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HUMAN HEALTH IMPACT DAMAGE FACTORS FOR SUBSTANCES RELEASED TO WATER: A PILOT STUDY FOR THIRTY INDUSTRIAL CHEMICALS

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Dr. Patrick Hofstetter, an expert on life cycle assessment of health impact assessment, is PI and coordinator of this pilot study. The other coauthors are from the Swiss Federal Institute of Technology in Lausanne where the database and the computer system resides. The specific thrust of this work is to calculate new damage factors for a list of chemicals which are deemed relevant to the automotive industry from a life cycle assessment point of view. This new information on the non-carcinogenic effects for substances released to water can be used in environmental health assessment and in Design for the Environment applications.

ABSTRACT

A newly developed decision model based on the output of GaBi, a life cycle assessment software tool used by GM, uses human health effects damage factors in units of disability adjusted life years (DALY) per kg of substance. However, the lack of damage factors for emissions to water (w) with non-cancer (nc) outcomes and issues with the CalTox model for cancer effects in water have not been resolved. A new set of damage factors (DALYw,nc) for thirty substances of interest to the auto industry were calculated in this pilot study. These values will help improve the accuracy of the recently developed LCA-DfE decision model in GM. A strategy to develop a more comprehensive set of damage factors for chemicals relevant to the global automotive industry and to selectively improve the impact assessment models is proposed.

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PURPOSE OF THIS RESEARCH

The primary aim of this feasibility study is to calculate new human health impact damage factors for non cancer effects as input to an LCA (Life Cycle Analysis) model for industrial chemicals that are released to water.

SUMMARY

1. Damage factors in Disability Adjusted Life Years per kg emission (DALY/Kg) were estimated for a total of thirty substances, including heavy metals and selected organic chemicals released to surface waters (see Table 1, page 16). DALY accounts for both mortality and morbidity outcomes, including the concept of Years of Life Lost (YLL) due to premature death as a result of a disease.
2. For the 30 substances, human exposure is predominantly via oral exposure from the consumption of drinking water and fish. Better data in predicting pollutant residence times in water and the water/sediment-to-human exposure pathways will improve the uncertainties of the estimates. Toxicological data for these compounds are based on peer reviewed data summaries. Further research is necessary to relate the observed non-cancer effect endpoints to DALYs, particularly at low doses and for the complex chemical mixtures in the environment.
3. The uncertainty range of the estimated damage factors is typically larger than for previously published damage factors for other substances by Goedkoop *et al*. (2000). Reasons are that none of the new damage factors rely on epidemiological studies and that the speciation of metals was not considered when calculating fate and exposure.
4. The significance of small quantities of moderately toxic substances such as Cr, Pb and other emissions to water will provide better insight in developing a green manufacturing strategy.

SIGNIFICANCE

The new damage factors for substances that may elicit non-cancer outcomes will improve the existing environmental health management and life cycle assessment models for emissions to water.

INTRODUCTION

Life cycle impact assessment (LCIA) methodology (Goedkoop *et al*. 2000), is of value in the assessment of the environmental impact of design alternatives of new products and processes. The existing method contains only a limited number of damage factors for selected stressors. Therefore, this study was undertaken to provide estimates for human health damage factors due to non-cancer effects for a selected list of 30 substances that are released to surface waters. This list was based on typical emissions linked to the automotive industry in LCA studies and new materials that may be used for the manufacture of future vehicles.

This application of these damage factors will enable the identification of emissions to water that may contribute to human health impact in a significant way from manufacturing processes. This is consistent with the current use of the LCA damage oriented impact assessment methodology in Europe and Japan. This approach will play an increasingly important role in light of the recent UNEP/SETAC Life Cycle initiative[1].

The approach taken follows the principles suggested in Hofstetter (1998), is similar to the Eco-Indicator'99 report (Goedkoop et al. 2000), and has been further developed and described for screening level damage factors in Pennington *et al*. (2002a/b). This follows the impact pathway analysis approach, where fate, exposure, likelihood (or risk), and damage (or impact) analysis are used to causally link emissions with damages to safeguard subjects – in this case to human beings and human health. European landscape parameters and population densities were used to be consistent with the other damage factors, noting the important updates in methodology made in IMPACT2002 – as described in detail in Section 2 below. The multimedia/multi-pathway approach developed at EPFL (Ecole polytechnique Fédérale de Lausanne), drawing heavily on the Eco-indicator 99 report and other earlier achievements, is used for the fate, exposure, and toxicological effect modeling.

[1] United Nations Environment Programme (UNEP) and the Society of Environmental Toxicology and Chemistry (SETAC) have joined forces to identify best available methods for inventory analysis, impact assessment, and life cycle management, see http://www.uneptie.org/sustain/lca/lca.htm

The thirty substances chosen are very diverse in nature and would, in a strict sense, need elaborate modeling that may differ from substance to substance. Instead, consistent with current best-available practice in LCA, simplified fate models and exposure assessment were used to estimate the damage factors. This caveat applies especially to metals and inorganics, where the given descriptions are not precise as to the valence state and compounds.

In order to be consistent with the methodology used in Eco-indicator'99, the substances were assigned to three cultural perspectives, i.e. **I**-Individualist, **H**-Hierarchist, **E**-Egalitarian. While the details on this approach are described in Hofstetter (1998) and summarized in Goedkoop et al. (2000), the underlying idea is that different societal groups request different degrees of evidence and confidence before they accept information to be robust enough for inclusion in decision support. The **I** perspective accepts only information that has been proven – if possible in the real world. Since representatives of the **I** perspective have a strong belief in the individual's ability to react in innovative ways to hazards if they should occur, they are confident that low evidence information is of no value. The **E** perspective is somewhat at the other extreme. They see low evidence information as an essential part of decision support in order to avoid future uncontrollable hazards. The precautionary principle is their guiding management style. The **H** perspective is often seen as somewhat in between these two extremes. The **H** perspective attempts to balance precautionary measures with societal acceptability. They also believe in a hierarchic regulatory system that is able to deal with risks.

The damage factors determined in this study are considered a first attempt to quantify the potentially relevant emissions in LCA studies. This is partly due to the generalizations adopted in the fate and exposure model for the broad group of chemicals studied, especially the metals. In addition, the lack of epidemiological data or accurate dose-response relationships for humans was an issue. Another important point is that the Eco-Indicator'99 model considered European geography, climate, food production, and population parameters. It will be necessary to make appropriate changes in these

parameters if the health effects metrics are to be applied for GM's global operations.

METHODOLOGY AND DAMAGE ANALYSIS

IMPACT 2002 is a tool that facilitates default estimation of regional and global scale toxicological risks and potential impacts to human health and ecosystems from the emissions inventory data in a life cycle assessment (Pennington *et al*. 2002a). Unlike traditional regulatory-orientated toxicological risk assessments, the risks and potential impacts are integrated over both time and space. The results of an LCA using this method can be interpreted in terms of estimates of the time and space integrated (or cumulative) risks and, where estimations are feasible, the potential impacts (or damage) attributable to toxicological effects (see Figure 1).

As the results are risk-based estimates they do not necessarily imply that actual impacts will occur. The risks attributable to one particular site may not reflect the full extent of risks from that site (due to allocation amongst co-products in the inventory stage of an LCA, that the functional unit may not reflect the full extent of annual production, etc.), and the risks may occur over multiple generations in the case of persistent compounds. The estimates will not reflect risks, or potential damage, at any one particular point in time.

Accounting for the potential impacts of non-cancer effects, and cross-comparison with cancer effects for human health remains particularly problematic. As recommended by Bare et al. (2000), cross-comparison of LCA results can be made at this time using the proposed Disability Adjusted Life Year (DALY) based measures, as well as drawing parallel insights directly from the risk-based estimates (which ignore differences in potential consequences).

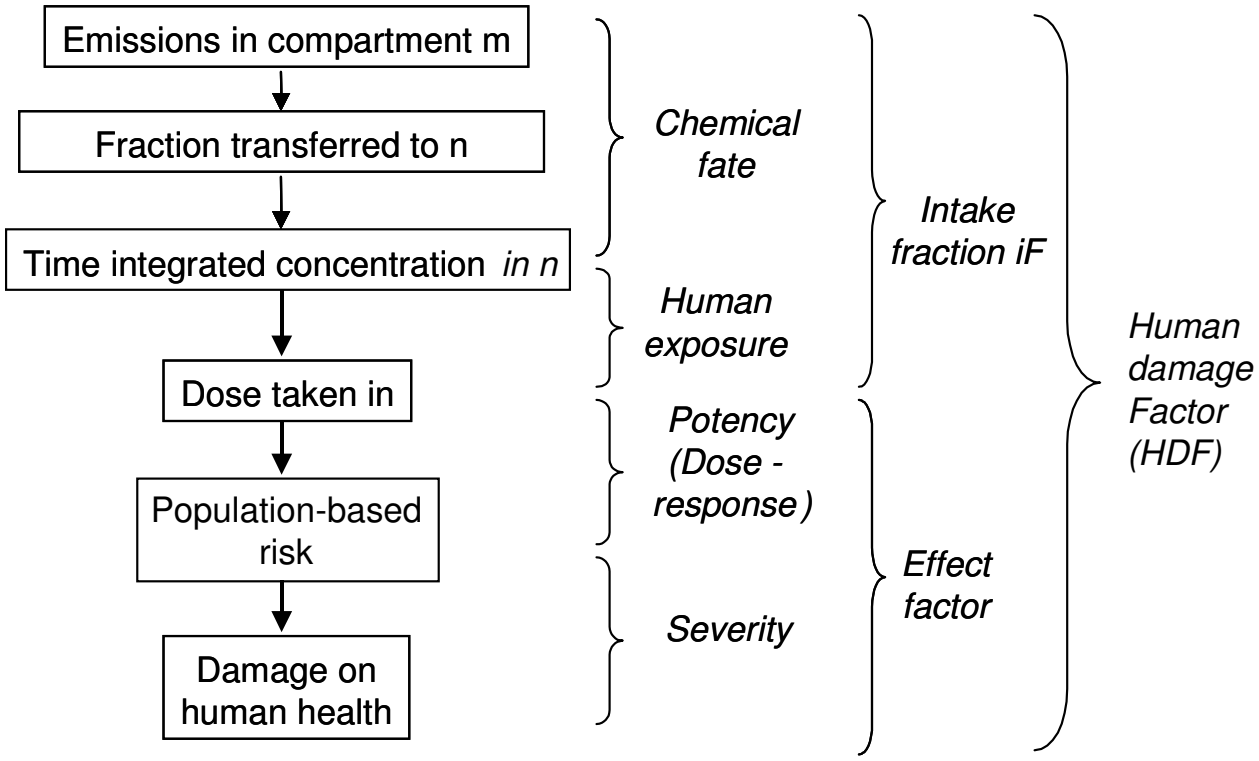

Figure 1 General framework for the calculation of toxicological characterization factors in Impact 2002 (Pennington *et al*. 2002a).

FATE & EXPOSURE MODELLING

The combined fate and exposure measure for human health is the intake fraction (iF). This fraction is dimensionless and represents the fraction of a chemical released that will ultimately result in human population exposure (Bennett *et al*. 2002). The mass distribution of a chemical in an n compartment model is calculated using a matrix algebra solution to the (pseudo-linear or marginal) first order differential equations (requiring no iterations and readily represented in most spreadsheet software):

$$\frac{\overline{d\mathrm{M}}}{d\mathrm{t}} = \overline{\mathrm{S}} - \overline{\mathrm{k}} \cdot \overline{\mathrm{M}} \qquad (1)$$

where $\overline{\mathrm{S}}$ is the vector of emissions S_n (kg/day), $\overline{\mathrm{M}}$ is the vector of masses M_n (kg), and

$\bar{k}$ is the rate coefficient matrix. The rate coefficient matrix $\bar{k}$ consists of off-diagonal elements $k_{n,m}$ representing intermedia or advective transport between compartments and the diagonal elements k_n representing total removal from compartment n.

Assuming steady-state (dM/dt = 0), an assumption adopted throughout the analysis in this report, we get:[2]

$$\bar{M} = -\bar{k}^{-1} \cdot \bar{S} \qquad (2)$$

Combining the mass balance results $\bar{M}$ with the volume vector $\bar{V}$, yields the concentration vector $\bar{C}$, describing the concentration distribution of a chemical in an n-compartment system.

If ΣS_n=1 kg/day, then the intake fraction in the n-compartment system is

$$iF = \sum_{1}^{n} C_n \cdot IR_n \qquad (3)$$

The intake rate (IR) associated with compartment n is:

$$IR_n = IR_{n,\,direct} + \sum_{e=1}^{n-1} BAF_{n,e} \cdot IR_{n,e} \qquad (4)$$

$IR_{n,direct}$ is the direct intake rate in kg/day (or m^3/day for inhalation). For example, if compartment n is air, then $IR_{n,direct}$ is the breathing rate multiplied by the population in the compartment. The second term is the indirect intake rate, via substrate e, that is linked to the concentrations in compartment n. For example, an air compartment (denoted n) may include cattle (denoted e). The cattle are exposed to contaminants in the air via inhalation. The contaminant concentration in the meat is estimated from $BAF_{n\,e}$ ($kg \cdot kg_e^{-3}$

[2] The dynamic solution to estimate $\bar{M}(t)$ is available in Pennington et al. (2002a).

per $kg \cdot kg_n^{-3}$). The meat is then processed, thus transferring the contaminant from air to the human food web at a production rate of IR_e (kg/day). In this example, IR_e is the quantity of meat produced in compartment n that will be ultimately consumed (irrespective of where it is consumed, as population-based, not individual, intake is estimated).

Analogous to aquatic ecotoxicological assessment measures, $BAF_{n\ e}$ quantifies the pollutant associated with an environmental concentration in compartment n to the concentration in substrate e:

$$BAF_{n\,e} = \frac{C_e}{C_n} \qquad (5)$$

where C is chemical concentration ($kg_{chemical}/kg_{substrate}$). Such bioaccumulation factors can be either measured or, for non-dissociating organic chemicals, estimated. BAF is currently estimated in IMPACT 2002 by multiplying predicted biotransfer factors (BT) [3] by specific intake rates ($BAF_{n,e} = BT \cdot IR'_e$), where IR' denotes the individual species uptake rate such as mass of vegetation consumed by a dairy cow per day.

RATE COEFFICIENT (K) ESTIMATION

Intermediate transfer and degradation rate coefficients ($k_{mn,\ int}$ and $k_{n,deg}$, respectively) are either entered by the user or, for organic chemicals, estimated using correlations. The parameters used to estimate these location-specific coefficients for each module are obtained with the help of GIS-based databases and tools, as described in Pennington et al. (2002a).

In addition to intermedia and degradation rate coefficients, the spatial model additionally requires the specification of advective air and water flow rate coefficients ($k_{mn,\ adv}$) to

[3] A biotransfer factor represents the ratio of chemical concentration in meat to the intake of that chemical by a species such as cattle [day/kg_{meat}]. Biotransfer factors are currently estimated in IMPACT 2002, as a default for organic chemicals, from the octanol-water partitioning coefficient (K_{ow}) using correlations suggested by Travis and Arms (1988) for 36 and 28 organic chemicals for beef and milk transfer, respectively.

account for the transport of chemicals between compartments (air cells and watersheds in the model). These advective flow rate coefficients are represented in matrices, as described in the following sections and first proposed by MacLeod et al. (2001). Combining the different coefficients $k_{mn,\ int}$, $k_{n,deg}$, and $k_{mn,\ adv}$ provides the overall rate coefficient matrix, $\bar{k}$.

Generic environmental compartment modules are included for air, surface water, soil, sediment, agricultural vegetation, and oceanic waters. The correlations are primarily based on Mackay *et al*. (1996) and Schwarzenbach *et al*. (1993).

FATE SCENARIO

The concentration of a chemical at a given location for an emission profile is readily estimated for releases to air, water, soil, and agricultural produce (pesticide applications, etc.) – water only in this report. Two model scenarios are currently available to provide estimates for releases in Western Europe (Pennington *et al*. 2002a):

- A "nested" a-spatial scenario for Western Europe. Typical of traditional multimedia models for initial insights, no spatial resolution is taken into account within each environmental medium (air, water, soil, sediment, vegetation).

- A "nested" scenario with spatial resolution in each environmental medium for Western Europe (number of compartments, n ≈ 600); providing space-dependent insights into the relationship between the emission of a chemical and concentration profiles. Contaminant transfer to humans via food production, water supply, and as a function of population distribution are not assumed to be uniform in this scenario, as further outlined in the next section. (*not used in this preliminary study*)

The Western European scenario models are nested, or linked, to a global a-spatial model to account for advective transfers between Western Europe and the rest of the world.

Such transfers would otherwise be neglected.

The Western Europe model consists of 135shandwork@whoi.edu, amcnichol@whoi.edu watershed zones, 124 oceanic zones, and 156 (2×2.5 degree) air grid cells. Each watershed zone consists of four compartments; land, surface water, sediment, and agricultural vegetation. Oceanic zones consist of a two-layer sea model and a bed sediment compartment. Advective transport between watersheds and between atmospheric grid compartments is taken into account, as described above.

For each watershed, location specific parameters are given. Not all the parameters were readily available for use in this model with the same precision; indeed the degree of precision may vary depending on the significance of a parameter. For some parameters default (generic) values were retained, pending future sensitivity analyses. The mean values in each of our watershed zones were determined using straightforward GIS manipulations.

EXPOSURE SCENARIO

Many current multimedia fate and exposure models adopt a subsistence-based exposure scenario. In this subsistence based scenario it is assumed that all the food that is eaten is produced in the same region where the people are living. Food is consumed at a rate equivalent to the population demand. In most cities, for example, this is clearly an erroneous assumption. Food and water supplies are often from different regions. The contaminant concentration in the food will, in general, correspond to the concentrations where the product was produced and not where it is consumed.

In the production-based approach proposed by Pennington *et al*. (2002a) it is assumed that a fraction of the food produced in one location will be eaten by a human or livestock - irrespective of where they live. The fraction of contaminant entering the human food chain, hence the oral intake fraction, is estimated from water supply, agricultural production, and livestock production statistics. Correction factors account for the fraction

of food produced that is eventually consumed (estimated from production statistics and food basket surveys). No differentiation is made whether the food is eaten in the same zone, or exported somewhere else (some models only account for the fraction of food produced locally that is eaten locally; neglecting contaminant export). However, in a region like Europe the export of contaminated European food, compared to its internal use, is in general small.

The current model does not compute individual intake distributions, as may be required in some risk assessments. The intake fraction is calculated on a population basis; the fraction of contaminant resulting in intake by the human population. Estimation of cohort or individual intake, other than population based averages, would require additional knowledge of import-export statistics between compartments m and n, and individual consumption profiles.

Inhalation intake is estimated by assuming an average inhalation rate and estimating the population in a given location (air compartment) from population density statistics. Inhalation and oral exposure in compartments outside of Western-Europe are currently taken into account by assuming a uniform world population density, water supply, and agricultural/livestock production levels.

POTENCY (DOSE-RESPONSE) MODULE

The comparative nature of LCA justifies caution when adopting the effect methods and policy-based measures used for regulatory-orientated toxicological risk screening (Burke *et al*. 1996). In response to the limitations, Crettaz and colleagues (Crettaz *et al*. 2002, Pennington *et al*. 2002b) proposed a methodology for addressing human health effects in LCA based on the risk assessment concept of a benchmark dose:

$$\beta = \frac{\beta_{ED10}}{\mathrm{BW} \cdot \mathrm{LT_h} \cdot \mathrm{N_{365}}} \qquad (10)$$

β_{ED10} Slope factor [risk per mg/kg - day]

BW	Body weight [kg/pers]
LT_h	Lifetime of humans [yr]
N_{365}	Number of days per year [days/yr]

Crettaz et al. (2002) focused on the quantification of the cancer effect measure in LCA. Derived from the health risk assessment concept of benchmark doses, the approach is based on the maximum likelihood estimate of the effect dose inducing a 10% risk over background, denoted the ED_{10}. The default linear slope factor, β_{ED10}, represents the slope of the straight line drawn from the reference point, ED_{10}, down to the origin of a dose-response curve. This default is not a function of the individual or a population cohort, hence location, although distinctions are retained between inhalation and oral exposures. Using bioassay data for 44 chemicals and a best-estimate extrapolation factor from the TD_{50}, Crettaz *et al*. (2002) proposed β_{ED10}s for more than 600 carcinogenic compounds.

Pennington *et al*. (2002b) proposed a new approach to calculate the health effect measure for non-cancer toxicological impacts for use in LCA. The measure is again derived from the health risk assessment concept of benchmark dose (ED_{10}) to estimate a default linear low-dose extrapolation (β_{ED10}). Preliminary β_{ED10} slope factors were calculated from bioassay data for 12 chemicals and, using best-estimate extrapolation factors from NO(A)EL and LO(A)EL data[4], facilitated the initial calculation of slope factors for an additional 403 compounds. In Section 3 we describe how the ED_{10} values were derived for this study.

DAMAGE MODULE

Severity differences are optionally taken into account in IMPACT 2002 by multiplying β by the Disability Adjusted Life Years per affected Person, $DALY_P$, as described in Crettaz *et al*. (2002) and Pennington *et al*. (2002b). The DALYp accounts for both mortality and morbidity outcomes, including the concept of Years of Life Lost (YLL) due to premature

death as a result of a disease.

Considering 17 types of cancer and using data on a world scale from Murray and Lopez (1996), the default DALYp value is 7 [year/incidence]. Prostate cancer has the lowest DALYp = 2.1 [year/incidence] and leukemia has the highest DALYp = 14.6 [year/incidence]. DALYp values are not readily available for most non-cancer effect endpoints at this time. Effects were subcategorised in terms of severity based on proposals by Burke *et al*. (1996). As a starting point, this simplistic classification was modified to be compatible with the DALY approach; adopting as a preliminary basis a $DALY_p$ (average DALY per person) of 7 [year/incidence] for category 1 (equivalent to that of cancer effects, which were originally in this category). Differences between the sub-categories were originally scaled subjectively by the International Life Science Institute (ILSI) panel using factors of 10 and this is retained. Hence, for categories 2 and 3, the $DALY_p$ values are 0.67 and 0.067 [year/incidence]. A default value of 0.67 DALY per incident case was proposed in Pennington *et al*. (2002b), in the absence of alternatives and to help provide preliminary insights in an LCA study. The same default value is used here.

CULTURAL PERSPECTIVE AND UNCERTAINTY CLASSIFICATION

For this preliminary analysis, the damage factors were developed with best-available, as far as possible, but still limited toxicological information in the context of the assessment endpoint (non-cancer human health effects). All the damage factors were estimated by the linear extrapolation of results from high dose studies to the low doses. Due to this assumption, the damage factors may not be used, in general, in the context of the **I** perspective. Potential exceptions with sufficient epidemiological data to derive a regulatory threshold are cadmium (oral), chromium (inhalation), mercury (inhalation), selenium (oral), zinc (oral), TCDD (oral, although monkey study), formaldehyde (oral), PCB 1254 (oral), styrene (inhalation), toluene (inhalation), xylene (inhalation). However, a default DALY per incidence was applied to these study results. The damage results are

[4] No and Low Observed (Adverse) Effect Levels.

therefore only preliminary at this stage, suggesting that classification under the **I** perspective is not valid in a strict sense.

For the majority of the chemicals toxicological data were available and were adequate to derive RfD (reference doses), or similar regulatory values. This implies high evidence of the associated effects and their likely relevance in the context of human health. Again, a default DALY per incidence was adopted. From these insights, and in conjunction with the preliminary values used in the fate and exposure models, we suggest that these damage factors are only adopted in the **E** and **H** perspectives – and only to provide screening insights.

A rough estimate on the uncertainty factors and 95% confidence interval is given in Table A1. This reflects the uncertainty associated with the fate and exposure model estimates (intake fractions), and factors of 10 (squared geometric standard deviation) for the dose-response slope and the DALYs per incident case. We assume independent lognormal parameter distributions and estimate the 95^{th} percentile confidence interval following the method presented in Hofstetter (1998). This does not reflect a careful uncertainty analysis but a guess that can indicate the confidence we have into the values and where further analysis may be needed to narrow down this uncertainty.

Exposure to most of the organic chemicals is associated with drinking water or fish consumption. The chemical parameters are considered to be of high quality, in general. As no significant partitioning is involved in the source-to-intake pathway the corresponding uncertainties are considered to be relatively low. For the metals, intake is again usually associated with fish consumption or drinking water. Most of the input data associated with the source-to-intake estimates are empirical, so a low uncertainty could similarly be assumed. However, following the method of Hofstetter (1998) a higher confidence interval is assigned. These insights are suitable for providing preliminary insights.

CHARACTERIZATION PROFILES FOR THE THIRTY SUBSTANCES

Table 1 presents the compiled results for the 30 chemicals (and chemical groups) in this study. The values reflect damage measured in disability adjusted life years due to non-cancer effects per unit mass of emission of the chemical to water (post treatment), with associated confidence intervals. Details on the environmental fate data and toxicological survey data are summarized in the tables in the Appendix section.

Table 1: Damage factors for non-cancer effects for emissions to water in Disability Adjusted Life Years (DALYs) per kg emission.

Chemical CAS#	Name	Non-cancer damage factor (DALY/kg)	Upper 95th percentile (DALY/kg)	Lower 95th percentile (DALY/kg)
1746-01-6	2,3,7,8 TCDD	**1.6E+03**	7.7E+04	3.4E+01
11097-69-1	PCB 1254 (under Aroclor 1254)	**7.2E-02**	3.4E+00	1.5E-03
822-06-0	HDI (1,6-Hexamethylene diisocyanate)	**5.1E-04**	1.4E-02	1.8E-05
7439-97-6	Mercury (inorganic)	**2.0E-04**	4.6E-02	8.4E-07
7782-49-2	Selenium and compounds	**1.5E-04**	3.4E-02	6.2E-07
7440-43-9	Cadmium	**5.2E-05**	4.3E-03	6.2E-07
7440-66-6	Zinc and compounds	**1.7E-05**	4.0E-03	7.2E-08
75-01-4	Vinyl chloride	**1.1E-05**	3.1E-04	4.0E-07
101-68-8	MDI (Monomeric Methyl Diphenyl Diisocyanate)	**8.7E-06**	4.1E-04	1.8E-07
56-23-5	Carbon tetrachloride	**6.6E-06**	1.8E-04	2.4E-07
7440-39-3	Barium and compounds	**4.3E-06**	3.6E-04	5.1E-08
7439-92-1	Lead and compounds (inorganic)	**2.7E-06**	6.5E-04	1.2E-08
71-43-2	Benzene	**1.3E-06**	5.5E-05	3.2E-08
7440-41-7	Beryllium and compounds	**6.7E-07**	5.6E-05	8.0E-09
7440-02-0	Nickel, soluble salts (no CAS#)	**5.4E-07**	4.5E-05	6.4E-09
62-53-3	Aniline	**2.7E-07**	7.4E-06	9.5E-09
7440-50-8	Cu	**2.6E-07**	2.1E-05	3.1E-09
7440-47-3	Chromium IV (18540-29-9)	**2.5E-07**	2.1E-05	3.0E-09
75-09-2	Dichloromethane (methylene chloride)	**2.3E-07**	6.4E-06	8.2E-09
106-99-0	1,3 butadiene	**2.3E-07**	6.5E-06	8.4E-09
95-63-6	1,2,4-trimethylbenzene	**1.2E-07**	3.4E-06	4.4E-09
7429-90-5	Aluminium	**9.7E-08**	2.3E-05	4.1E-10
1634-04-4	MTBE	**5.0E-08**	1.4E-06	1.8E-09
100-41-4	Ethylbenzene	**4.0E-08**	1.1E-06	1.4E-09
50-00-0	Formaldehyde	**2.3E-08**	6.4E-07	8.3E-10
108-88-3	Toluene	**1.6E-08**	4.6E-07	5.9E-10
110-80-5	2-Ethoxyethanol	**7.3E-09**	2.0E-07	2.6E-10
1330-20-7	Xylene (all isomers)	**7.1E-09**	2.0E-07	2.6E-10
67-56-1	Methanol	**2.0E-09**	5.5E-08	7.1E-11
107-21-1	Ethylene glycol	**1.5E-09**	4.2E-08	5.4E-11
100-42-5	Styrene	**1.0E-09**	2.8E-08	3.7E-11
78-93-3	MEK (Methyl Ethyl Ketone)	**5.5E-10**	1.5E-08	2.0E-11
57-55-6	Propylene glycol	**1.0E-10**	2.9E-09	3.7E-12

As expected, the magnitude of damage varies widely, i.e., 13 orders of magnitude from 1E-10 to 1.6E3 DALYs/kg and still eight orders of magnitude if dioxins are excluded. This wide range justifies this type of screening factors even if the range of the confidence intervals are large. They will be able to discriminate between substances and allow to prioritize the further analysis in concrete case studies. Additional insights on the relevance of different pathways, adopted uncertainty factors, and a comparison – where available – with cancer effects from Crettaz *et al*. (2002) are provided in Table A1.

The comparison with cancer effects taken from Crettaz *et al*. (2002) (Table A1) shows that for some substances cancer (toluene, ethyl benzene, carbon tetrachloride), for others non-cancer endpoints dominate. In cases where substances show both carcinogenic and non-carcinogenic effects we expect some correlation due to the common fate and exposure analysis. However, as Table A1 shows, the dose-response relationships can be very different and makes that the correlation for damage factors is rather poor.

Factors from Hertwich *et al*. (2001) were not adopted, partly due to the limited coverage of chemicals for this case study. Advances have been made in the context of LCA in recent models. These are outlined in the methodology section. One key improvement includes moving away from regulatory thresholds to effect measures more applicable in comparative assessments like LCA. Published figures by Hertwich *et al*. (2001) similarly do not include data pertinent to the calculations for metal emissions to surface waters, including oral effect factors and some partitioning parameters. This should be kept in mind when HTP values from Hertwich *et al*. (2001) are compared with damage factors in Goedkoop *et al*. (2000) and this study. Main differences include the different geographical area (U.S. *versus* Europe) and the different dose-response information used for non-cancer effects and the different methodology for oral effect factors for carcinogenic effects. Nevertheless, a regression of the new DALYw,nc values against the HTP values of Hertwich *et al* (2001) for non-carcinogenic outcomes for organic chemicals showed a remarkable correlation with a correlation coefficient r^2 of 0.88 (see Figure 2).

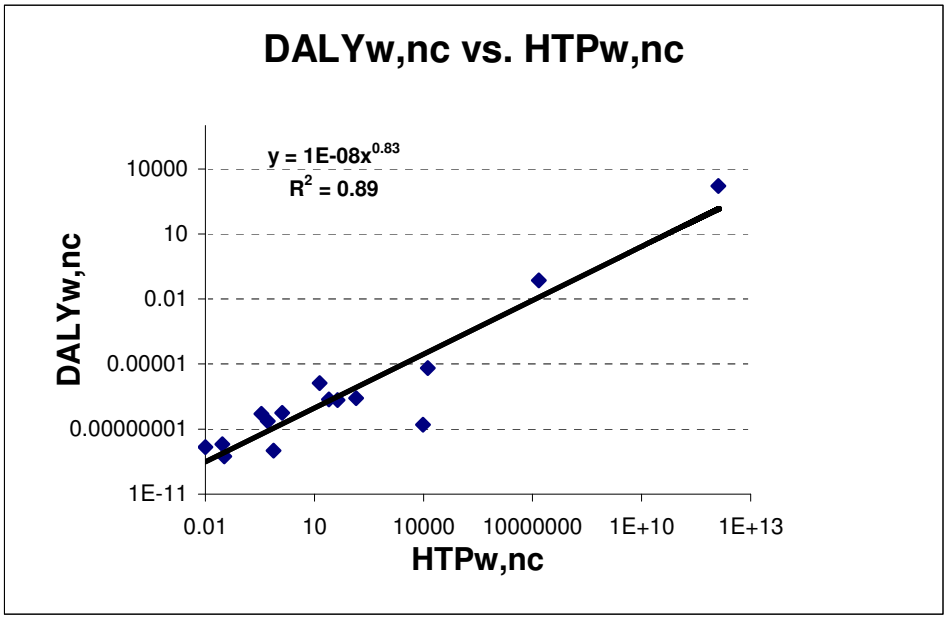

Figure 2. New DALYw,nc values correlated against HTPw,nc values by Hertwich (2001)

No final results are given for inhalation for metals (and metal compounds) released to water, as these chemicals remain predominantly in the water and sediment compartments with negligible transfer to air.

Table A2 summarizes the chemical fate parameter data, including the data source, adopted in this study for metals. With the exception of aluminum, the data are provided by Huijbregts (1999). Exposure data for aluminum were presented in the Agency for toxic Substances and Disease Registry (ATSDR) profiles. Fate data were derived using expert judgment, from Thallium data where available. While four partitioning coefficients are presented based on measurements, the suspended solids/sediments solids to pure water partitioning coefficients are generally only important for metal (and metal compound) emissions to water.

Tables A3 summarizes the chemical fate parameter data, including source, adopted in

this study for the organic chemicals. These data were primarily based on values in Mackay *et al*. (1991-1997), Howard *et al*. (1991), and models from Syracuse Research Corporation – in that order of preference. For emissions to water, the octanol-water partitioning coefficient (Kow), the Henry's Law constant (H), the water degradation half-life, and the air degradation half-life are of primary importance. The air half-life is only of importance for chemicals that are strongly volatilized from surface waters. Residence times in sediments, for the chemicals addressed here, are predominantly controlled by intermedia transport and, hence, degradation half-lives in sediments are typically not important (burial and intermedia transport rates often controlling). Contaminant concentrations in soils for emissions to water will usually be negligible in the context of human exposure (assuming irrigation of crops does not result in high contaminant concentrations).

Table A4 summarizes the fate and exposure model calculations. The results used directly in the damage factor calculations are presented in the form of intake fractions (the fraction of the chemical emission that results in intake by the human population). For metals/metal compounds emitted to surface waters, intake via inhalation will be negligible (methyl mercury is a key exception, but this is not addressed in this pilot study). For the organic chemicals, the intake fraction associated with inhalation can be up to an order of magnitude lower than that for oral intake. This may therefore be relevant if humans are more sensitive to inhalation exposure for a given substance. Intake is predominantly associated with drinking water and fish.[5] The last three columns in Table A4 indicate the fate of the chemical from the water compartment. For the organic chemicals, degradation is often dominant. In some cases, the fraction transferred to air can be high as most of the chemical transferred to air is subsequently removed *via* degradation. Metals and metal compounds are often removed from the environment *via* transfer to sediments and burial, or advection to oceanic water.[6]

Table A5 presents the results of a detailed survey of toxicological data for the 30

[5] Additional input of measured bioaccumulation factors for metals and metal compounds is desirable to estimate intake associated with fish consumption for these compounds.

[6] The current model does not include human exposure pathways that are indirectly associated with contaminants in sediments, which may result in underestimating human intakes for persistent chemicals.

chemicals (and chemical groups). As far as possible the data are from IRIS, ATSDR, and US EPA Region 9 – in that order of preference. These sources represent the best toxicological database to date. The final Effective Dose 10 (ED_{10}), as used in the damage calculations, the associated calculation assumptions, the principle study, and the critical effect in this study are also summarized. Both oral and inhalation data were collected, as far as possible, for organic chemicals. For metals, emphasis was placed on oral exposure – although inhalation toxicological data are given where found. Additionally, while this study focused only on non-cancer effects, data relating to the likelihood of cancer effects are summarized.

While the critical effects are reported in Table A5, these data do not, in all cases, provide a full summary of all the likely effects associated with these compounds – particularly at low concentrations and in the context of complex mixtures. Disability Adjusted Life Years per incidence were based here on a default value of 0.67 DALYs per incidence (DALYp) and not estimated based on the endpoint information provided.

RESEARCH NEEDS

Since the utility of the LCA-DfE models is intended for GM's global operations, additional sets of regional damage factors for human health should be developed. The specific focused areas to improve the LCA-DfE decision model are:

A) Assess direct impacts of emissions from manufacturing processes using available material flow data, TRI, and energy use data, B) Compare the direct impacts of automotive industry, using data available in IO analysis tools (Sylvatica at Carnegie-Mellon University), C) Assess direct and indirect impacts of automotive industry using available IO analysis tools, and, D) Build a model for upstream and downstream model using data from task A and linking it with available IO analysis tools.

This new study should enhance the environmental management system and the environmental impact assessment methods. The transformation of the present version of Eco-Indicator'99 report to a practical database with a more global perspective will be

essential:

1. Recalculate all damage factors for North American conditions (including different landscape parameters, climate, population, food production, *etc.*) or extending the IMPACT 2002 model to a global scale.
2. Update the method based on best available evidence in the scientific literature and new developments (RECIPE, Japanese Eco-Indicator, traffic noise). Further, include here additional stressors that may be relevant for GM.
3. Provide a conceptual overview on changes in priorities when applied to other world regions (e.g., Latin America, Asia, Japan, Australia).
4. Apply the metrics in a comprehensive environmental health decision making software tool, specific to GM.

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APPENDIX

Table A1 Overall results for 30 chemicals

Table A2 Metals fate input data

Table A3 Organic chemicals fate input data

Table A4 Intake fraction results

Table A5 Toxicological survey data

TABLE A1: OVERALL RESULTS FOR 30 CHEMICALS

Chemical CAS#	Name	Non-cancer characterisation factor (cumulative risk/kg)	Non-cancer damage factor (DALY/kg)	Oral Intake fraction for water emission	Inhalation intake fraction for water emission	Oral non-cancer dose-response (Beta) risk/kg	Inhalation non-cancer dose-response (Beta) risk/kg	*Cancer damage factor (DALY/kg)*	Inhalation/oral Damage Fraction	Oral drinking water/oral total intake	Most important pathway	Square geometric standard deviation for fate and exposure analysis	Total square geometric standard deviation	Upper 95th percentile bound non-cancer damage factor (DALY/kg)	Lower 95th percentile bound non-cancer damage factor (DALY/kg)
95-63-6	1,2,4-trimethylbenzene	1.8E-07	**1.2E-07**	1.1E-05	1.3E-07	1.3E-02	3.7E-01		3.7E-01	6.3E-01	drinking water	2	27.9	3.4E-06	4.4E-09
822-06-0	HDI (1,6-Hexamethylene diisocyanate)	7.6E-04	**5.1E-04**	5.7E-06	1.0E-07	1.3E+02	1.3E+02		1.8E-02	8.4E-01	drinking water	2	27.9	1.4E-02	1.8E-05
78-93-3	MEK (Methyl Ethyl Ketone)	8.2E-10	**5.5E-10**	2.0E-06	7.5E-08	4.2E-04	4.4E-05		4.0E-03	1.0E+00	drinking water	2	27.9	1.5E-08	2.0E-11
7782-49-2	Selenium and compounds	2.2E-04	**1.5E-04**	8.7E-05	0	2.5E+00			0	1.5E-01	food	80	235.0	3.4E-02	6.2E-07
75-09-2	dichloromethane (methylene chloride)	3.4E-07	**2.3E-07**	8.6E-06	3.3E-06	3.8E-02	4.2E-03	*1.2E-07*	4.3E-02	1.0E+00	drinking water	2	27.9	6.4E-06	8.2E-09
75-01-4	vinyl chloride	1.7E-05	**1.1E-05**	6.6E-06	3.6E-07	2.5E+00	5.2E-02		1.1E-03	9.9E-01	drinking water	2	27.9	3.1E-04	4.0E-07
7440-66-6	Zinc and compounds	2.5E-05	**1.7E-05**	4.1E-05	0	6.2E-01			0	2.2E-01	food	80	235.0	4.0E-03	7.2E-08
7440-50-8	Cu	3.8E-07	**2.6E-07**	1.6E-05	0	2.5E-02			0	7.1E-01	drinking water	20	83.5	2.1E-05	3.1E-09
7440-47-3	Chromium IV (18540-29-9)	3.8E-07	**2.5E-07**	7.7E-06	0	4.9E-02	1.5E+00		0	8.0E-01	drinking water	20	83.5	2.1E-05	3.0E-09
7440-43-9	Cadmium	7.7E-05	**5.2E-05**	1.0E-05	0	7.5E+00			0	8.4E-01	drinking water	20	83.5	4.3E-03	6.2E-07
7440-41-7	Beryllium and compounds	1.0E-06	**6.7E-07**	1.6E-05	0	6.4E-02	3.3E+01		0	8.5E-01	drinking water	20	83.5	5.6E-05	8.0E-09
7440-39-3	Barium and compounds	6.4E-06	**4.3E-06**	3.6E-05	0	1.8E-01			0	3.7E-01	drinking water	20	83.5	3.6E-04	5.1E-08
7440-02-0	Nickel, soluble salts (no CAS#)	8.0E-07	**5.4E-07**	1.8E-05	0	4.5E-02	4.3E+00		0	7.2E-01	drinking water	20	83.5	4.5E-05	6.4E-09
7439-97-6	Mercury (inorganic)	2.9E-04	**2.0E-04**	9.1E-05	0	3.2E+00	7.2E+01		0	8.6E-02	food	80	235.0	4.6E-02	8.4E-07
7439-92-1	Lead and compounds (inorganic)	4.1E-06	**2.7E-06**	1.7E-05	0	2.5E-01	2.5E-01		0	2.2E-01	food	80	235.0	6.5E-04	1.2E-08
7429-90-5	Aluminium	1.4E-07	**9.7E-08**	1.8E-05	0	7.8E-03			0	7.2E-01	drinking water	80	235.0	2.3E-05	4.1E-10
71-43-2	benzene	2.0E-06	**1.3E-06**	4.2E-06	9.1E-08	2.1E-01	1.2E+01	*2.4E-07*	1.2E+00	9.8E-01	inhalation	6	41.1	5.5E-05	3.2E-08
67-56-1	methanol	2.9E-09	**2.0E-09**	2.0E-06	1.8E-08	1.5E-03			0	1.0E+00	drinking water	2	27.9	5.5E-08	7.1E-11
62-53-3	aniline	4.0E-07	**2.7E-07**	4.4E-06	2.0E-09	8.9E-02	1.3E-01	*2.2E-07*	6.5E-04	1.0E+00	drinking water	2	27.9	7.4E-06	9.5E-09
57-55-6	propylene glycol	1.6E-10	**1.0E-10**	5.0E-06	2.3E-13	3.1E-05	7.5E-02		1.1E-04	1.0E+00	drinking water	2	27.9	2.9E-09	3.7E-12
56-23-5	carbon tetrachloride	9.9E-06	**6.6E-06**	9.2E-06	2.4E-05	1.0E+00	1.4E-02	*1.1E-05*	3.5E-02	9.4E-01	drinking water	2	27.9	1.8E-04	2.4E-07
1746-01-6	2,3,7,8 TCDD	2.4E+03	**1.6E+03**	4.7E-04	2.5E-08	5.1E+06		*9.7E+01*	0.0E+00	8.1E-03	food	8	47.6	7.7E+04	3.4E+01
50-00-0	formaldehyde	3.4E-08	**2.3E-08**	2.0E-06	2.1E-10	1.5E-02	2.2E+01		1.5E-01	1.0E+00	drinking water	2	27.9	6.4E-07	8.3E-10
1634-04-4	MTBE	7.5E-08	**5.0E-08**	6.7E-06	1.4E-07	1.1E-02	5.0E-04		9.4E-04	1.0E+00	drinking water	2	27.9	1.4E-06	1.8E-09
1330-20-7	Xylene (all isomers)	1.1E-08	**7.1E-09**	7.2E-06	1.6E-07	1.2E-03	1.1E-02		1.9E-01	8.6E-01	drinking water	2	27.9	2.0E-07	2.6E-10
11097-69-1	PCB 1254 (under Aroclor 1254)	1.1E-01	**7.2E-02**	8.7E-04	2.1E-07	1.2E+02			0	4.3E-03	food	8	47.6	3.4E+00	1.5E-03
110-80-5	2-Ethoxyethanol	1.1E-08	**7.3E-09**	5.7E-06	4.4E-10	1.9E-03	1.9E-03		7.7E-05	1.0E+00	drinking water	2	27.9	2.0E-07	2.6E-10
108-88-3	toluene	2.4E-08	**1.6E-08**	7.2E-06	1.4E-07	3.3E-03	5.5E-03	*9.4E-08*	3.3E-02	9.4E-01	drinking water	2	27.9	4.6E-07	5.9E-10
107-21-1	Ethylene glycol	2.2E-09	**1.5E-09**	2.0E-06	8.5E-11	1.1E-03			0	1.0E+00	drinking water	2	27.9	4.2E-08	5.4E-11
106-99-0	1,3 butadiene	3.5E-07	**2.3E-07**	4.2E-06	3.5E-08	8.3E-02	8.3E-02		8.4E-03	9.8E-01	drinking water	2	27.9	6.5E-06	8.4E-09
101-68-8	MDI (Monomeric Methyl Diphenyl Diisocyanate)	1.3E-05	**8.7E-06**	6.6E-06	1.8E-10	2.0E+00	2.0E+00		2.7E-05	8.5E-02	food	8	47.6	4.1E-04	1.8E-07
100-42-5	styrene	1.5E-09	**1.0E-09**	4.7E-06	2.9E-08	3.0E-04	3.8E-03		8.0E-02	8.8E-01	drinking water	2	27.9	2.8E-08	3.7E-11
100-41-4	ethylbenzene	5.9E-08	**4.0E-08**	7.8E-06	1.4E-07	7.6E-03	3.0E-04	*6.1E-08*	6.9E-04	8.6E-01	drinking water	2	27.9	1.1E-06	1.4E-09

TABLE A2: METALS FATE INPUT DATA

CAS#	Molec. Weight	Suspended solids - water part coeff (L-water/Kg-solids)	Sediment solids - water part coeff (L-water/Kg-solids)	Soil solids - water part coeff (L-water/Kg-solids)	particle - gas part coeff (dimensionless)	BCF (kg-water/kg-fish)	B_milk [day/L]	B_meat [day/kg]	B_eggs [day/kg]	Bulk plant-soil concentration ratio [m3-soil/kg-plant]
7429-90-5	30	1,51E+03	1,00E+03	1,85E+02	1,00E+12	100	2,0E-04	1,5E-03	2,0E-04	3,13E-06
7440-39-3	137	1349	1000	60	1,0E+12	216	3,4E-04	1,0E-04	3,4E-04	3,90E-07
7440-41-7	9	851	603	38	1,0E+12	19	8,8E-07	3,0E-06	8,8E-07	2,34E-06
7440-43-9	112	128825	85114	200	1,0E+12	38	1,5E-03	5,0E-03	1,5E-03	1,17E-05
7440-47-3	52	288403	190546	110	1,0E+12	40	1,1E-03	9,2E-03	1,1E-03	1,56E-07
7440-50-8	64	50118	33884	221	1,0E+12	120	1,7E-03	1,3E-02	1,7E-03	7,80E-06
7439-97-6	201	170000	110000	170	2,5E+09	3030	4,6E-04	1,6E-03	4,6E-04	1,17E-06
7440-02-0	59	7943	5248	359	1,0E+12	87	9,7E-04	2,0E-03	9,7E-04	5,46E-06
7439-92-1	207	650000	430000	1905	1,0E+12	500	2,5E-04	1,0E-03	2,5E-04	7,80E-08
7782-49-2	79	589	417	20	1,0E+12	500	3,9E-03	1,3E-02	3,9E-03	7,80E-06
7440-66-6	65	109648	72444	334	1,0E+12	1000	9,7E-03	1,2E-02	9,7E-03	7,80E-06

TABLE A3: ORGANIC CHEMICALS FATE INPUT DATA

CAS#	H (PA m3 mol-1)	Source	Log Kow	Source	air (days)	Source	water (days)	Source	soil (days)	Source	sediment (days)	Source	Molecular Weight
63-6	6.1E+02	LTOX	3.78	LTOX	7.1E-01	ckay et al. Handbook	2.3E+01	ckay et al. Handbook	7.1E+01	ckay et al. Handbook	2.3E+02	Mackay et al. Handbook	1.2E+02
-99-0	2.6E+05	ES	1.99	ES	2.1E-01	ckay et al. Handbook	7.1E+00	ckay et al. Handbook	2.3E+01	ckay et al. Handbook	7.1E+01	Mackay et al. Handbook	5.4E+01
07-31-9	1.5E+00	LTOX	6.10	LTOX	7.1E+00	ckay et al. Handbook	2.3E+01	ckay et al. Handbook	7.1E+02	ckay et al. Handbook	2.3E+03	Mackay et al. Handbook	3.2E+02
-80-5	1.2E-02	LTOX	-0.32	LTOX	1.2E+00	vard & Boethling Handbook	1.1E+01	LTOX	1.8E+01	LTOX	4.4E+01	CALTOX	9.0E+01
53-3	1.9E-01	LTOX	0.90	LTOX	2.1E-01	ckay et al. Handbook	7.1E+00	ckay et al. Handbook	7.1E+00	ckay et al. Handbook	7.1E+01	Mackay et al. Handbook	9.3E+01
43-2	5.9E+02	ES	2.17	ES	7.1E-01	ckay et al. Handbook	7.1E+00	ckay et al. Handbook	2.3E+01	ckay et al. Handbook	7.1E+01	Mackay et al. Handbook	7.8E+01
23-5	3.2E+03	ES	2.64	ES	7.1E+02	ckay et al. Handbook	7.1E+01	ckay et al. Handbook	2.3E+02	ckay et al. Handbook	7.1E+02	Mackay et al. Handbook	1.5E+02
09-2	2.9E+02	ES	1.25	ES	7.1E+01	ckay et al. Handbook	7.1E+01	ckay et al. Handbook	2.3E+02	ckay et al. Handbook	7.1E+02	Mackay et al. Handbook	8.5E+01
-41-4	8.1E+02	ES	3.13	ES	7.1E-01	ckay et al. Handbook	2.3E+01	ckay et al. Handbook	7.1E+01	ckay et al. Handbook	2.3E+02	Mackay et al. Handbook	1.1E+02
-21-1	6.1E-03	LTOX	-1.36	LTOX	2.3E+00	ckay et al. Handbook	2.3E+00	ckay et al. Handbook	2.3E+00	ckay et al. Handbook	7.1E+00	Mackay et al. Handbook	6.2E+01
00-0	3.2E-02	ES	0.35	ES	2.1E-01	ckay et al. Handbook	2.3E+00	ckay et al. Handbook	2.3E+00	ckay et al. Handbook	7.1E+00	Mackay et al. Handbook	3.0E+01
-06-0	4.9E+00	WIN (Bond)	3.20	YSPROP Database	2.0E+00	WIN (Estimated)	8.7E+00	of USM and HYDROWIN	8.7E+00	mated (1*water)	3.5E+01	Estimated (4*soil)	1.7E+02
-68-8	9.1E-02	WIN (Bond)	5.22	YSPROP Database	9.2E-01	PWIN	6.3E-01	vard & Boethling Handbook	6.3E-01	vard & Boethling Handbook	3.5E+01	Estimated (4*soil)	2.5E+02
93-3	3.6E+00	LTOX	0.29	LTOX	7.1E+00	ckay et al. Handbook	2.3E+00	ckay et al. Handbook	2.3E+00	ckay et al. Handbook	2.3E+01	Mackay et al. Handbook	7.2E+01
56-1	4.5E-01	LTOX	-0.77	LTOX	7.1E+00	ckay et al. Handbook	2.3E+00	ckay et al. Handbook	2.3E+00	ckay et al. Handbook	7.1E+00	Mackay et al. Handbook	3.2E+01
4-04-4	7.0E+01	LTOX	0.94	LTOX	7.1E-01	ckay et al. Handbook	2.3E+01	ckay et al. Handbook	2.3E+01	ckay et al. Handbook	7.1E+01	Mackay et al. Handbook	8.8E+01
97-69-1	1.8E+01	LTOX	6.41	LTOX	6.6E+01	LTOX	2.8E+02	LTOX	9.4E+02	LTOX	1.3E+04	CALTOX	3.3E+02
55-6	1.3E-05	WIN (Group)	-0.92	YSPROP Database	8.3E-01	PWIN	8.7E+00	of USM and HYDROWIN	8.7E+00	mated (1*water)	3.5E+01	Estimated (4*soil)	7.6E+01
-42-5	2.8E+02	ES	3.05	ES	2.1E-01	ckay et al. Handbook	7.1E+00	ckay et al. Handbook	2.3E+01	ckay et al. Handbook	7.1E+01	Mackay et al. Handbook	1.0E+02
-88-3	9.2E+02	ES	2.69	ES	7.1E-01	ckay et al. Handbook	2.3E+01	ckay et al. Handbook	7.1E+01	ckay et al. Handbook	2.3E+02	Mackay et al. Handbook	9.2E+01
01-4	2.2E+03	ES	1.38	ES	2.3E+00	ckay et al. Handbook	2.3E+01	ckay et al. Handbook	7.1E+01	ckay et al. Handbook	2.3E+02	Mackay et al. Handbook	6.3E+01
0-20-7	6.9E+02	LTOX	3.11	LTOX	9.7E-01	vard & Boethling Handbook	1.8E+01	LTOX	1.5E+01	LTOX	2.7E+02	Howard & Boethling Handbook	1.1E+02

TABLE A4: INTAKE FRACTION RESULTS

Chemical		Oral Intake Fraction - Production Based (dimensionless)	Inhalation Intake Fraction -Production Based (dimensionless)	Water Concentration (kg/m3)	Oral Intake via Drinking Water (dimensionless)	Oral Intake via Fish (dimensionless)
95-63-6		1,06E-05	1,32E-07	2,38E-10	6,73E-06	3,87E-06
106-99-0		4,17E-06	3,51E-08	1,46E-10	4,1E-06	7,06E-08
1746-01-6		0,000472	2,48E-08	2,59E-10	3,84E-06	0,000464
110-80-5		5,68E-06	4,35E-10	2,01E-10	5,68E-06	1,06E-09
62-53-3		4,44E-06	2,05E-09	1,57E-10	4,43E-06	9,02E-09
71-43-2		4,24E-06	9,13E-08	1,47E-10	4,14E-06	1,01E-07
56-23-5		9,17E-06	2,44E-05	2,94E-10	8,63E-06	5,35E-07
75-09-2		8,56E-06	3,33E-06	2,89E-10	8,52E-06	3,48E-08
100-41-4		7,81E-06	1,37E-07	2,37E-10	6,72E-06	1,08E-06
107-21-1		2E-06	8,53E-11	7,14E-11	2E-06	4,87E-11
50-00-0		2E-06	2,08E-10	7,13E-11	2E-06	1,38E-09
822-06-0		5,72E-06	1,02E-07	1,7E-10	4,79E-06	8,81E-07
101-68-8		6,61E-06	1,81E-10	2,26E-11	5,63E-07	6,02E-06
78-93-3		1,97E-06	7,53E-08	7,02E-11	1,97E-06	1,21E-09
67-56-1		1,99E-06	1,77E-08	7,11E-11	1,99E-06	1,54E-10
1634-04-4		6,73E-06	1,39E-07	2,37E-10	6,72E-06	1,48E-08
11097-69-1		0,000874	2,15E-07	3,5E-10	3,74E-06	0,000833
57-55-6		4,99E-06	2,25E-13	1,77E-10	4,99E-06	2,88E-10
100-42-5		4,73E-06	2,92E-08	1,48E-10	4,16E-06	5,7E-07
108-88-3		7,15E-06	1,42E-07	2,36E-10	6,7E-06	4,55E-07
75-01-4		6,65E-06	3,55E-07	2,34E-10	6,61E-06	3,46E-08
1330-20-7		7,16E-06	1,6E-07	2,19E-10	6,19E-06	9,64E-07
7429-90-5		1,85E-05		3,79E-10	1,32E-05	5,17E-06
7440-39-3		3,61E-05		3,79E-10	1,32E-05	2,27E-05
7440-41-7		1,56E-05		3,79E-10	1,33E-05	2,38E-06
7440-43-9		1,03E-05		3,76E-10	8,71E-06	1,07E-06
7440-47-3		7,66E-06		3,74E-10	6,13E-06	1,06E-06
7440-50-8		1,56E-05		3,77E-10	1,1E-05	3,83E-06
7439-97-6		9,13E-05		3,75E-10	7,85E-06	8,33E-05
7440-02-0		1,79E-05		3,78E-10	1,29E-05	4,67E-06
7439-92-1		1,65E-05		3,73E-10	3,67E-06	1,27E-05
7782-49-2		8,75E-05		3,79E-10	1,33E-05	7,27E-05
7440-66-6		4,11E-05		3,76E-10	9,18E-06	2,87E-05

TABLE A5: TOXICOLOGICAL SURVEY DATA

CAS#	Name	Adopted in study	Source (for IRIS, only date of extraction is given. IRIS data are used preferentially here.)	Route	ED10 (mg/kg-day	ED10 Calculation	Critical Effect	Experimental dose	principle study (references can be found in the publication mentioned in column "Source"))	Confidence in study (study, database, & measure, unless otherwise stated)	Weight of Evidence (carcinogenic)
7429-90-5	Aluminium	y	ATSDR (May 1999)	Oral	7.2E+00	mouse-human - /13, NOAEL to ED10: *1.5	Neurotoxicity	NOAEL: 62 mg/kg-day	mouse, 6 weeks, Golub MS, Donald JM, Gershwin ME, Keen CL. 1989. Effects of aluminum ingestion on spontaneous motor activity of mice. Neurotoxicol Teratol. 11: 231-235.		
7440-39-3	Barium and compounds	y	IRIS (August 2002)	Oral	3.2E-01	no sub-chronic to chronic factor used in original study, NOAEL to ED10: *1.5	No adverse effect (other effect: Increased kidney weight. Increased kidney weight.)	NOAEL (ADJ): 0.21 mg/kg-day	Subchronic human study and community exposure study, Wones et al., 1990; Brenniman and Levy, 1984	Medium	D (Not classifiable as to human carcinogenicity)
7440-41-7	Beryllium and compounds	y	IRIS (August 2002)	Oral	8.8E-01	dog-human: /1.6	Small intestinal lesions	BMD10: 0.46 mg/kg-day, Maximum likelihood estimate = 1.4 mg/kg-day	Dog dietary study, Morgareidge et al., 1976	Study - Medium, others low/medium	B1 (Probable human carcinogen - based on limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals)
7440-41-7	Beryllium and compounds	y	IRIS (August 2002)	Inhalation	1.7E-03	20 m3/person-day, 70 kg/person, LOAEL to ED10: *0.3	Beryllium sensitization and progression to CBD	LOAEL (HEC): 0.02 mg/m3	Occupational study, Kreiss et al., 1996	Medium	
7440-41-7	Beryllium and compounds	y	ATSDR (Sept 2000)	Oral	9.4E-02	dog-human: /1.6, NOAEL to ED10: *1.5	One dog in the 1 mg beryllium/kg/day group died after 70 weeks. Ulcerative gastrointestinal lesions were observed in this animal. No beryllium-related hematological, serum chemistry, urinalysis, organ weight, or body weight alterations were observed in any beryllium-exposed groups. No other histological alterations were observed.	NOAEL: 0.1 mg beryllium/kg/day; gastrointestinal effects	Groups of five male and live female Beagle dogs were fed a diet containing 0, 5, or 50 ppm beryllium as beryllium sulfate tetrahydrate (0,0. 1, or 1 mg beryllium/kg/day for males and 0,0.2, or 1 mg beryllium/kg/day for females) for 172 weeks. (Morgareidge et al. 1976)		
7440-43-9	Cadmium	y	IRIS (August 2002)	Oral	7.5E-03	NOAEL to ED10: *1.5	Significant proteinuria	NOAEL: 0.005 mg/kg-day	Human studies involving chronic exposures, U.S. EPA, 1985	database and measure - high	B1 (Probable human carcinogen - based on limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals)
7440-43-9	Cadmium		ATSDR (March 1999)	Oral	3.2E-03	NOAEL to ED10: *1.5	Renal damage (proteinuria)	NOAEL: 0.0021 mg/kg/day	Human - 1850 exposed and 294 unexposed inhabitants in the Ishikawa prefecture. Nogawa et al. (1989)		

7440-47-3	Chromium (11115-74-5)		ATSDR (Sept 2000)	Inhalation	4.3E-05	20 m3/person-day, 70 kg/person, , LOAEL to ED10: *0,3	Nasal irritation (p<0.05), mucosal atrophy (p<0.05), and ulceration (p<0.01), and decreases in spirometric parameters (forced vital capacity, forced expired volume in 1 second, and forced mid-expiratory flow) were observed in workers occupationally exposed to 0.002 mg chromium(VI)/m 3 as chromic acid with a median exposure period of 2.5 years. About 60% of the exposed subjects were smokers, but no consistent association between exposure and cigarette smoking was observed. Short-term peak exposures to chromic acid correlated better with nasal septum damage than with 8-hour mean concentrations.	LOAEL: 0.002 mg chromium(VI)/m 3 , adjusted to 0.0005 mg chromium(VI)/m 3 for continuous exposure, for respiratory effects.	Eighty-five male and 19 female chrome plating workers exposed to chromic acid were assessed for nose, throat, and chest symptoms, were inspected for effects in nasal passages, and were given pulmonary function tests. (Lindberg E, Hedenstierna G. 1983. Chrome plating: Symptoms, findings in the upper airways, and effects on lung function. Arch Environ Health 38:367-374.)		
7440-47-3	Chromium IV (18540-29-9)	y	IRIS (Sept 1998)	Oral	1.1E+00	sub-chronic - chronic: /3.3, NOAEL to ED10: *1.5	Not reported	NOAEL (ADJ): 2.5 mg/kg-day	Rat, 1-year drinking water study, MacKenzie et al., 1958	Low	D (Not classifiable as to human carcinogenicity) (Oral route)
7440-47-3	Chromium IV (18540-29-9) particulates	y	IRIS (Sept 1998)	Inhalation	3.8E-02	Rat to human dosimetric factor (IRIS): *2.1, sub-chronic-chronic: /3.3	Lactate dehydrogenase in bronchioalveolar lavage fluid	BMC10 (ADJ): 0.034 mg/m3, maximum likelihood estimates of 0.036 - 0.078 mg/m3	Rat subchronic study, Glaser et al., 1990; Malsch et al., 1994	Medium (database not available)	A (Human Carcinogen) (Inhalation route)
7440-50-8	Cu		IRIS (August 2002)								D (Not classifiable as to human carcinogenicity)
7440-50-8	Cu	y	ATSDR	Oral	2.3E+00	sub-chronic - chronic: /3.3, rat to human: /6, LOAEL to ED10: *0.3	Essential element, but liver and kidneys possible target organs.	Insufficient robust data to estimate regulatory guidline. Majority of subchronic studies appear to report a LOAEL approx. 150 mg CU/kg -day	Rat (subchronic - up to 18 weeks)		
7439-97-6	Elemental mercury (excludes methyl mercury)	y	IRIS (August 2002)	Inhalation	7.7E-04	20 m3/person-day, 70 kg/person, LOAEL to ED10: *0.3	Hand tremor; increases in memory disturbances; slight subjective and objective evidence of autonomic dysfunction	LOAEL (ADJ): 0.009 mg/m3	Human occupational inhalation studies, Fawer et al., 1983; Piikivi and Tolonen, 1989; Piikivi and Hanninen, 1989; Piikivi, 1989; Ngim et al., 1992; Liang et al., 1993	Medium	D (Not classifiable as to human carcinogenicity)
7439-97-6	Mercury (inorganic)	y	ATSDR (June 2001)	Oral	1.7E-02	sub-chronic - chronic: /3.3, rat to human: /6, NOAEL to ED10: *1.5	Renal effects	NOAEL: 0.23 mg Hg/kg/day	Fischer 344 rats (10/sex/group) were administered 0, 0.23, 0.46, 0.93, 1.9, or 3.7 mg Hg/kg/day as mercuric chloride in deionized water by oral gavage once daily 5 days per week for 26 weeks. (NTP. 1993. NTP technical report on the toxicology and carcinogenesis studies of mercuric chloride (CAS no. 7487-94-7) in F344/N rats and B6C3F1 mice (gavage studies). NTP TR408.)		
7439-97-6	Mercury (metallic vapour)		ATSDR (June 2001)	Inhalation	5.3E-04	20 m3/person-day, 70 kg/person, LOAEL to ED10: *0.3	increased frequency of tremors	LOAEL (adj): 0.0062 mg/m3	Hand tremors were measured in 26 male workers exposed to metallic mercury and 25 control males working in the same facilities, but not exposed to mercury. (Fawer RF, de Ribaupierre Y, Guillemin MP, et al. 1983.Measurement of hand tremor induced by industrial exposure to metallic mercury. British Journal of Industrial Medicine 40:204-208.)		
7440-02-0	Nickel	y	ATSDR (May 1997)	Inhalation	1.3E-02	20 m3/person-day, 70 kg/person (rat to human already taken into account), NOAEL to ED10: *1.5	Active inflammation and lung fibrosis in rats	NOAEL: 0.03 mg/m3	Groups of 65 male and 65 female F344 rats were exposed to nickel sulfate hexahydrate at 0, 0.03, 0.06, and 0.11 mg nickel/m3 (MMAD = 1.8-3.1 µm) 6 hours/day, 5 days/week for 2 years. (NTP 1996c)		
7440-02-0	Nickel, soluble salts (no CAS#)	y	IRIS(Dec 1996)	Oral	1.3E+00	rat to human: /6, NOAEL to ED10: *1.5	Decreased body and organ weights	NOAEL: 5 mg/kg-day	Rat chronic oral study, Ambrose et al., 1976	Medium (low study)	
7439-92-1	Lead and compounds (inorganic)		IRIS (August 2002)								B2 (Probable human carcinogen - based on sufficient evidence of carcinogenicity in animals)

7439-92-1	Lead and compounds (inorganic)	y	ATSDR	(likely same, regardless route of exposure)	2.3E-01	rat to human: /6, NOAEL to ED10: *1.5	Multiple effects (decrease in ALAD activity at LOEL)	Lack of clear threshold and robust study data, so no regulatory measure proposed. "Most robust" chronic study NOAEL: 0.9 mg/kg-day	Rat - 2 year, ad. lib. (Azar et al. 1973)		
7782-49-2	Selenium and compounds	y	IRIS (August 2002)	Oral	2.3E-02	NOAEL to ED10: *1.5	Clinical selenosis	NOAEL: 0.015 mg/kg-day	Human epidemiological study, Yang et al., 1989b	Study - medium, others - High	D (Not classifiable as to human carcinogenicity)
7782-49-2	Selenium (elemental)		ATSDR (March 2001)	Oral	2.3E-02	NOAEL to ED10: *1.5	Nail disease (selenosis)	NOAEL: 0.015 mg/kg-day	Data were collected on selenium levels in the diet, blood, nails, hair, urine, and milk of residents, and the incidence of clinical symptoms of selenosis (morphological changes in finger nails) was compared with dietary intake of selenium and selenium levels in blood. (Yang G, Zhou R. 1994. Further observations on the human maximum safe dietary selenium intake in a seleniferous area of China. J Trace Elem Electrolytes Health Dis 8:159-165.)		
13494-80-9	Tellurium		NIOSH, RTECS (August 2002)	Inhalation			lethal concentration (50% kill)	lethal concentration (50% kill) > 2420 mg/m3/4 hour	Acute rat, 4 hour		
13494-80-9	Tellurium		NIOSH, RTECS (August 2002)	Oral			lethal dose (50% kill)	lethal dose (50% kill) - mouse 20 mg/kg, rat 83 mg/kg	Acute rat and mouse studies		
13494-80-9	Tellurium						Tellurium accumulates in the blood, liver, kidneys, lungs, thyroid and spleen. These organs may be affected by acute poisoning. Chronic exposure may resultin respiratory depression and circulatory collapse. The NIOSH REL and OSHA PEL fortellurium are both 100 micrograms/cubic meter of air.	TLV and OSHA PEL similar to those of Selenium (located above tellurium in periodic table)			
7440-66-6	Zinc and compounds	y	IRIS (August 2002)	Oral	9.1E-02	sub-chronic - chronic: /3.3, LOAEL to ED10: *0,3	47% decrease in erythrocyte superoxide dismutase (ESOD) concentration in adult females after 10 weeks of zinc exposure	LOAEL: 1.0 mg/kg-day	Human diet supplement study, Yadrick et al., 1989	Medium	D (Not classifiable as to human carcinogenicity)
7440-66-6	Zinc		ATSDR(May 1994)	Oral	3.8E-01	sub-chronic - chronic: /3.3, NOAEL to ED10: *1.5	hematological effects (decreased hematocrit, serum ferritin, and erythrocyte dismutase)	NOAEL: 0.83 mg Zn/kg-day	Women given zinc gluconate supplements for 10 weeks (Yadrick et al. 1989).		
95-63-6	1,2,4-trimethylbenzene	y	US EPA Region 9 Superfund data Preliminary Remediation Goals (NCEA)	Oral	4.5E+00	RfD to ED10 conversion of Pennington et al. (2002b)		RfD: 5E-02 mg/kg-day			
95-63-6	1,2,4-trimethylbenzene	y	US EPA Region 9 Superfund data Preliminary Remediation Goals (NCEA)	Inhalation	1.5E-01	RfD to ED10 conversion of Pennington et al. (2002b)		RfD: 1.7E-03 mg/kg-day			
106-99-0	1,3 butadiene		IRIS (August 2002)								B2 (Probable human carcinogen - based on sufficient evidence of carcinogenicity in animals)
106-99-0	1,3 butadiene	y	ASTDR	Inhalation	6.8E-01	(ppm to mg/m3: *Mw/24.45 = 54/24.45), multiply by 5/7 and 6/24 to adjust for continuous duration. No mouse to human inhalation factor assumed, 20 m3/person-day, 70 kg/person, LOAEL to ED10: *0,3	Multiple effects	No standard proposed, as mortalitly observed in lowest robust bioassay. LOAEL (increased mortality) 20 ppm	Mouse, 65 weeks, 5 days per week, 6 hours per day (Melnick et al. 1989, 1990)		

106-99-0	1,3 butadiene	y	US EPA Region 9 Superfund data Preliminary Remediation Goals	Oral	6.8E-01			same as inhalation via route extrapolation (for cancer effects)			
1746-01-6	2,3,7,8 TCDD	y	ATSDR (Dec 1998)	Oral	1.1E-08	sub-chronic - chronic: /3.3 (no monkey to human factor), LOAEL to ED10: *0.3	Altered social behavior	LOAEL: 5 ppt dietary concentration is equivalent to a daily dose of 1.2 x 10 -4 µg/kg/day.	Groups of 8 female rhesus monkeys were fed a diet containing 0, 5, or 25 ppt 2,3,7,8-TCDD for a total of 16.2 ± 0.4 month (Schantz et al. 1992)		
110-80-5	2-Ethoxyethanol	y	IRIS (August 2002)	Inhalation	2.9E+01	20 m3/person-day, 70 kg/person (interspecies dosimetry extrapolation already taken into account), NOAEL to ED10: *1.5	Decreased testis weight, seminiferous tubule degeneration and decreased hemoglobin	NOAEL (HEC): 68 mg/m3	New Zealand White Rabbit subchronic toxicity study, Barbee et al., 1984	Medium	
110-80-5	2-Ethoxyethanol	y		Oral	2.9E+01	Assumed equivalent to inhalation.					
62-53-3	aniline	y	IRIS (August 2002)	Inhalation	4.4E-01	sub-chronic - chronic: /3.3, 20 m3/person-day, 70 kg/person, inhalation rat to human factor included, NOAEL to ED10: *1.5	Lack of toxicity (other effect: Mild spleen toxicity.)	NOAEL (HEC): 3.4 mg/m3	20-26 week inhalation rat, guinea pig and mouse study, Oberst et al., 1956	Low	B2 (Probable human carcinogen - based on sufficient evidence of carcinogenicity in animals)
62-53-3	aniline	y	US EPA Region 9 Superfund data Preliminary Remediation Goals (NCEA)	Oral	6.2E-01	RfD to ED10 conversion of Pennington et al. (2002b)		RfD: 7E-03 mg/kg-day			
71-43-2	benzene		IRIS (August 2002)								A (Human Carcinogen)
71-43-2	benzene	y	ATSDR (June 1997)	Inhalation	4.6E-03	sub-chronic - chronic: /3.3 (original study used mouse to human extrapolation based on ventilation rates - not adopted here, no adjustment made), (ppm to mg/m3: *78/24,45), 20 m3/person-day, 70 kg/person, LOAEL to ED10: *0.3	Increased rapid response time (Less Serious minimal LOAEL). No adverse effect on immunological/lymphoreticular indices, including spleen weight and bone marrow panel, or body, liver, or kidney weight. No effect on blood or brain acetylcholinesterase.	LOAEL (adj): 0.056 ppm	Forty adult Kunming male mice were divided into 4 groups (5/group) and exposed to 0, 0.78, 3.13 and 12.52 ppm benzene for 2 hours/day, 6 days/week for 30 days. (Li et al. 1992)		
71-43-2	benzene	y	US EPA Region 9 Superfund data Preliminary Remediation Goals (NCEA)	Oral	2.7E-01	RfD to ED10 conversion of Pennington et al. (2002b)		RfD: 3E-03 mg/kg-day			
56-23-5	carbon tetrachloride	y	IRIS (August 2002)	Oral	5.4E-02	sub-chronic - chronic: /3.3, rat to human: /6, NOAEL to ED10: *1.5	Liver lesions	NOAEL: 0.71 mg/kg-day	Subchronic rat gavage study, Bruckner et al., 1986	Study - high, others - medium	B2 (Probable human carcinogen - based on sufficient evidence of carcinogenicity in animals)
56-23-5	carbon tetrachloride	y	ATSDR (May 1994)	Inhalation	4.1E+00	sub-chronic - chronic: /3.3, 20 m3/person-day, 70 kg/person, no inhalation rat to human factor included, (ppm to mg/m3: *154/24.45), NOAEL to ED10: *1.5	Fatty degeneration was evident at concentrations of 10 ppm and cirrhosis occurred at 50 ppm.	NOAEL: 5 ppm	Rat - 173-205 days, 7 hrs per day, 5 days per week. (Adams et al. 1952). No studies found for chronic non-cancer effects, as not relevant compared to cancer effect incidences.		
56-23-5	carbon tetrachloride		ATSDR (May 1994)	Oral	5.4E-02	sub-chronic - chronic: /3.3, rat to human: /6, NOAEL to ED10: *1.5	Absence of detected adverse hepatic effects	NOAEL: 1 mg/kg-day To accound for the study's intermittent exposure (5 days/week), the MRL was derived using an adjusted NOAEL of 0.71 mg/kg/day.	Rats, 5 days per week for 12 weeks. Corn oil gavage. (Bruckner et al. 1986)		
75-09-2	dichloromethane (methylene chloride)	y	IRIS (August 2002)	Oral	1.5E+00	rat to human: /6, NOAEL to ED10: *1.5	Liver toxicity	NOAEL: 5.85 mg/kg-day	2-year rat drinking water bioassay (NOAEL for males), National Coffee Association, 1982	Study - high, others - medium	B2 (Probable human carcinogen - based on sufficient evidence of carcinogenicity in animals)

75-09-2	dichloromethane (methylene chloride)	y	ATSDR (July 2000)	Inhalation	1.3E+01	20 m3/person-day, 70 kg/person (interspecies blood-gas extrapolation already taken into account), (ppm to mg/m3: *85/24,45), NOAEL to ED10: *1.5	Liver histopathology in female rats	NOAEL(HEC): 8.9 ppm	Groups of 90 male and 108 female Sprague-Dawley rats were exposed to these concentrations for 6 hours/day, 5 days/week for 2 years. (Nitschke KD, Burek JD, Bell TJ, et al. 1988a. Methylene Chloride: A 2-year inhalation toxicity and oncogenicity study in rats. Fundam Appl Toxicol 11:60-67.)		
75-09-2	dichloromethane (methylene chloride)		ATSDR (July 2000)	Oral	1.5E+00	rat to human: /6, NOAEL to ED10: *1.5	Histopathology was only observed in the liver; therefore, the liver is the critical target organ. Marginal liver changes were only observed in mice at the highest dose level tested. Statistically significant cellular changes (hepatic foci, areas of cellular alterations) were observed in all dose groups in the rat except for the lowest.	NOAEL: 6 mg/kg/day	Fischer-344 rats (85/sex/dose) and B6C3F1 mice (50–200/sex/dose) were exposed to methylene chloride in deionized drinking water at target concentrations aimed at exposing rats to 0, 5, 50, 125, or 250 mg/kg/day and mice to 0, 60, 125, 185, and 250 mg/kg/day for 104 weeks. (Serota D, Thakur, AK, Ulland BM, et al. 1986a. A two year drinking water study of dichloromethane in rodents. I. Rats. Food Chem Toxicol 24:951-958.)		
100-41-4	ethylbenzene	y	IRIS (August 2002)	Oral	7.4E+00	sub-chronic - chronic: /3.3, rat to human: /6, NOAEL to ED10: *1.5	Liver and kidney toxicity	NOEL: 97.1 mg/kg-day	Rat subchronic to chronic oral bioassay, Wolf et al., 1956	Low	D (Not classifiable as to human carcinogenicity)
100-41-4	ethylbenzene	y	IRIS (August 2002)	Inhalation	1.9E+02	20 m3/person-day, 70 kg/person (interspecies dosimetry extrapolation already taken into account), NOAEL to ED10: *1.5	Developmental toxicity	NOAEL (HEC): 434 mg/m3	Rat and rabbit developmental inhalation studies, Andrew et al., 1981; Hardin et al., 1981	Low	
100-41-4	ethylbenzene		ATSDR (May 1999)	Inhalation	1.8E+02	20 m3/person-day, 70 kg/person, (interspecies dosimetry extrapolation already taken into account), (ppm to mg/m3: *106/24,45), NOAEL to ED10: *1.5	Developmental toxicity (skeletal anomolies)	NOAEL: 97ppm	Rats, Andrew et al. 1981		
107-21-1	Ethylene glycol	y	IRIS (August 2002)	Oral	5.0E+01	rat to human: /6, NOAEL to ED10: *1.5	Kidney toxicity	NOAEL: 200 mg/kg-day	Chronic rat oral feeding study, DePass et al., 1986a	High	
107-21-1	Ethylene glycol		ATSDR (Dec. 1995)	Oral	5.0E+01	rat to human: /6, NOAEL to ED10: *1.5	Renal toxicity	NOAEL: 200 mg/kg-day	Groups of 130 male and female rats fed diets for 24 months. (Woodside 1982, DePass et al. 1986)		
50-00-0	formaldehyde	y	IRIS (August 2002)	Oral	3.8E+00	rat to human: /6, NOAEL to ED10: *1.5	Reduced weight gain, histopathology in rats	NOAEL: 15 mg/kg-day	Rat 2-year bioassay, Til et al., 1989	Study - high, others - medium	B1 (Probable human carcinogen - based on limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals)
50-00-0	formaldehyde	y	ATSDR (April 1999)	Inhalation	2.5E-03	20 m3/person-day, 70 kg/person, (ppm to mg/m3: *30/24.45)	Clinical symptoms of mild irritation of the eyes and upper respiratory tract and mild damage to the nasal epithelium were observed in workers exposed for 10.4 years (range 1-36 years) to an average TWA concentration of 0.24 ppm (range: 0.04 to 0.4 ppm), LOAEL to ED10: *0.3	LOAEL: 0.24 ppm	Holmstrom et al. (1989c) examined histological changes in nasal tissue specimens from a group of 70 workers in a chemical plant that produced formaldehyde and formaldehyde resins for impregnation of paper, a group of 100 furniture factory workers working with particle board and glue components, and a nonexposed, control group of 36 office workers in the same village as the furniture factories. (Holmstrom M, Wilhelmsson B, Hellquist H, et al. 1989c. Histological changes in the nasa mucosa in persons occupationally exposed to formaldehyde alone and in combination with wood dust. Acta Otolaryngol (Stockh) 107:120-129.)		

50-00-0	formaldehyde		ATSDR (April 1999)	Oral	3.8E+00	rat to human: /6, NOAEL to ED10: *1.5	Gastrointestinal effects	NOAEL: 15 mg/Kg/day	Administered to groups of weanling SPF-bred rats (Cpb:WU; Wistar random) in their drinking water for 2 years. (Til HP, Woutersen RA, Feron VJ, Hollanders VHM, Falke HE, Clary JJ (1989). Two-year drinking-water study of formaldehyde in rats. Fd. Chem. Toxic 27: 77-86.)		
822-06-0	HDI (1,6-Hexamethylene diisocyanate)	y	IRIS (August 2002)	Inhalation	4.3E-04	20 m3/person-day, 70 kg/person (accounts for interspecies dosimetry adjustment), NOAEL to ED10: *1.5	Degeneration of olfactory epithelium	NOAEL (HEC): 0.001 mg/m3	Rat chronic inhalation study, Mobay, Inc., 1989	Study - high, others - medium	
822-06-0	HDI (1,6-Hexamethylene diisocyanate)		ATSDR (Feb 1998)	Inhalation	5.3E-04	multiply by 5/7 and 6/24 to adjust for continuous duration. No rat to human inhalation factor assumed, 20 m3/person-day, 70 kg/person, (ppm to mg/m3: *168/24,45), LOAEL to ED10: *0.3	Nasal cavity epithelial hyperplasia	LOAEL: 0.005 ppm	60 male and 60 female rats exposed for 5 days/week for 6 hours/day for 2 years. (Mobay Corporation 1989)		
822-06-0	HDI (1,6-Hexamethylene diisocyanate)	y	US EPA Region 9 Superfund data Preliminary Remediation Goals	Oral	4.3E-04			same as inhalation via route extrapolation			
101-68-8	MDI (Monomeric Methyl Diphenyl Diisocyanate)	y	IRIS (August 2002)	Inhalation	2.9E-02	20 m3/person-day, 70 kg/person (interspecies dosimetry extrapolation already taken into account)	Hyperplasia of olfactory epithelium	BMC10 (HEC): 0.06 mg/m3, Maximum likelihood estimate (HEC) = 0.1 mg/m3	Rat inhalation studies, Reuzel et al., 1990, 1994b	Study - high, others medium	
101-68-8	MDI (Monomeric Methyl Diphenyl Diisocyanate)	y	US EPA Region 9 Superfund data Preliminary Remediation Goals	Oral	2.9E-02			same as inhalation via route extrapolation			
78-93-3	MEK (Methyl Ethyl Ketone)	y	IRIS (August 2002)	Oral	1.3E+02	sub-chronic - chronic: /3.3, rat to human: /6, NOAEL to ED10: *1.5	Decreased fetal birth weight	NOAEL: 1771 mg/kg-day	Multigeneration/ developmental rat feeding study, Cox et al., 1975	Low	D (Not classifiable as to human carcinogenicity)
78-93-3	MEK (Methyl Ethyl Ketone)	y	IRIS (August 2002)	Inhalation	1.3E+03	20 m3/person-day, 70 kg/person (interspecies dosimetry extrapolation already taken into account), NOAEL to ED10: *1.5	Decreased fetal birth weight	NOAEL (HEC): 2978 mg/m3	Mouse developmental study, Schwetz et al., 1991; Mast et al., 1989	Study-medium, others - low	
67-56-1	methanol	y	IRIS (August 2002)	Oral	3.8E+01	sub-chronic - chronic: /3.3, rat to human: /6, NOAEL to ED10: *1.5	Increased SAP and SGPT, and decreased brain weight	NOEL: 500 mg/kg-day	Rat oral subchronic study, U.S. EPA, 1986	Database - low, others - medium	
1634-04-4	MTBE	y	IRIS (August 2002)	Inhalation	1.1E+02	20 m3/person-day, 70 kg/person (interspecies dosimetry extrapolation already taken into account), NOAEL to ED10: *1.5	Increased absolute and relative liver and kidney weights and increased severity of spontaneous renal lesions (females), increased prostration (females), and swollen periocular tissue (males and females)	NOAEL (HEC): 259 mg/m3	Chronic rat 24-month inhalation study, Chun et al., 1992	Medium	
1634-04-4	MTBE	y	ATSDR (July 1996)	Oral	5.1E+00	sub-chronic - chronic: /3.3, rat to human: /6, LOAEL to ED10: *0,3	Decreased BUN levels	LOAEL: 100 mg/kg/day	Groups of 10 male and 10 female Sprague-Dawley rats were treated by gavage with MTBE in corn oil at doses of 0, 100, 300, 900, and 1,200 mg/kg/day, 7 days/week for 90 days. (Robinson et al. 1990)		
1634-04-4	MTBE		ATSDR (July 1996)	Inhalation	1.1E+02	20 m3/person-day, 70 kg/person (interspecies dosimetry extrapolation already taken into account), (ppm to mg/m3: *88/24,45), NOAEL to ED10: *1.5	No increased incidence and severity of chronic progressive nephropathy	NOAEL: 400 ppm (71 ppm HEC)	Groups of 50 male and 50 female Fischer 344 rats were exposed to 0, 400, 3,000, or 8,000 ppm MTBE 6 hours/day, 5 days/week for up to 24 months. (Chun et al. 1992)		

11097-69-1	PCB 1254 (PCBs)		ATSDR (September 2000)	Oral	4.5E-04	sub-chronic - chronic: /3.3 (no monkey to human factor), LOAEL to ED10: *0.3	Decreased antibody response. Interpretation of the adversity of this effect is complicated by a lack of data on immunocompetence and the essentially inconclusive findings in the other tested end points; however, support for the 0.005 mg/kg/day LOAEL is provided by mild clinical manifestations of toxicity at the same dose. In other pertinent information section, eyelid and toe/finger nail changes were observed in some monkeys at doses as low as 0.005 mg/kg/day (Arnold et al.1993a).	LOAEL: 0.005 mg/kg/day	Groups of 16 female Rhesus monkeys self-ingested capsules containing Aroclor 1254 in a glycerol/corn oil mixture (1:1). (Tryphonas et al. 1989, 1991a)		
11097-69-1	PCB 1254 (under Aroclor 1254)	y	IRIS (August 2002)	Oral	4.5E-04	sub-chronic - chronic: /3.3 (no monkey to human factor), LOAEL to ED10: *0.3	Ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	LOAEL: 0.005 mg/kg-day	Monkey clinical and immunologic studies, Arnold et al., 1994a,b; Tryphonas et al., 1989, 1991a,b	Medium	
57-55-6	propylene glycol	y	ASTDR (Dec 1995)	Inhalation	7.4E-01	sub-chronic - chronic: /3.3, multiply by 5/7 and 6/24 to adjust for continuous duration. No rat to human inhalation factor assumed, 20 m3/person-day, 70 kg/person, LOAEL to ED10: *0.3	Nasel Hemorrhaging	LOAEL: 160 mg/m3	Rat - 19 males, 19 femals, 5 days per week, 6 hours per day, 13 weeks(Suber et al. 1989)		
57-55-6	propylene glycol	y	HEAST	Oral	1.8E+03	RfD to ED10 conversion of Pennington et al. (2002b)		RfD: 20 mg/kg-day			
100-42-5	styrene	y	IRIS (August 2002)	Oral	1.9E+02	dog-human: /1.6, NOAEL to ED10: *1.5	Red blood cell and liver effects	NOAEL: 200 mg/kg-day	Dog subchronic oral study, Quast et al., 1979 (565 days, 1 per day)	Medium	
100-42-5	styrene	y	IRIS (August 2002)	Inhalation	1.5E+01	20 m3/person-day, 70 kg/person (interspecies dosimetry extrapolation already taken into account), NOAEL to ED10: *1.5	CNS effects	NOAEL (HEC): 34 mg/m3	Occupational study, Mutti et al., 1984	Medium	
100-42-5	styrene		ATSDR(sept 1992)	Oral	1.5E+01	sub-chronic - chronic: /3.3, rat to human: /6, NOAEL to ED10: *1.5	Liver enzyme changes	NOAEL: 200 mg/kg-day (oral intermediate-duration RfD of 0.2 mg/kg/day)	Rat - 60 day, 6 days per week, 1 per day (Srivastava et al. 1982).		
100-42-5	styrene		ATSDR(sept 1992)	Inhalation	9.1E+00	20 m3/person-day, 70 kg/person, (ppm to mg/m3: *104/24,45), LOAEL to ED10: *0.3	Although the lung and liver are both affected by chronic exposure, neurological effects such as decreased short term memory or impaired visuomotor performance seem to be the most sensitive indicators of toxicity	LOAEL: 25 ppm	Chronic studies are available that investigated the adverse health effects of styrene on workers in the plastics industry. Study adopted (Mutti et al. 1984a)		
108-88-3	toluene	y	IRIS (August 2002)	Oral	1.7E+01	sub-chronic - chronic: /3.3, rat to human: /6, NOAEL to ED10: *1.5	Changes in liver and kidney weights (other effect: Degeneration of nasal epithelium.)	NOAEL: 223 mg/kg-day	13-week rat gavage study, NTP, 1989	Study - high, others - medium	D (Not classifiable as to human carcinogenicity)
108-88-3	toluene	y	IRIS (August 2002)	Inhalation	1.0E+01	20 m3/person-day, 70 kg/person, LOAEL to ED10: *0.3	Neurological effects (other effect: Degeneration of nasal epithelium.)	LOAEL (HEC): 119 mg/m3	Occupational study, Foo et al., 1990	Medium	
108-88-3	toluene		ATSDR (June 2000)	Oral	3.5E-02	sub-chronic - chronic: /3.3, mouse to human: /13, LOAEL to ED10: *0.3	Neurological effects	LOAEL: 5 mg/kg/day	Male CD-1 mice (5 per group) were administered toluene in their drinking water for a 28-day period. (Hsieh GC, Sharma RP, Parker RDR et al. 1990b. Evaluation of toluene exposure via drinking water on levels of regional brain biogenic monoamines and their metabolites in CD-1 mice. Ecotox Environ Safety 20: 175–184.)		
108-88-3	toluene		ATSDR (June 2000)	Inhalation	1.1E+01	20 m3/person-day, 70 kg/person, (ppm to mg/m3: *92/24,45), LOAEL to ED10: *0,3	Alcohol-and age-adjusted color vision impairment	LOAEL: 35 ppm	Three groups of Croatian workers were examined by means of interviews, medical examination, and color vision testing using the Lanthony 15 Hue desaturated panel in standard conditions. (Zavalic, M, Mandic, Z, Turk, R et al. 1998a. Quantitative assessment of color vision impairment in workers exposed to toluene. Am J Ind Med 32: 297-304.)		
75-01-4	vinyl chloride	y	IRIS (August 2002)	Oral	2.3E-02	rat to human: /6, NOAEL to ED10: *1.5	Liver cell polymorphism	NOAEL (HED): 0.09 mg/kg-day	Rat chronic feeding study, Til et al., 1983, 1991	Study - high, database medium/high. Measure - medium	A (Human Carcinogen)

75-01-4	vinyl chloride	y	IRIS (August 2002)	Inhalation	1.1E+00	20 m3/person-day, 70 kg/person, NOAEL to ED10: *1.5	Liver cell polymorphism	NOAEL (HEC): 2.5 mg/m3	Rat chronic feeding study, Til et al., 1983, 1991	Study - high, database medium/high. Measure - medium	
75-01-4	vinyl chloride	y	ATSDR (Oct 1996)	Oral	9.0E-04	rat to human: /6, LOAEL to ED10: *0.3	Increase in the number of hepatocellular basophilic foci indicating cellular alteration.	LOAEL: 0.018 mg/kg/day	Groups of 100 Wistar rats of each sex (except for highest dose group, which contained 50 of each sex) were administered vinyl-chloride-monomer-enriched polyvinyl chloride powder in feed 4 hours/day for 149 weeks. (Til et al. 1983, 1991)		
75-01-4	vinyl chloride	y	ATSDR (Oct 1996)	Inhalation	2.2E+00	20 m3/person-day, 70 kg/person (interspecies blood-gas extrapolation already taken into account), (ppm to mg/m3: *62/24,45), no sub-chronic to chronic extrapolation considered in original study, LOAEL to ED10: *0.3	Increases in liver-to-body weight ratio	LOAEL: 10 ppm	Groups of 75 adult male Wistar rats were exposed to 0, 10, 100, or 3,000 ppm vinyl chloride by inhalation 6 hours/day, 6 days/week for 12 months, with sacrifices at 3, 6, 12, and 18 months after initial exposure. (Bi et al. 1985)		
1330-20-7	Xylene (all isomers)	y	IRIS (August 2002)	Oral	4.5E+01	rat to human: /6, NOAEL to ED10: *1.5	Hyperactivity, decreased body weight and increased mortality (males)	NOAEL: 179 mg/kg-day	Chronic rat gavage study, NTP, 1986	Medium	D (Not classifiable as to human carcinogenicity)
1330-20-7	Xylene (all isomers)	y	ATSDR (August 1995)	Inhalation	5.2E+00	20 m3/person-day, 70 kg/person, (ppm to mg/m3: *106/24,45), LOAEL to ED10: *0.3	Neurological	LOAEL: 14 ppm	Human - average 7 years, 8 hours per day. (Uchida et al. 1993)		
1330-20-7	Xylene (all isomers)		ATSDR (August 1995)	Oral	1.1E+01	sub-chronic - chronic: /3.3, rat to human: /6, NOAEL to ED10: *1.5	Renal effects	NOAEL: 150 mg/kg-day	Rat, 90 day, 1 time per day. (Condie et al. 1988)		

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Dr. David Pennington joined the Life Cycle and Sustainable Development Group, headed by Professor Olivier Jolliet, in October 2000. His research continues to focus on the development and analysis of multimedia chemical fate, exposure and toxicological models to support the development of environmental comparison indicators (also known as characterization factors). Environmental indicators are used in the comparison of chemicals and emissions in application domains such as chemical screening, process design, the life cycle assessment (LCA) of products and sustainable development. Dr. Pennington is on the editorial board of the International Journal of Life Cycle Assessment and continues to be involved in external collaborations, such as SETAC's life cycle impact assessment (LCIA) working groups on the fate and exposure, human toxicological effects and ecological toxicological effects. His teaching interests include life cycle assessment, modelling the multimedia fate and exposure of chemicals in the environment and chemical risk assessment.

Since 1999, **Prof. Olivier J. Jolliet** is assistant professor in sustainable development at the Swiss Federal Institute of Technology - Lausanne (EPFL). His research focuses on the development of environmental assessment methods to determine action priorities, especially for bio-materials and for

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Born in 1972 in Switzerland, **Manuele Margni** graduated in Rural Engineering at the Swiss Federal Institute of Technology Zurich (ETHZ) in 1996. Then he spent 3 months at the Universitade Estadual Paulista, Sao Paolo (Brazil) to study the anaerobic fermentation of rural organic waste and biogaz production. In 1996/97 he took part in a Master program in environmental engineering and management at the EPFL and graduated with a research project in Life Cycle Assessment on the evaluation of widely used pesticides on arable crops in Switzerland. In 1998 he worked with Shell (Switzerland) in the department of “Quality, Health, Safety and Environment” on the organization of emergency plans for Shell fuelling services for the Zurich and Geneva airports and for their subsidiary companies. In the same year he co-founded a consultancy for Quality, Environmental and Safety Systems (ISO standards, EMAS, national and international safety standards, etc.) and environmental engineering’s projects. He joined the sustainable development group of the EPFL in 1999, whilst retaining a key role in his consultancy.

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