of the e_i

$$\Delta e_i = e_i \;\; \Delta E/E \quad . \tag{11}$$

Whereas that assumption is realistic for Hg because most Hg is first emitted to the air (natural emissions being mostly volcanic), the distributions of Δe_i and e_i can be different for pollutants such as As because part of the total exposure comes from geological As that enters directly into the food chain. Unfortunately no information is available on the distribution of the Δe_i, and so we use Eq.11 with $\Delta E = IF\, \Delta Q$ to obtain the impact rate increase

$$\Delta I_{thr} = s_{ERF}\, IF\, \Delta Q \sum_{i=p_{thr}}^{p} e_i/E \quad \text{where } E = \sum_{i=0}^{p} e_i \quad . \tag{12}$$

As with the passage from Eq.3 to Eq.6 we thus obtain the damage cost D_{thr} for the threshold case

$$D_{thr} = P\, s_{ERF}\, IF\, f_{thr} \tag{13}$$

where

$$f_{thr} = \sum_{i=p_{thr}}^{p} e_i / \sum_{i=0}^{p} e_i \tag{14}$$

is the fraction of the incremental collective exposure that is above threshold.

For As, Cd and Cr we estimate the fraction f_{thr} on the basis of a recent survey of exposures in France where urinary concentrations of the toxic metals have been measured for a representative sample of the population [InVS 2011]. To relate the urinary concentrations to intake, we use conversion factors that we have found in the literature. The exposure

distributions are very close to lognormal and the authors indicate the geometric mean μ_g. Therefore we choose the geometric standard deviation σ_g such that the resulting distribution is a good match for the data; σ_g turns out to be close to 2. Knowing the probability distribution it is straightforward to evaluate f_{thr} of Eq.14.

3. Uncertainty

The calculation of damage costs involves many assumptions and model parameters that are more or less uncertain. A rigorous uncertainty analysis would involve a detailed examination of each element of the calculation to estimate its probability distribution. Then the probability distribution of the result should be determined by a Monte Carlo calculation. That would be a major undertaking, way beyond the scope of this paper. But as a start we list in Table 3 the main elements of the impact pathway analysis with a qualitative indication of their uncertainties.

In particular the calculation of the intake fraction from ingestion is so complex that no Monte Carlo analysis has been published as far as we have been able to ascertain. The closest to an uncertainty assessment of intake fractions that we have been able to find are Spadaro and Rabl [2004] and Huijbregts et al [2004]. Spadaro and Rabl compare their results (which are based on the model of EPA [1998]) with the CalTox model [McKone and Enoch 2002] and find agreement within a factor of about 2 for As, Cr and Ni, and within a factor of 20 for Cd and Pb. Spadaro and Rabl also carry out a sensitivity analysis by varying the most important input parameters over a wide range (threefold for most): the corresponding change of the intake fraction is a factor of at most two. Huijbregts et al compare a large set of intake fractions calculated by CalTox with those calculated by USES-LCA; they find that on average the agreement can be characterized as a geometric standard deviation of about six to eight (the square root of their uncertainty factors which are for 95% confidence intervals).

Here we use, as a simple alternative to a detailed Monte Carlo calculation, the approach of Spadaro and Rabl [2008b] and Rabl, Spadaro and Holland [2014] who have shown that the uncertainty of damage costs can be estimated in terms of lognormal distributions and geometric standard deviations. The lognormal distribution is appropriate because the calculation essentially a product of factors. In practice the distribution of a product is approximately lognormal even if the number of factors is small, provided the distributions of the most uncertain factors are themselves not too far from lognormal. Spadaro and Rabl [2008b] have found that to be the case for damage costs.

Table 3. The main elements of the impact pathway analysis (IPA) and main sources of uncertainty. Not explicitly listed is the role of choices by the analyst when the available information is not sufficient (being incomplete, only indirectly relevant, ambiguous, contradictory or too difficult to find). The magnitude of the uncertainties can be very different from case to case.

Step of IPA	**Element**	**Main Uncertainties**
Emission	for calculation per kg of pollutant for calculation per unit product (kWh, tonne waste etc)	None. emission per unit product can be quite uncertain.
Dispersion (calculation of exposure or IF)	Exposure from inhalation Exposure from ingestion	Modeling of atmospheric dispersion, wet and dry deposition, and for reactive pollutants their chemical transformations. Modeling the pathways of the pollutants into soil, water and the food chain. Delays between emission and exposures.
Impact (ERF)	RR Other factors needed for ERF Extrapolation to lower exposures	Confidence intervals as reported. Possible biases of the epidemiological studies because of their design. Background rates Exposure conversion factors Extrapolation from study population to population of concern. Form of ERF (linearity, threshold).
Cost (Monetary valuation)	Studies of willingness-to-pay (WTP) to avoid a loss.	Confidence intervals as reported. Variability between studies
	Linking impact to cost estimates	Relation between endpoint in epidemiological study and endpoint for which there are cost data.
	Discounting of impacts that occur in the future	Time of onset, discount rate, severity of impacts (note medical progress).
	Benefit transfer	Extrapolation from population of WTP study to population of concern.

This finding has a simple interpretation: if the damage cost has a lognormal distribution with a geometric mean μ_g and a geometric standard deviation σ_g, the probability is approximately 68% for the true cost to be in the interval $[\mu_g/\sigma_g, \mu_g \sigma_g]$ and 95% for it to be in the interval $[\mu_g/\sigma_g^2, \mu_g \sigma_g^2]$, in other words

$$\text{the 68\% CI is } [\mu_g/\sigma_g, \mu_g \sigma_g] \quad \text{and} \quad \text{the 95\% CI is } [\mu_g/\sigma_g^2, \mu_g \sigma_g^2] \quad . \qquad (6)$$

The geometric standard deviation σ_g of the product $z = x_1 x_2 x_3 \ldots x_n$ of uncorrelated factors x_i is given by

$$[\ln(\sigma_g)]^2 = [\ln(\sigma_{g,1})]^2 + [\ln(\sigma_{g,2})]^2 + ... + [\ln(\sigma_{g,n})]^2 \quad . \tag{7}$$

where $\sigma_{g,i}$ is the geometric standard deviation of x_i. Thus it suffices to estimate the $\sigma_{g,i}$ for each of the factors of the damage cost calculation. For factors for which the confidence intervals are approximately symmetric, in particular the RR of most epidemiological studies, an equivalent geometric standard deviation can be estimated by the equation

$$\sigma_g = \sqrt{\frac{\mu+\sigma}{\mu-\sigma}} \quad . \tag{8}$$

where μ is the ordinary mean and σ the ordinary standard deviation.

Thus it suffices to estimate the geometric standard deviations for each of the key factors of the damage cost calculation, namely intake fraction, exposure-response function (ERF), and monetary valuation. For the ERF we consider two contributions: the relative risk RR of a health effect and additional factors such as the relation between biomarker and intake. We estimate the σ_g of ERF and monetary valuation by looking at the endpoints with the highest damage cost. Even though such an approach is admittedly quite rough, it is sufficient for practical purposes because the utilization of an environmental cost-benefit analysis is not sensitive to the precise value of the uncertainty: for example the choice of a decision maker is unlikely to be different if σ_g is 3.5 or 4.5.

For the intake fraction we argue that the results of Huijbregts et al are far too pessimistic. The environmental pathway modeling of atmospheric emissions has to begin with the dispersion in the atmosphere, and that step determines the inhalation intake fraction. Comparing the inhalation intake fraction results of CalTox and USES-LCA, Huijbregts et al find that the disagreement corresponds to geometric standard deviations larger than 30. We believe, for the following reasons, that such enormous disagreement stems from unrealistic treatment of atmospheric dispersion by one or both of these models.

Long term inhalation exposure due to primary pollutants (including metals) can be calculated with geometric standard deviations of 2 or better, as shown by numerous validation studies, including the Monte Carlo analysis of Spadaro and Rabl [2008b] and comparisons between calculated and measured concentrations [e.g. Rabl, Spadaro, Holland 2014]. Furthermore the ratio of ingestion dose and inhalation dose of metals has generally been observed to be in the range of several tens to several hundreds (see e.g. the reports by WHO and ATSDR [9]). Thus the ratios of ingestion over inhalation intake of Spadaro and Rabl [2004], about 50 to 80 in Table 1, are very plausible. Exposure distributions are approximately lognormal, and the ratios of lognormal quantities are also lognormal. Taking simple numbers for the sake of illustration, if 68% of the ratios ingestion/inhalation are between 30 and 300, σ_g for that ratio is the square root of 300/30 = 3.16..., and if 95% of the ratios ingestion/inhalation are between 30 and 300, σ_g for that ratio is the fourth root of 300/30 = 1.778.... That suggests that the ratio ingestion/inhalation for typical conditions can be estimated within a factor of about 2 to 3 even without calculation. We also note that the sensitivity analysis in Table A5 of Spadaro and Rabl [2004] indicates an uncertainty range of about 0.4 to 2, very much smaller than

[9] Of course the ratios implied by measured data have to be interpreted with caution because they may not correspond to steady state conditions and part of the ingestion dose may be due to metals of natural origin in soil or ground water.

implied by Huijbregts et al. If, for the sake of illustration, σ_g is 2 for the inhalation dose and 2 (or alternatively 2.5) for the ratio ingestion/inhalation, Eq.7 implies that σ_g for ingestion is 2.7 (or alternatively 3.2). In view of these considerations we set σ_g for intake equal to 3.

Table 4 shows our choices for the $\sigma_{g,i}$ of the key factors of the damage cost calculation. For monetary valuation we take $\sigma_{g,i}$ to be 2, based on Spadaro and Rabl [2008b]. The last column of this table shows the contribution of the $[\ln(\sigma_{g,i})]^2$ to the total of Eq.7. Because of the quadratic combination of terms only the largest $\sigma_{g,i}$ make a significant contribution. The resulting geometric standard deviation σ_g of the damage cost is 4.1, which we round off to 4. We emphasize that these are typical estimates; for specific endpoints of specific metals the uncertainty can be somewhat different as the reader can test by changing the $\sigma_{g,i}$ in Eq.7. Additional uncertainty due to thresholds can be estimated by comparing the results with and without threshold in Table 5.

Table 4. Typical $\sigma_{g,i}$ of the key factors of the damage cost calculation.

	$\sigma_{g,i}$	$ln(\sigma_{g,i})^2$
Intake fraction IF	3	*1.21*
Relative risk RR	1.5	*0.16*
Other factors for ERF	1.5	*0.16*
Monetary valuation	2	*0.48*
Total σ_g	**4.1**	***2.02***

4. Results and Conclusions

Our damage cost estimates are summarized in Table 5. For the derivation, see our companion papers in this series. They include discounting at a discount rate of 4%. The choice of the threshold, if any, is indicated, together with the fraction f_{thr} of the incremental exposure above threshold. For comparison the last row for each metal shows the values of ExternE [2008]; they are much lower because only cancers were taken into account for As and Cd, and only IQ loss for Hg and Pb.

Since these metals (with the exception of much of the Hg) condense onto particles in the air and $PM_{2.5}$ causes severe health impacts, the reader might wonder if the ERFs for $PM_{2.5}$ should also be taken into account. That would raise questions of double counting and questions about the role of the composition of $PM_{2.5}$ for those ERFs. However, the damage cost per kg of $PM_{2.5}$ emitted by industrial installations is on the order of 20 €/kg, and even if one were to simply add such a contribution, it would be negligible.

Table 5. Summary of damage costs contributions for each of the metals, in €/kg, for industrial emissions in the EU. Costs are in $€_{2010}$.
Cases are without f_{thr}, with units YOLL for non-cancer mortality and IQ points for IQ loss.
f_{thr} *= fraction of incremental exposure above threshold.*
The last line for each metal shows the value used by ExternE [2008], if any.

a) Arsenic

Threshold = 3.0E-04 mg_{As}/kg_{body}/day *of EPA, applied to all endpoints.* f_{thr} *= 0.80.*
ExternE [2008] had used 530 €/kg_{As}.

Endpoint	**Cases/kg_{As}**	**Undiscounted, no threshold**	**Lag [yr]**	**Discount factor**	**Discounted, no threshold**	**Discounted, with threshold**
Non-cancer mortality	9.95E-03	2,606	10	0.68	1,760	1,413
Cancer deaths	5.96E-04	1,787	20	0.46	816	655
Non-fatal cancers	6.54E-04	105	20	0.46	48	38
Chronic bronchitis	1.58E-03	332	10	0.68	224	180
IQ loss	4.93E-02	715	0	1.00	715	574
Infant deaths	9.88E-06	59	10	0.68	40	32
Diabetes	1.30E-02	2,411	10	0.68	1,629	1,308
Total cost, $€_{2010}/kg_{As}$		8,014			5,231	**4,200**

b) Cadmium.

Threshold = 0.1 μg/kg_{body}/day [ATSDR 2012]*, applied to all endpoints.* f_{thr} *= 0.98.*
ExternE [2008] had used 84 €/kg_{Cd}.

Endpoint	**Cases/kg_{Cd}**	**Undiscounted, no threshold**	**Lag [yr]**	**Discount factor**	**Discounted, no threshold**	**Discounted, with threshold**
Mortality	1.633	137,187	10	0.68	92,679	91,168
Non-fatal cancers	0.005	868	20	0.46	396	390
Fractures	0.016	1,899	10	0.68	1,283	1,262
Total $€_{2010}/kg_{Cd}$		139,954			94,358	**92,820**

c) Mercury.

Threshold = 6.7E-03 mg_{Hg}/kg_{body}/day [EPA 2001] and f_{thr} = 0.44.
ExternE [2008] had used 3,400 €/kg_{Hg}.

Endpoint	**Cases/kg_{Hg}**	**Undiscounted, no threshold**	**Lag [yr]**	**Discount factor**	**Discounted, no threshold**	**Discounted, with threshold**
Mortality	0.56	46,723	10	0.68	31,564	13,888
IQ loss	1.36	4,257	0	1	4,257	1,873
Total $€_{2010}/kg_{Hg}$		50,980			**35,821**	**15,761**

d) Lead.

Threshold = 0 and f_{thr} = 1 for all endpoints.
ExternE [2008] had used 278 €/kg$_{Pb}$.

Endpoint	Cases/kg$_{Pb}$	Undiscounted, no threshold	Lag [yr]	Discount factor	Discounted, no threshold
Mortality	0.27	24,531	10	0.68	16,572
IQ loss	0.29	3,948	0	1	3,948
Anemia	1.44E-04	49	10	0.68	33
Total €$_{2010}$/kg		25,528			**20,553**

It is interesting to look at the implications of these results for combustion of coal and incineration of waste. Table 6 shows the damage costs due to atmospheric emission of toxic metals, together with CO_2 and the classical air pollutants. The emissions can vary from one installation to another and getting representative measured data is not easy. Therefore we show both the emission limits in the EU and actual emissions in France. Since the emission limits are formulated in terms of flue gas concentrations, we have translated them to g/t$_{waste}$ by assuming 5150 Nm^3/t$_{waste}$. The data for incinerators in France are based on CITEPA [2013], the European Pollutant Release and Transfer Register (E-PRTR) and discussions with Olivier Guichardaz of Dechets-Infos. The toxic metals make a very significant contribution, especially Cd, Hg and Pb. To put the numbers in perspective, note that the private cost of waste incineration is on the order of 100 €/t$_{waste}$: the damage costs are still very significant, even after major reductions compared to incinerators of the past.

Table 6. Damage costs due to atmospheric emissions by waste incinerators. Costs are in €$_{2010}$.

		Limit values EC [2000]		Actual emissions, France 2011	
Pollutant	**€$_{2010}$/kg**	**g/t waste**	**€$_{2010}$/t waste**	**g/t waste**	**€$_{2010}$/t waste**
CO_2	0.021	863867	18.14	863867	18.14
PM_{10}	7.65	51.5	0.39	7.3	0.06
SO_2	6.20	257.5	1.60	54.3	0.34
NO_2	5.36	1030	5.52	600.0	3.22
As	4200.04	0.072	0.30	0.013	0.06
Cd	92819.64	0.209	19.41	0.007	0.62
Hg	15761.29	0.258	4.06	0.053	0.84
Pb	20553.02	0.567	11.64	0.093	1.92
Total			**61.07**		**25.19**
Toxic metals			**35.41**		**3.43**

[a] actual emissions because no limit value for CO_2

Both coal and oil contain toxic metals such as As, Pb and Hg, some of which escapes through the smoke stacks of power plants. The amounts of these trace metals can vary greatly with the origin and type of oil or coal. In Table 7 we show emissions data and damage costs for hard coal condensing power plants in the EU27 according to ExternE [2008]. The largest contributions are due to Hg and Pb, but even those are small compared to the damage costs of the classical air pollutants and greenhouse gases, and compared to the private cost of electricity, around 10 €cent/kWh.

However, what matters for policy applications is not the absolute magnitude but the comparison of the damage cost to the cost of pollution abatement (both upstream and end of pipe) for each pollution source. New cost-benefit studies for the abatement of toxic metal emissions are advisable, with particular attention to older more polluting plants that may still be operating. We also emphasize that even though the uncertainties are large, it is not the uncertainties themselves that matter but their effect on policy choices. As shown by Rabl, Spadaro and van der Zwaan [2005], despite such uncertainties the results are very useful for environmental policy because they can help avoid costly mistakes.

Table 7. Emission (to air) and damage cost of toxic metals from coal fired power plants. Emissions data of ExternE [2008]. Costs are in €$_{2010}$.

Pollutant	**€$_{2010}$/kg**	**kg/kWh**	**€cent$_{2010}$/kWh**
CO2	0.021	7.30E-01	1.533
PM10	7.65	2.74E-05	0.021
SO2	6.20	5.51E-04	0.342
NO2	5.36	5.54E-04	0.297
As	4200.04	7.98E-09	0.003
Cd	92819.64	3.92E-10	0.004
Hg	15761.29	2.54E-08	0.040
Pb	20553.02	3.43E-08	0.070
Total			**2.31**
Toxic metals			**0.12**

The methodology demonstrated in this paper should likewise be used for any other pollutant that might entail significant health impacts. In particular we have found that new epidemiological findings yield impacts that are far larger than what had been suspected. This highlights once again [10] the importance of periodically performing or revising studies of this kind, with an eye open to possible surprises.

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Abbreviations and symbols

D = damage cost, €/kg
DALY = disability adjusted life year
E = collective exposure, as intake in mg/yr
e = individual exposure, as intake in mg/yr
ERF = exposure-response function

[10] A similar surprise happened when externality studies started around 1990 and found health impacts of air pollution far larger than expected.

f_{thr} = fraction of collective incremental exposure that is above threshold
I = impact rate, cases/yr
IF = intake fraction, mg/kg
LE = life expectancy
OR = odds ratio
P = unit cost ("price") of end point, €/case
PPP = purchasing power parity
Q = emission rate of pollutant, kg/yr
QALY = quality adjusted life year
RfC (RfD) = reference concentration (dose) = maximum ambient concentration (dose) for lifetime exposure that is not likely to cause harmful effects
RR = relative risk
s_{ERF} = slope of ERF, cases/mg
VOLY = value of a life year
VPF = value of prevented fatality (value of statistical life)
YOLL = years of life lost

μ_g =geometric mean
σ_g = geometric standard deviation

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