A flexible matrix algebra framework for the multimedia multipathway modeling of emission to impacts

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Abstract

When assessing human health or ecosystem impacts of chemicals several calculation steps need to be addressed. Matrix algebra solving techniques are a useful approach to structure and solve the system of mass balance equations assessing chemical fate in environmental multimedia models. We suggest expanding this matrix approach towards a framework which includes the exposure, effect, and damage assessment for human health and ecosystems, also applicable to spatial modeling. Special emphasis is laid upon interpretation of the physical meaning of different elements within the matrices. The proposed framework provides several advantages such as simplified updating or extending of models to new impact pathways, possibility of covering various models within the same framework and transparency. Interpretation of intermediate and final results is facilitated, e.g., allowing for direct identification of dominating exposure pathways. Model comparability and evaluation is well supported, as the four matrices contain all intermediate results in a clear and interpretable way, independent from parameters, such as amount and place of emission. Multidisciplinary work is strongly facilitated enabling the linkage of different models from various disciplines together, since each of its modules defines a clear interface of intermediate results. This framework was reviewed by an independent expert panel within a UNEP/SETAC workshop, and adopted as starting-point for new advances in modeling environmental toxic releases within the UNEP/SETAC Life Cycle Initiative.

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1. Introduction

The prediction of the impact of a chemical released into the environment is vital for decision making, e.g., when choosing the substance with the least environmental impact for a chemical product formulation (comparative assessment) or estimating the potential risk of a new substance (environmental risk assessment). Thus, the link from an emission to its impact needs to be modeled in order to predict and quantify the latter. In this paper we define and interpret a mathematical framework for multimedia chemical fate, exposure and human health/ecotoxicological effect models fully based on matrix algebra for both generic and geographically differentiated applications. We demonstrate its potential to update, extend, compare, and evaluate models, as well as to understand and interpret intermediate and final model results.

Several models and methods have been developed to predict the impact of a chemical released into the environment in the context of both life cycle impact assessment (LCIA) and environmental risk assessment (ERA). These methods relate environmental emissions to impacts (or risk factors) combining multimedia fate and multipathway exposure estimates with effect assessment data. Using the CalTOX model, McKone et al. (McKone et al., 2001; McKone and Bodnar, 2001; McKone, 2001) and Hertwich et al. (2001) calculated Human Toxicity Potentials (HTP) and Ecological Toxicity Potentials (ETP), providing generic factors to assess emissions of the U.S.
Toxic Release Inventory (TRI). Starting from the Simple Box model (Brandes et al., 1996), Huijbregts developed the USES–LCA model for Europe providing HTP, as well as marine, fresh water and terrestrial ETPs for use in LCA applications (Huijbregts, 1999). In the context of regulatory risk assessment the EUSES model (EC, 1996), based on Simple Box, assesses chemical risk for high production volume chemicals in the European Union. The BETR model (MacLeod et al., 2001) is a spatially resolved fate model for the U.S. More recently, Pennington et al. developed a spatially resolved Western European model, IMPACT 2002, relating generic or spatially resolved environmental emissions to human intake and subsequent impacts on human health and ecosystem quality (Pennington et al., 2005).

Updating, extending, comparing, interpreting and evaluating these models are often hampered by significant differences in their structure, setup and intermediate/final results. It is therefore crucial to have well interpretable intermediary coefficients whose order of magnitude could be directly checked.

Bridging this gap towards a consistently applied and interpreted matrix algebra framework is the starting point for this paper. We suggest expanding the matrix algebra framework, which is currently applied only to the fate calculations, to the exposure and effect assessment, hence, over the whole modeling steps from an emission to the damage on human health and ecosystems. This study specifically aims to address the following challenges:

1) Introduce an innovative framework based on a unique skeleton, enabling model flexibility in terms of algorithm updates and system extensions, such as new compartments, media, exposure pathways or effect types.
2) Set and interpret clear and easily intercomparable intermediate parameters and results throughout the whole source to damage assessment steps, independent from the emission scenario.
3) Propose a unified and consistent framework for both human health and ecosystem impacts.
4) Show how import/export of food can be considered in a spatial differentiated assessment.
5) Illustrate the framework with an example.

2. Selected approach

All above-mentioned models follow similar schemes. They first determine how the chemical mass distributes in the environment (fate analysis) based on mass conservation principles, solving the system of differential mass balance equations, with each equation representing the mass balance in the respective environmental medium and considering first order removal rates. Apart from the pure fate models, BETR and Simple Box, the other models then estimate the chemical intake by humans via the food and inhalation exposure pathways (exposure analysis) and finally assess the potency of the exposure by comparison with a Reference Dose (RfD) or a dose–response relationship (effect assessment). For human health, the latter accounts for the potential risks linked to the toxic intakes and the severity due to a certain illness. A similar procedure is adopted to assess ecosystem damage linking fate analysis with an effect assessment accounting for an increase in stress on species for a change in contaminant concentrations in the considered medium.

Matrix algebra solving techniques have already been used to solve the system of mass balance equations to assess chemical fate in multimedia models like Simple Box (Brandes et al., 1996), USES–LCA (Huijbregts, 1999) and IMPACT 2002 (Pennington et al., 2005). However, this solving technique was restricted to the fate assessment only and no interpretation of physical processes within the fate matrix has been done. Pennington et al. (2005) and Margni et al. (2004b) started interpreting the transfer rate coefficient matrix and the fate matrix in terms of intermediate transfer fractions – a measure of the multimedia partitioning tendencies of a chemical – and feedback correction factors. The latter being a multiplier that accounts for the fraction of an emission that returns to the medium of release after transfer to other media providing a measure of the level of coupling between media. An initial framework proposal was made within the OMNITOX project (Guinée et al., 2004; Molander et al., 2004) and reviewed in 2003 by a group of experts within the UNEP–SETAC Life Cycle Initiative (Jolliet et al., 2006) that recommended its use as a basis for further Life Cycle Toxicity developments. The present paper extends this initial attempt introducing a damage assessment, fully interpreting each matrix, detailing each calculation step, and operationalizing its application through a practical example.

We first present the overall matrix framework for human health impacts through a set of clear, intermediary parameters and results. As the framework for ecotoxicity effects is very similar, it has been summarized in the main text and its detailed description has been included in the Supporting information. We then separately determine how to set-up and calculate the fate, exposure and effects steps through matrix algebra for both non-spatial models and models comprising any kind of spatial differentiation. In parallel, we present the results of an illustrative example with heptachlor (CAS 76-44-8), applying the matrix framework to the IMPACT 2002 model (Pennington et al., 2005). A special emphasis will be laid upon interpretation of the physical meaning and the information provided by the different elements within the matrices. The Supporting information includes a complementary overview for a two box spatial system and a list of all variables, symbols and indices used throughout this paper.

3. General framework for human health impacts

Assessing the toxicological effects on the human health of a chemical emitted into the environment, whether released on purpose (e.g. pesticides), as a by-product from industrial processes, or by accident, implies a cause–effect-chain assessment.

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3 Jolliet and Rosenbaum were not part of the review committee, but only the reporters and hence co-authors of the workshop report; the reviewers are named in the report.
It links the emission source (emission flow vector $\vec{S}$ in kg/day) to the mass in the environmental compartments (mass vector $\vec{M}$ in kg), to the substance intake by the overall population (intake flow vector $\vec{I}$ in kg/day), and to the resulting number of cases of various morbidity risks (risk vector $\vec{N}$: number of cases). For comparative studies, the assessment can be extended to a damage step by determining the life years adversely affected by mortality and morbidity risks (damage vector $\vec{D}$: years/day).

Similarly, for regulatory risk applications one could express the results in terms of risk ratio, i.e. the predicted risk divided by a reference dose.

The links of this cause–effect chain can be modeled using matrices according to the successive steps of fate, exposure, effects, and damage:

$$D = DF \cdot EF \cdot U \cdot XF \cdot FF \cdot S' = DF \cdot EF \cdot U \cdot XF \cdot M'$$

where:

- **Fate ($FF$)** links the quantity released into the environment to the chemical masses (or concentrations) in a given compartment. It accounts for multimedia and spatial transport between the environmental media (e.g. air, water, soil, etc.). It is quantified by the fate matrix $FF$; a column denotes the source compartment $m$ and a row denotes the destination compartment $i$, i.e. where the chemical is transferred into. The size of $FF$ is determined by the number of environmental compartments $n_i$ considered ($n_i$ and the number of source compartments $n_m$ are equal, since every destination compartment can also be a source compartment, hence $n_m=n_i$), and thus be $(n_i \times n_i)$. The fate factor $FF_{i,m}$ [day] can be interpreted as the increase of chemical mass in compartment $i$ [kg] due to an emission in compartment $m$ [kg/day].

- **Exposure ($XF$)** relates the amount found in a given environmental compartment to the chemical intake by humans. It can be distinguished between direct intake (e.g. by breathing air and drinking water, etc.), indirect intake through bioconcentration processes in animal tissues (e.g. meat, milk and fish) and intake by dermal contact. It is quantified by the exposure matrix $XF$ that contains exposure factors (or exposure rates); a column denotes a destination compartment $i$ and a row denotes the exposure pathway $xp$ (e.g. meat, milk and fish). The size of $XF$ will be determined by the number of exposure routes $n_{xp}$ and the number of environmental compartments $n_i$ considered, and thus be $(n_{xp} \times n_i)$. The exposure factor $XF_{xp,i}$ [1/day] is the equivalent rate of ingestion of the medium by humans.

- **Intake fraction ($iF = XF \cdot FF$)**: The fate and exposure matrices can also be aggregated into an intermediary matrix:
the intake fraction matrix \( \overline{IF} \): a column denotes an emission compartment \( m \) and a row denotes the exposure pathway \( xp \) (e.g. meat, milk and fish). The size of \( \overline{IF} \) is determined by the number of exposure pathways \( n_{xp} \) and the number of compartments \( n_i \) considered, and will thus be \( (n_{xp} \times n_i) \). The intake fraction \( iF_{xp,i} \) \([\text{kg intake/kg emitted}]\) can be interpreted as the fraction of an emission into a source compartment \( m \) that is taken in by the overall population through a given intake pathway \( xp \). \( iF \) is defined and interpreted by Bennett and coworkers (Bennett et al., 2002a,b). For further calculation, \( \overline{IF} \) is aggregated by inhalation, ingestion and dermal exposure routes via multiplication with a “pseudo-unitarian” matrix \( U \) and becomes \( \overline{IF}_{xr} \).

d) **Effects (EF)** relates the quantity taken in via a given exposure route by a population to the adverse effects (or potential risk) of the chemical on an organism. It is quantified by the effect matrix \( \overline{EF} \) containing effect factors; a column denotes an exposure route \( xr \) (e.g. inhalation, ingestion or dermal) and a row denotes an effect type \( ef \) (e.g. cancer, non-cancer). The size of \( \overline{EF} \) will be determined by the number of effect types \( n_{ef} \) and the number of exposure routes \( n_{xr} \) considered, and thus be \( (n_{ef} \times n_{xr}) \). The effect factor \( EF_{ef,xr} \) \([\text{number of cases/kg intake}]\) can be interpreted as the increase in the number of cases of a given morbidity (e.g. cancer or non-cancer diseases) risk \([\text{dimensionless}]\) in the exposed population per unit mass ingested or inhaled \([\text{kg intake}]\) — itself due to an emission source in compartment \( m \).

e) **Damage (DF)** distinguishes between differences in the severity of disabilities caused by a disease in terms of affected life years, e.g., discriminating between a lethal cancer and a skin irritation. It is quantified by the damage matrix \( \overline{DF} \) containing damage factors; each line represents a damage severity type \( d \) (e.g. years of life lost, years of life disabled) due to the different effects \( ef \) (e.g. cancer, non-cancer) considered in each column. The size of \( \overline{DF} \) will be determined by the number of effect types \( n_{ef} \) and the number of damage types \( n_d \) considered, and thus be \( (n_{ef} \times n_d) \). The damage factor \( DF_{ef,xd} \) \([\text{years/case}]\) represents adversely affected life years per case (interpretation of the unit depends on the choice of the severity factor, see below for a further discussion). Generally, it can be interpreted as the severity associated to an adverse effect \( ef \) (e.g. cancer or non-cancer diseases). It is also possible to only consider the likelihood of an adverse effect without any severity weighting by simply omitting the \( \overline{DF} \) matrix for the calculations, but this implies that all effect types should be kept separate unless an equal severity weighting is implicitly assumed.

f) **The human damage factor matrix (HDF)** \( (HDF_{d,m} = \overline{DF} \cdot \overline{EF} \cdot \overline{U} \cdot \overline{XF} \cdot \overline{FF}) \) combines all these steps and expresses the damage per unit emitted into the environment; a row denotes the considered damage \( d \) (e.g. years of life lost, years of life disabled) — or effect \( ef \) if omitting \( \overline{DF} \) — and a column denotes the initial emission compartment \( m \). The size of \( \overline{HDF} \) is determined by the number of damage types \( n_d \) and the number of environmental compartments \( n_i \) considered, and thus be \( (n_d \times n_i) \). The interpretation of the human damage factor \( HDF_{d,m} \) \([\text{year/kg emitted}]\) depends on the choice of the severity factor and will for example represent an increase in adversely affected life years \([\text{year}]\) as a consequence of an emission in compartment \( m \) \([\text{kg emitted}]\). If the severity

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**Fig. 2. Overview of the matrices with meaning of rows and columns.**
The way each matrix is calculated and interpreted is more precisely described in the subsequent paragraphs. Fig. 2 provides a descriptive summary of the features of most matrices defined throughout this paper.

Several authors demonstrated the mathematical relationship between the steady-state solution for a continuous emission and the time-integrated solution for a mass of chemical released into the environment on a generic level (Mackay and Seth, 1999; Heijungs, 1995). Therefore, similarly as for a mass flow release, the same equation can be written to relate mass emitted (i.e. as pulse emission), time integrated mass and intake, and time and volume integrated number of cases, and damage (at steady state, with linear modeling) as in Fig. 1 and Eq. (2):

\[ D' = DF \cdot EF \cdot U \cdot XF \cdot FF \cdot X' = DF \cdot EF \cdot U \cdot XF \cdot \int \dot{M} dt \]

\[ = \int DF \cdot EF \cdot \frac{\dot{M}}{V_x} \cdot \int \frac{\dot{M}}{X' dt} = \int DF \cdot EF \cdot \frac{\dot{M}}{X' dt} \]

\[ = \int DF \cdot \frac{\dot{M}}{X' dt} = \int HDF \cdot \frac{\dot{M}}{X' dt} \]

(2)

4. Illustrative example and detailed matrix interpretation for human health

The inputs to the matrices are detailed below and their interpretation discussed in detail and illustrated using a simplified version of the IMPACT 2002 model (available at http://www.sph.umich.edu/riskcenter/jolliet/impact2002.htm) with five compartments – air (a), surface water (w), soil (s), sediment (d), and vegetation (v) – at steady-state conditions. This framework serves as a library which can be filled with different models for each sub-calculation. Hence, it is not limited to one model.

4.1. The fate matrix

Per definition the fate matrix \( \mathbf{FF} \) equals the negative inverse of the transfer rate coefficient matrix \( \dot{k} \): \( \mathbf{FF} = -\dot{k}^{-1} \). The \( \dot{k} \) matrices and some further background are given in the Supporting information. The fate matrix is given here reflecting the heptachlor example:

\[
\mathbf{FF} = \begin{pmatrix}
FF_{a,a} & FF_{a,w} & FF_{a,d} & FF_{a,v} \\
FF_{w,a} & FF_{w,w} & FF_{w,d} & FF_{w,v} \\
FF_{d,a} & FF_{d,w} & FF_{d,d} & FF_{d,v} \\
FF_{v,a} & FF_{v,w} & FF_{v,d} & FF_{v,v}
\end{pmatrix}
\]

\[
= \dot{k}^{-1} = \begin{pmatrix}
1.53 & 3.27 \cdot 10^{-1} & 1.25 & 3.98 \cdot 10^{-3} & 1.51 \\
1.26 \cdot 10^{-2} & 2.51 \cdot 10^{-1} & 1.43 \cdot 10^{-2} & 3.06 \cdot 10^{-1} & 1.24 \cdot 10^{-2} \\
1.35 \cdot 10^{-1} & 2.90 \cdot 10^{-2} & 1.86 \cdot 10^{-1} & 3.52 \cdot 10^{-4} & 1.34 \cdot 10^{-1} \\
1.38 \cdot 10^{-3} & 2.76 & 1.57 \cdot 10^{-3} & 3.26 \cdot 10^{-2} & 1.36 \cdot 10^{-3} \\
6.16 \cdot 10^{-1} & 1.32 \cdot 10^{-1} & 5.04 \cdot 10^{-1} & 1.60 \cdot 10^{-3} & 1.38
\end{pmatrix}
\]

The elements of the fate matrix \( \mathbf{FF} \) are the fate factors, and have a unit of [day].

The following analysis and interpretation of the fate matrix applies:

1. Residence time: The diagonal elements \( \mathbf{FF}_{m,m} \) describe the effective residence time in the respective compartments \( m \), i.e. the inverse of the effective rate constant introduced by Bennett et al. (1998). In our example, heptachlor has its longest residence time in sediment (326 days) and in water (25 days), and its shortest in vegetation (1.4 days) and air (1.5 days).

2. Mass in the environment: A column of \( \mathbf{FF} \) describes the mass in the environment resulting from a unit emission flow in the corresponding compartment. Hence, dividing each element by the sum of the respective column indicates the repartition of the resulting mass between all destination compartments due to the emission compartment represented by this column. Thus revealing, e.g., into which compartment(s) a chemical mainly partitions. This quantification shows that heptachlor is found more than 89% in the emission compartment when released into water, soil, or sediment, with, e.g., 1.5 kg heptachlor in air at steady state for an emission of 1 kg/day in Europe. An emission into air stays at 67% in air, and 27% will be found on vegetation. When eventually released onto vegetation, 50% of the chemical will be found in air, while 45% will rest on vegetation.

3. Inter-compartment transfer fractions: Each non-diagonal element can also be expressed as a fraction transferred from the source compartment \( i \) multiplied by the effective residence time in the destination compartment. This means that dividing each element in a row by the residence time (the diagonal element) provides the transferred fractions from media \( i \) to \( j \): \( f_{ij} = FF_{ij}/FF_{i,i} \) for an equal emission into all compartments. The transferred fractions already include the sum of all possible transfer pathways through a third media. For example, the fraction of an air emission of heptachlor transferred to water (via air–water and air–soil–water) amounts to \( FF_{w,a}/FF_{w,w} = 1.26 \cdot 10^{-2}/2.51 \cdot 10^{-1} = 0.05\% \).

4. Feedback factors: The feedback factor, which is the product of the corresponding diagonal elements of \( \dot{k} \) and fate matrix, yields the fraction of heptachlor being transferred back into the compartment of origin (Margni et al., 2004b). For our example, this feedback fraction amounts to 80% for air and vegetation, whereas it is negligible for soil (1%), water (0.02%), or sediment (0.01%). Combined with the interpretation of the \( \dot{k} \) matrix (in the Supporting information) this leads to the conclusion that a rapid transport from vegetation to air takes place, followed by a fast degradation in air. Whereas, sediment degradation is much slower and no other removal process is of remarkable influence. The moderate residence time in soil is mainly driven by the transport into air, while most of the substance in water is degraded at a moderate rate compared to the sediment residence time.
For spatially resolved calculations, the $\mathbf{K}$ matrices for each zone $z$, $\mathbf{k}(z)$, are combined in the diagonal of an overall $\mathbf{K}$ matrix for $z$ spatial cells, resulting in a corresponding fate matrix as illustrated below:

$$
\mathbf{K} = \begin{pmatrix}
\mathbf{k}(1) & \mathbf{k}(1 \rightarrow 2) & \ldots & \mathbf{k}(1 \rightarrow z) \\
\mathbf{k}(2 \rightarrow 1) & \mathbf{k}(2) & \ldots & \mathbf{k}(2 \rightarrow z) \\
\vdots & \vdots & \ddots & \vdots \\
\mathbf{k}(z \rightarrow 1) & \mathbf{k}(z \rightarrow 2) & \ldots & \mathbf{k}(z)
\end{pmatrix}
$$

$$
\mathbf{F} = \begin{pmatrix}
\mathbf{F}(1) & \mathbf{F}(1 \rightarrow 2) & \ldots & \mathbf{F}(1 \rightarrow z) \\
\mathbf{F}(2 \rightarrow 1) & \mathbf{F}(2) & \ldots & \mathbf{F}(2 \rightarrow z) \\
\vdots & \vdots & \ddots & \vdots \\
\mathbf{F}(z \rightarrow 1) & \mathbf{F}(z \rightarrow 2) & \ldots & \mathbf{F}(z)
\end{pmatrix}
$$

The elements populating the off-diagonal matrices of $\mathbf{K}$ describe interzonal advection rates (e.g. from sea water of zone 1 to sea water of zone 2). The diagonal elements (matrices) of $\mathbf{F}$ describe the effective residence times within each zone, while all off-diagonal elements account for residence times in the destination compartment due to advective transport from one zone to another. See Supporting information for an illustrative spatial calculation.

4.2. The exposure matrix

The exposure pathway matrix consists of two parts reflecting direct and indirect exposure rates aggregated from the $\mathbf{XF}$ matrix, which is thus given by:

$$
\mathbf{XF} = \begin{pmatrix}
\mathbf{XF}_{\text{direct inh,a}} & 0 & 0 & 0 & 0 \\
0 & \mathbf{XF}_{\text{direct w,w}} & 0 & 0 & 0 \\
0 & 0 & \mathbf{XF}_{\text{direct v,v}} & 0 & 0 \\
0 & 0 & 0 & \mathbf{XF}_{\text{direct d,d}} & 0 \\
1.69 \times 10^{-6} & 0 & 0 & 0 & 0 \\
0 & 4.47 \times 10^{-7} & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 2.22 \times 10^{-4} \\
0 & 1.94 \times 10^{-5} & 0 & 0 & 0 \\
3.51 \times 10^{-9} & 2.31 \times 10^{-9} & 2.66 \times 10^{-11} & 0 & 5.88 \times 10^{-7} \\
1.33 \times 10^{-8} & 2.08 \times 10^{-8} & 1.87 \times 10^{-10} & 0 & 3.78 \times 10^{-6} \\
1.83 \times 10^{-10} & 2.65 \times 10^{-11} & 7.12 \times 10^{-13} & 0 & 1.26 \times 10^{-7}
\end{pmatrix}
$$

where $\mathbf{XF}[1/\text{day}]$ is the exposure pathway rate coefficient. The exposure matrix $\mathbf{XF}$ for heptachlor includes five direct pathways related to the respective compartments and four indirect pathways via fish, meat, milk, and eggs.

The following analysis and interpretation of the exposure matrix applies:

1. Direct exposure: The top rows constitute a direct exposure square matrix in which off-diagonal elements are equal to zero and the diagonal elements reflect the direct exposure pathway rate coefficient determined as:

$$
\mathbf{XF}_{\text{direct}, xp,i} = \frac{\mathbf{IR}_{xp,i}}{\rho_i \cdot V_i}
$$

where $\mathbf{IR}_{xp,i}$ [kg/day] symbolizes the direct intake rate of a medium $i$ by the overall population (e.g. direct consumption of vegetation or water, or the inhalation of air) polluted at a certain level via an exposure pathway $xp$, $\rho_i$ is the bulk density of medium $i$ [kg/m$^3$], $V_i$ [m$^3$] is the volume of the respective medium $i$ linked to the respective exposure pathway. For direct intake of freshwater for example, the direct exposure rate corresponds to the fraction of the total mass of drinking water, ingested daily by humans, that is 0.45 ppm of the total freshwater ingested daily for heptachlor. The inverse of this coefficient therefore represents the equivalent time required by the population to inhale or ingest the whole mass in the medium and amounts to 6125 years for drinking water in Europe. The direct intake of soil and sediment is assumed to be zero. All pathways starting from the second row and column are representing ingestion pathways and should be kept separate from inhalation as the dose–response differs according to the exposure route. The direct intake of soil and sediment was assumed in this approach. For inaccessible media like underground soil the diagonal element will be zero too.

2. Indirect exposure pathways: The bottom rows (i.e. only rows added) represent the indirect exposure pathways, each row corresponding to a different exposure substrate (e.g. meat, dairy produce, vegetables, and fish) polluted by a respective compartment (column). Each coefficient represents the increase in exposure by substrate $xp$ due to an increase in concentration in medium $i$ and is given by:

$$
\mathbf{XF}_{\text{indirect}, xp,i} = \frac{\mathbf{BAF}_{xp,i} \cdot \mathbf{IR}_{xp}}{\rho_i \cdot V_i}
$$

where $\mathbf{IR}_{xp}$ [kg/day] is the ingestion rate of substrate $xp$ at population level, $\mathbf{BAF}_{xp,i} = C_{xp,i}/C_{[kgp/kg]}$ is the bioaccumulation factor (steady-state concentration ratio between substrate $xp$, such as meat or milk, and a specific compartment $i$). $\mathbf{BAF}$ can be determined from biotransfer factors (Rosenbaum, 2006; Dowdy et al., 1996; Travis and Arms, 1988) or carry over rates and animal-specific data on breathing rates, feed, water, and soil ingestion. The indirect intake rate can be interpreted as the equivalent intake rate of the polluted medium $i$ due to the exposure substrate $xp$. For example, the exposure by eating a contaminated fish and taking in his body burden represents for heptachlor an equivalent intake of water of 19 ppm of the total freshwater ingested daily and is more than 40 times higher than the direct freshwater intake. This means that the coefficients within the same column (for a given compartment) can be directly compared, enabling identification of the most significant exposure pathways. The sum of all elements per column represents the total transfer rate to the population per increment of mass in the respective compartment.

The $\mathbf{XF}$ matrix also enables the comparison of direct and indirect exposure contribution. For heptachlor, we find that direct exposure is dominant for emissions to air (100% inhalation) and
vegetation (98%). Fish is the dominating pathway for water emissions (98%), while milk represents the highest ingestion exposure pathway for soil (87%) and air emissions (78%). Rows could be added to the matrix to represent dermal exposure, where IR is replaced by an uptake rate proportional to the duration of exposure, also the EF needs to be specific for this exposure route.

For spatially resolved calculations, exposure matrices can be determined for each zone \( z \) \( \Xi F(\bar{z}) \), applying the respective zone dependent parameters. Finally, the exposure matrices for each zone \( z \), \( \Xi F(\bar{z}) \), can be combined into an overall exposure route matrix \( \Xi F \) for spatial cells as illustrated below:

\[
\Xi F = \begin{pmatrix}
\Xi F(1) & \Xi F(2) & \cdots & \Xi F(z-1) \\
\Xi F(1) & \Xi F(2) & \cdots & \Xi F(z-2) \\
\vdots & \vdots & \ddots & \vdots \\
\Xi F(1) & \Xi F(2) & \cdots & \Xi F(z)
\end{pmatrix}
\]

The off-diagonal elements (matrices) represent the food transfer (import/export) between zones. Thus, they account for a food substrate produced and contaminated in one zone and then transported into another zone to be consumed there. This is a similar concept to the interzonal advective transport rates in the spatial fate matrix. Interpretation including interzonal transfer of contaminated food substrates and media can be discussed similarly as above. Summing up all elements in a row, one could determine the total exposure rate of the population living in the zone located on the diagonal via the corresponding exposure route. The sum of all elements per column represents the total exposure rate from the selected compartment and zone on the diagonal to the populations living in all the zones. It includes the export via food or exported media, such as tap water.

### 4.3. Intake fraction — combining fate and exposure matrix

Intake fraction (iF) is defined as the fraction of mass of a chemical released into the environment that is ultimately taken in by the human population as a result of food contamination, inhalation, and dermal exposure (Bennett et al., 2002b). A high value, such as \( iF = 0.001 \) for dioxin (Margni et al., 2004a; Bennett et al., 2002a), reflects that humans will take in 1 part of 1000 of the mass of a chemical released. iF is calculated by multiplying the fate with the exposure factor:

\[
iF = \Xi F \cdot \Xi F = \begin{pmatrix}
\text{iF}_{\text{a,inh}} & \text{iF}_{\text{a,w}} & \text{iF}_{\text{a,v}} & \text{iF}_{\text{d,inh}} & \text{iF}_{\text{d,w}} & \text{iF}_{\text{d,v}} & \text{iF}_{\text{v,inh}} & \text{iF}_{\text{v,w}} & \text{iF}_{\text{v,v}} \\
\text{iF}_{\text{a,inh}} & \text{iF}_{\text{a,w}} & \text{iF}_{\text{a,v}} & \text{iF}_{\text{d,inh}} & \text{iF}_{\text{d,w}} & \text{iF}_{\text{d,v}} & \text{iF}_{\text{v,inh}} & \text{iF}_{\text{v,w}} & \text{iF}_{\text{v,v}} \\
\text{iF}_{\text{a,inh}} & \text{iF}_{\text{a,w}} & \text{iF}_{\text{a,v}} & \text{iF}_{\text{d,inh}} & \text{iF}_{\text{d,w}} & \text{iF}_{\text{d,v}} & \text{iF}_{\text{v,inh}} & \text{iF}_{\text{v,w}} & \text{iF}_{\text{v,v}} \\
\text{iF}_{\text{a,inh}} & \text{iF}_{\text{a,w}} & \text{iF}_{\text{a,v}} & \text{iF}_{\text{d,inh}} & \text{iF}_{\text{d,w}} & \text{iF}_{\text{d,v}} & \text{iF}_{\text{v,inh}} & \text{iF}_{\text{v,w}} & \text{iF}_{\text{v,v}} \\
\text{iF}_{\text{a,inh}} & \text{iF}_{\text{a,w}} & \text{iF}_{\text{a,v}} & \text{iF}_{\text{d,inh}} & \text{iF}_{\text{d,w}} & \text{iF}_{\text{d,v}} & \text{iF}_{\text{v,inh}} & \text{iF}_{\text{v,w}} & \text{iF}_{\text{v,v}} \\
\text{iF}_{\text{a,inh}} & \text{iF}_{\text{a,w}} & \text{iF}_{\text{a,v}} & \text{iF}_{\text{d,inh}} & \text{iF}_{\text{d,w}} & \text{iF}_{\text{d,v}} & \text{iF}_{\text{v,inh}} & \text{iF}_{\text{v,w}} & \text{iF}_{\text{v,v}} \\
\text{iF}_{\text{a,inh}} & \text{iF}_{\text{a,w}} & \text{iF}_{\text{a,v}} & \text{iF}_{\text{d,inh}} & \text{iF}_{\text{d,w}} & \text{iF}_{\text{d,v}} & \text{iF}_{\text{v,inh}} & \text{iF}_{\text{v,w}} & \text{iF}_{\text{v,v}} \\
\text{iF}_{\text{a,inh}} & \text{iF}_{\text{a,w}} & \text{iF}_{\text{a,v}} & \text{iF}_{\text{d,inh}} & \text{iF}_{\text{d,w}} & \text{iF}_{\text{d,v}} & \text{iF}_{\text{v,inh}} & \text{iF}_{\text{v,w}} & \text{iF}_{\text{v,v}} \\
\text{iF}_{\text{a,inh}} & \text{iF}_{\text{a,w}} & \text{iF}_{\text{a,v}} & \text{iF}_{\text{d,inh}} & \text{iF}_{\text{d,w}} & \text{iF}_{\text{d,v}} & \text{iF}_{\text{v,inh}} & \text{iF}_{\text{v,w}} & \text{iF}_{\text{v,v}} \\
\text{iF}_{\text{a,inh}} & \text{iF}_{\text{a,w}} & \text{iF}_{\text{a,v}} & \text{iF}_{\text{d,inh}} & \text{iF}_{\text{d,w}} & \text{iF}_{\text{d,v}} & \text{iF}_{\text{v,inh}} & \text{iF}_{\text{v,w}} & \text{iF}_{\text{v,v}}
\end{pmatrix}
\]

The difference with the exposure matrix is that the column refers here to the emission compartment (not the destination compartment) and already takes into account the multi-media transfers between compartments. The following analysis and interpretation of the intake fraction matrix applies:

1. **Intake fraction for individual pathways**: Each element represents an actual intake fraction, e.g., the fraction of the kg emitted which is taken in by the human population, for the considered release compartment (column) and the exposure pathway (row). For example, the intake fraction through vegetation intake for an heptachlor emission to air amounts to 0.14 per thousands (1 kg emitted to air, 0.14 g ingested by the population).

2. **Pathways contributions**: The ratio of each element in a column with the sum of all elements of the same exposure route (e.g. inhalation, ingestion, dermal) within this column yields the contribution of this pathway to the corresponding route (e.g. the contribution of exposure via fish consumption relative to overall ingestion exposure). For heptachlor, we find that over 90% of oral exposure is taken in via vegetation when emitted to air, soil, and vegetation, and over 98% via fish when emitted to water or sediment.

3. **Aggregation by exposure route**: For further calculation the intake fraction matrix \( iF \) can be aggregated by routes of exposure (e.g. inhalation, ingestion, or dermal) via multiplication with a “pseudo-unitarian” matrix \( U \) as follows:

\[
iF_{\text{aggregated}} = U \cdot iF = \begin{pmatrix}
1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 \\
\end{pmatrix}
\]

The role of \( U \) is to attribute each pathway to an exposure route and to sum the individual exposure pathways into main routes of exposure (inhalation, ingestion, dermal). The size of \( U(n_x \times n_y) \) is given by the total number of exposure routes in \( iF_{\text{aggregated}} \) (determining the number of rows) and the total number of exposure pathways in \( iF \) (determining the number of columns). In the heptachlor example, only inhalation and ingestion were considered. A third line could be added in the \( U \)-matrix to account for dermal exposure in a more general case.

4. **Comparison of exposure routes**: While the elements of \( iF_{\text{aggregated}} \) need to be multiplied with the source vector \( S \) to yield actual intake, an interpretation of \( iF_{\text{aggregated}} \) is already possible. For heptachlor, by ingestion is one to two orders of magnitude higher than by inhalation, even for an emission to air. For the ingestion pathways, iF are fairly close per unit emission flow to air, water, soil, and vegetation, while inhalation iF strongly depends on the emission compartment. The iF for sediment are only valid for releases to sediment which is a rather unusual emission scenario. The vegetation iF represent an emission to vegetation which could be realistic for specific chemicals such as...
pesticides. According to Eq. (1) the multiplication of IF by the source vector yields the overall amount taken in by the whole population, enabling to determine which emission in which compartment contributes dominantly to each exposure route.

For spatially resolved calculations the intake fraction matrices for each zone, \( iF_{xz}(z) \), are represented in the overall intake fraction matrix \( iF_{x} \) for \( z \) spatial cells as illustrated below:

\[
iF_{x} = \begin{pmatrix}
    iF_{x1}(1) & iF_{x1}(2) & \cdots & iF_{x1}(z+2) \\
    iF_{x2}(1) & iF_{x2}(2) & \cdots & iF_{x2}(z+2) \\
    \vdots & \vdots & \ddots & \vdots \\
    iF_{x(z+2)}(1) & iF_{x(z+2)}(2) & \cdots & iF_{x(z+2)}(z+2)
\end{pmatrix}
\]

The off-diagonal elements (matrices) represent the fraction of a chemical that is taken in by a population living in the zone on the respective row due to a contamination of food produced and imported from the zones on the selected column; this latter being linked to an emission into compartment \( i \) of the zone of origin. Hence, they describe the transfer of a chemical between zones due to import/export of food (as represented in \( XF \)) and natural advective transport of pollutants (as represented in \( FF \)). For example, it can correspond to the fraction of an emission occurred in a zone in Spain contaminating a wheat culture in another zone in France that is exported and ultimately eaten in a third zone in Switzerland. Thus, the elements (matrices) on the diagonal represent the internal (zone) IFs and the off-diagonal elements represent the fraction of an emission “exported” to another zone (within the same column) or “imported” into the zone (from within the same row); all together representing the total intake fraction due to an emission in the zone of origin.

Applying the interpretation analysis, as described above for one zone, reveals the respective interpretations now including interzonal trade of contaminated food substrates/media and advective transport. After multiplication with the source vector, summing up all elements in a row, one could determine the total IF in the respective zone due to emissions into any compartment in any zone in the system. The sum of all elements per column represents the total IF due to an emission into this zone.

4.4. The human health effects matrix

The human health effects matrix \( EF \) is constituted by the human health effect factors [cases/kg intake]:

\[
EF = \begin{pmatrix}
    EF_{c. inh} & EF_{c. ing} \\
    EF_{nc. inh} & EF_{nc. ing}
\end{pmatrix} = \begin{pmatrix}
    1.29 & 1.17 \\
    0 & 1.40
\end{pmatrix}
\]

The effect matrix shows a 1.3 increase in cases of cancer per kg heptachlor inhaled, respectively 1.2 cases per kg ingested. The increase in non-cancer cases amounts to 1.4 cases per kg ingested. The use of only two general effect classes “cancer” and “non-cancer” represents current practice (Huijbregts et al., 2005; Crettaz et al., 2002; Pennington et al., 2002), as it is very difficult to predict human endpoints from animal dose–response curves (Owens, 2002). If information becomes available, the framework can be easily extended to distinguish between a wider number of effect types such as disease classes or types of cancer. For example, according to Owens (2002), Burke et al. (1996), or Fava et al. (1993), this can be done by simply adding new rows containing the respective effect factors. Similarly, effects via additional exposure routes could be considered by adding new columns for this route in parallel to the exposure and intake fraction matrices.

A more detailed description of the applied human health effects model can be found in the corresponding section of the Supporting information.

In common practice, simplifying assumptions consider the effects not being spatially dependent, i.e. they do not differ between zones. Thus, in a spatially resolved system the elements are currently just repeated for each zone:

\[
EF = \begin{pmatrix}
    EF & 0 & \cdots & 0 \\
    0 & EF & \cdots & 0 \\
    \vdots & \vdots & \ddots & \vdots \\
    0 & 0 & \cdots & EF
\end{pmatrix}
\]

If more detailed data for each zone are available (e.g. due to varying sensitivity of populations to a specific effect, a varying composition (age groups) of a population, or varying toxicity of a chemical due to differing conditions from one zone to another) a zone dependent effect factor can easily be applied by accordingly populating the diagonal elements of the matrix above. Off-diagonal elements (matrices) are presently set to zero, but could represent a secondary effect linked to an epidemic propagation or emigration of exposed persons from one zone to another.

4.5. The damage matrix

The damage matrix links the number of incidences to the potential consequences of a chronic toxicological effect. It accounts for severity of the diseases/incidents considered in the human health effects matrix. A possible option proposed by Murray and Lopez (1996) is to consider different severity contributions defined as “Years of Life Lost per affected Person” (YLL\(_{p}\) [year/case]) and “Years of Life lived with a Disability per affected Person” (YLD\(_{d}\) [years/case]).

The damage factor matrix \( DF \) can be defined as:

\[
DF = \begin{pmatrix}
    YLL_{c} & 0 & \cdots & 0 \\
    0 & YLL_{nc} & \cdots & 0 \\
    \cdots & \cdots & \ddots & \cdots \\
    0 & 0 & \cdots & YLD_{d} \cdot W_{c}
\end{pmatrix} \cdot \begin{pmatrix}
    11 & 0 \\
    0 & 1.7 \\
    0.5 & 0 \\
    0 & 1
\end{pmatrix}
\]

where YLL are calculated on the basis of number and age of death for a given disease and have been on separate lines to keep results on cancer and non cancer distinct as proposed by the UNEP/SETAC Life Cycle Initiative (Jolliet et al., 2004). YLD should be kept in separate rows for each disease or effect considered, as severity could differ. Optionally, this information can be combined into a single vector using disability weights for
each effect \((W_{ef})\) to yield the “Disability Adjusted Life Years per affected Person” \((\text{DALY}_{p}\text{[year/person]})\). \(\text{DF}\) can then be aggregated into a disability adjusted life years damage vector \(\text{DF}_{\text{DALY}} = (11W_{ef1},W_{ef2},\ldots) \cdot \text{DF}\).

Assuming default damage severity factors for cancer and non-cancer effects as proposed by Crettaz et al. (2002) and Pennington et al. (2002) and recently improved by Huijbregts et al. (2005), the damage matrix for heptachlor means that 11 YLL are lost per cancer case plus 0.5 DALY’s due to Years of Life Disabled, yielding a total of 11.5 DALY per cancer case. This amounts to \(1 + 1.7 = 2.7\) DALY per non-cancer case.

In current practice, human health effects data have not been considered to vary over space. However, severity is spatially dependent, as the fraction of people receiving a treatment differs between zones and continents. If YLL and YLD are available for each zone they are applied accordingly in a spatially resolved system, otherwise the diagonal elements are just resolved system, otherwise the diagonal elements are just.

for each effect \((\text{P})\), as discussed above. Hence, these could now be determined for any possible emission scenario without having to solve the differential equation each time.

4.6. The human damage matrix

The human damage matrix for heptachlor is then calculated as:

\[
\text{HDF} = \begin{pmatrix}
\text{HDF}_{\text{YLL}_{1},a} & \text{HDF}_{\text{YLL}_{1},w} & \text{HDF}_{\text{YLL}_{2},a} & \text{HDF}_{\text{YLL}_{2},d} & \text{HDF}_{\text{YLL}_{c},v} \\
\text{HDF}_{\text{YLD}_{1},a} & \text{HDF}_{\text{YLD}_{1},w} & \text{HDF}_{\text{YLD}_{2},a} & \text{HDF}_{\text{YLD}_{2},d} & \text{HDF}_{\text{YLD}_{c},v} \\
\text{HDF}_{\text{YLD}_{1},c} & \text{HDF}_{\text{YLD}_{2},c} & \text{HDF}_{\text{YLD}_{3},c} & \text{HDF}_{\text{YLD}_{4},c} & \text{HDF}_{\text{YLD}_{5},c} \\
\end{pmatrix}
\]

\[
= \begin{pmatrix}
1.8\cdot10^{-3} & 6.8\cdot10^{-3} & 1.5\cdot10^{-3} & 8.3\cdot10^{-5} & 4.1\cdot10^{-3} \\
3.3\cdot10^{-4} & 1.3\cdot10^{-3} & 2.7\cdot10^{-5} & 1.5\cdot10^{-5} & 7.4\cdot10^{-4} \\
8.4\cdot10^{-5} & 3.1\cdot10^{-5} & 6.9\cdot10^{-5} & 3.8\cdot10^{-6} & 1.8\cdot10^{-4} \\
2.0\cdot10^{-4} & 7.4\cdot10^{-4} & 1.6\cdot10^{-4} & 9.0\cdot10^{-6} & 4.4\cdot10^{-4} \\
\end{pmatrix}
\]

and

\[
\text{HDF}_{\text{DALY}} = \begin{pmatrix}
\text{HDF}_{\text{DALY}_{1},a} & \text{HDF}_{\text{DALY}_{1},w} & \text{HDF}_{\text{DALY}_{2},a} & \text{HDF}_{\text{DALY}_{2},d} & \text{HDF}_{\text{DALY}_{c},v} \\
\end{pmatrix}
\]

\[
= \begin{pmatrix}
2.4\cdot10^{-7} & 9.1\cdot10^{-3} & 2.0\cdot10^{-7} & 1.1\cdot10^{-5} & 5.4\cdot10^{-7} \\
\end{pmatrix}
\]

It shows that for heptachlor the cancer impact is dominated by the Years of Life Lost rather than Years of Life Disabled and that both cancer and non cancer are of the same order of magnitude, i.e. between 0.01 and 0.001 DALY per kg emitted in air, water soil or vegetation.

The actual intermediate results environmental mass \(M\), intake \(I\), risk \(N\) and severity \(D\) are dependent on the source vector \(S\), which defines the release compartment(s) and amount (s), as discussed above. Hence, these could now be determined for any possible emission scenario without having to solve the differential equation each time.

5. Application to impacts on ecosystems

As the general framework for ecosystems is very similar to that for human health, it has been detailed in the Supporting information and only its interpretation and application to the impact of heptachlor on aquatic ecosystems is provided below.

The aquatic ecotox effect characterization factors can be expressed as the multiplication of three factors: the fraction transferred from emission compartment \(i\) to freshwater \((f_{i,w} = \text{FF}_{w,i}/\text{FF}_{w,w})\) multiplied by the residence time in water \((\text{FF}_{w,w})\) and the effect factor in water:

\[
\text{EDF}_{\text{aqu,i}} = f_{i,w} \cdot \text{FF}_{w,w} \cdot \text{EEF}_{\text{aqu,w}}
\]

Based on Payet and Jollivet (2004), \(\text{EEF}_{\text{aqu,w}} = 757\) [PAF m³/kg] for heptachlor and one gets:

\[
\text{EDF} = (\text{EDF}_{\text{aqu,a}} \cdot \text{EDF}_{\text{aqu,g}} \cdot \text{EDF}_{\text{aqu,d}} \cdot \text{EDF}_{\text{aqu,v}})
\]

\[
= (9.5 \cdot 1.9 \cdot 10^{-2} \cdot 1.1 \cdot 10^{-3} \cdot 2.3 \cdot 10^{-2} \cdot 9.4)
\]

Section 4.1 has shown that the residence time in water amounts to 25 days, indeed yielding an ecosystem characterization factor of \(\text{EDF}_{\text{aqu,w}} = 25 \cdot 757 = 19,000\) [PAF m²/day/kg]. As the transferred fraction from air to water amounts to 0.05% (Section 4.1), the factor for an emission to air is more than two orders of magnitude smaller: \(\text{EDF}_{\text{aqu,a}} = 0.05\% \cdot \text{EDF}_{\text{aqu,w}} = 9.5\) [PAF m³/day/kg]. A further description of the applied ecotoxicity model can be found in the corresponding section of the Supporting information.

6. Discussion

Hereafter, advantages and improvements of the matrix framework are discussed in detail.

This framework provides a high level of flexibility in terms of updating and adding supplementary functionality such as new exposure pathways or new effect types. For an update or extension of the model by e.g. adding a new compartment, exposure pathway, or effect type, the corresponding matrices are simply extended by new rows or columns. This can particularly contribute to the seamless inclusion of latest scientific findings into policy based decision tools. For example, introducing emissions to indoor air is simply carried out by adding a compartment, i.e. an additional row and column in the fate matrix, and an additional column in the exposure and intake fraction matrices.

Interpretability of intermediate and final results is facilitated, e.g., allowing the identification of dominating exposure pathways. Keeping the communication of results and findings of a model application in the focus, this can contribute to the identification of the main points of interest of a modeling exercise. For example, it would enable an automated extraction of those findings (such as the dominant exposure pathway for humans) into a more policy/decision relevant summary report.

Model comparability and evaluation is well supported, as the four matrices for fate, exposure, effects, and severity contain almost all of the intermediate results or information produced by the models in a clear and interpretable way. Their low dependency on scenario-related parameters, such as amount and place of emission is an important characteristic in this context. For a given compartment height and advection rate, the fate and the exposure matrices are independent of the
compartment size and volume, therefore facilitating the comparison of models run for different landscape characteristics. Hence, a direct comparison of the matrices of two models yields significant advantages against comparing single results which often need to be made consistent.

Computational efficiency is significantly increased as the matrix framework allows characterizing multiple emission scenarios in one calculation run (e.g. emission to air, water or soil, or variations in the amounts released), while current multimedia models usually need to run separately for each emission scenario. All the information included in the four matrices is now actually kept until the end and can thus be used for the final result. This yields at least a three times faster calculation plus the time needed to prepare each model run in the context of comparative assessments, where usually three emission compartments (air, water, and soil) are considered separately. Furthermore, for each chemical the model would only need to be run once, and then the matrices can be stored and directly used for further calculations with varying emission amounts and compartments. This calculation efficiency is not essential for non-spatial models, but can become important when going for spatially resolved calculations on a large number of chemicals.

Multidisciplinary work is strongly facilitated enabling the linkage of different models from various disciplines together, since each of its modules defines a clear interface of intermediate results. In recent years the comparison and integration of tools from different scientific fields, notably (conservative) environmental risk assessment and comparative risk/impact assessment, has become an important topic (Pant et al., 2004; Sleeswijk et al., 2003; Cowell et al., 2002; Olsen et al., 2001; Owens, 1997; Guinée and Heijungs, 1993). Creating flexible toolboxes has been seen as an important step towards integrating valuable insights from varying areas of research. They can be complemented by scientists from several multidisciplinary backgrounds and can be explored, as well as used somewhat independently from the originally intended context of each contribution.

7. Conclusions

It was demonstrated how the matrix framework is established and applied to any toxicological multimedia fate, multipathway exposure and effect model. An extensive interpretation of each matrix was presented, including in a spatially explicit context.

This framework has been reviewed and validated by an independent panel of experts in the field of fate and exposure of pollutants and Ecotoxicology within a UNEP/SETAC workshop (Jolliet et al., 2006) and was suggested as a starting-point for new advances in modeling environmental toxic releases. It has been adopted by the UNEP/SETAC Life Cycle Initiative as a framework providing a flexible library which can be filled with different models for each sub-calculation step (Jolliet et al., 2006). Instead of adopting one universal model, the framework can host several models which more flexibly suit specific applications. For example, they could cover specific substance classes or exposure scenarios in the context of policy- and decision-support (e.g. assessing population exposure to disperse emissions into the environment, worker exposure, and indoor air exposure).

While this paper focuses on steady-state modeling, it should be noted that in principle this framework can also be applied to dynamic systems, solving the mass balance differential equation system using eigenvectors and eigenvalues, as extensively explored by Charles (2004) for different model settings. However, the other matrices would then eventually need to be populated applying data reflecting the respective time step(s). Other subjects to be further investigated are the possibilities of inclusion of processes directly related to the model structure, such as metal speciation and the inclusion of uncertainty calculations within the framework. Accounting for non-linear relationships could be explored using “local linear solutions”.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.envint.2007.01.004.

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