View metadata, citation and similar papers at core.ac.uk
brought to you by CORE
provided by RERO DOC Digital Library

Corner: UNEP/SETAC Life Cycle Initiative

Establishing a Framework for Life Cycle Toxicity Assessment

Findings of the Lausanne Review Workshop

Olivier Jolliet[1,2]*, Ralph Rosenbaum[1], Peter M. Chapman[3], Tom McKone[4], Manuele Margni[5], Martin Scheringer[6], Nico van Straalen[7] and Frank Wania[8]

[1] Ecole Polytechnique Fédérale de Lausanne, Institute of Environmental Science and Technology, 1015 Lausanne, Switzerland
[2] Center for Risk Science and Communication, Department of Environmental Health Sciences, School of Public Health, University of Michigan, Ann Arbor, MI 48109, USA
[3] Golder Associates, North Vancouver, BC, Canada V7P 2R4
[4] School of Public Health, University of California, Berkeley, California, USA
[5] CIRAIG, Ecole Polytechnique de Montreal, C.P. 6079, Montreal, Canada, H3C 3A
[6] Swiss Federal Institute of Technology, Zurich, Switzerland
[7] Free University, Amsterdam, The Netherlands
[8] University of Toronto at Scarborough, Scarborough, Ontario, Canada

With contributions of the workshop participants[9]

* **Corresponding author** (ojolliet@umich.edu)

DOI: http://dx.doi.org/10.1065/lca2006.03.002

In December 2003, The UNEP/SETAC Life Cycle Initiative convened a review workshop in Lausanne Switzerland in collaboration with the European project team OMNIITOX (Molander et al. 2004). The aim of this review workshop was to identify how models can be used to establish toxicity for Life Cycle Impact Assessment, what attributes such models need, and then to identify one or more models that meet these requirements and what it would take to establish and endorse a base model. To achieve this goal, experts in the field of fate and exposure of environmental contaminants (Tom McKone[4], Martin Scheringer[6] and Frank Wania[8]) and in ecotoxicology (Peter M. Chapman[3] and Nico van Straalen[7]) were invited to review and comment on a preliminary proposal for a base model as developed within the OMNIITOX project. Beyond the OMINIITOX project itself, the main results for this workshop are recommendations on an overall framework for toxicity assessment and on a process to arrive at recommendations on characterization factors.

[9] Besides the organizers and reviewers, other workshop participants were: Alain Dubreuil – Chair of the LCA steering committee of SETAC North America, Helias Udo de Haes – Director of the SETAC-UNEP Life Cycle Initiative, Olivier Jolliet – LCIA Programme Manager of the Life Cycle Initiative, Michael Hauschild – Delegate of the OMNIITOX steering committee, Johan Tivander – OMNIITOX coordinator, Chen Sau Soon – Environmental and Energy Technology Centre Malaysia, Guido Sonnemann – UNEP secretary of the Life Cycle Initiative, Bo Wahlström – Senior Scientific Advisor UNEP chemicals in Geneva, David Pennington – EU-Joint Research Centre Ispra, Marta Schuhmacher – Tarragona University, and from the OMNIITOX team: Jeroen Guinée (CML), Arjan de Koning (CML), Henrik Fred Larsen (DTU), Stig Irving Olsen (DTU), Till Bachmann (USTUTT), Ralph Rosenbaum (EPFL), Manuele Margni (EPFL), Jérôme Payet (EPFL), Rana Pant (P&G), Diederik Schowanek (P&G), Franz Møller Christensen (JRC-ECB).

To establish an agreed base model within the Life Cycle Initiative the following main milestones were set:

1. It is important to first agree on a roadmap and a process leading to a proper base model or framework.
2. Next develop or adopt a suitable model framework. This framework will serve as a library of model elements, and thus needs to be stable in time and yet flexible enough to 'house' changing parameter values and algorithms within model elements, as science advances.
3. The model components characterising chemical fate, exposure and toxic effects have to be adopted or further developed. These components will change over time, constantly being adapted to the latest knowledge and scientific advances.
4. To successfully implement this model, sponsors need to be convinced about the need for a long term support of the system.

Main findings and recommendations were as follows:

The workshop experts recommended the proposed matrix algebra framework (see **Appendix I**) to the Life Cycle Initiative as an elegant and efficient solution due to its stable, yet flexible structure. It provides clearly defined interfaces connecting different parts of the modelling chain (fate, exposure and effects). Updates and even extensions of model components are easily implemented and it serves as a repository of information on the algorithms/model elements needed.

Another important part of the review was the concept of a tiered modelling approach which consists of a simplified base model, statistically derived from the more sophisticated mechanistic base model, and accounts for sensitivity and

© 2006 ecomed publishers (Verlagsgruppe Hüthig Jehle Rehm GmbH), D-86899 Landsberg and Tokyo • Mumbai • Seoul • Melbourne • Paris

dominant mechanisms in the emissions-to-exposure pathway. The participants suggested that the chemical training set for the statistical analysis should be based on hypothetical chemicals to avoid a bias. As more data and information become available, the reviewers recognized the value of using complex (data rich) and simplified (data poor) base models to provide a compatible tiered strategy. This provides adaptive modelling and assessment, while preserving consistency by the strong connection between the two tiers/models. This original approach could approximate a solution to the paradox between broad substance coverage with a cost of higher uncertainty and the alternative of smaller coverage but with less uncertainty; the latter being quantified by means of, for example, confidence limits on the regression parameters.

Based on these findings, the roadmap leading to recommended characterisation factors as a major result of the workshop was established as follows:

The matrix structure, as proposed by the developers of OMNIITOX, was adopted as the framework.

- The model library will consist of:
  - Processes and matrix factors,
  - Substance data and estimation tools,
  - Geographic data (landscape data, etc.).
- The OMNIITOX proposals will provide examples as the basis for a call for input from researchers around the world.
- Provided that funding will be available, the chosen model elements will be implemented.

In the more specific area of ecological endpoints, the reviewers noted that the focus on effect concentrations (EC) expressed in terms of the fraction x of species impacted (ECx) are appropriate for LCA objectives rather than the no observed effects concentration (NOEC) values that are test-concentration-dependent. Similarly, the use of a harmful concentration to 50 per cent of species [HC50 (of EC50)] based on the geometric mean of all species is also appropriate for a comparative assessment. Chronic data were considered as a preferable to acute data as a basis for toxicity, but the use of acute data was accepted as a basis for extrapolating chronic values. Biomagnification should be included when known and secondary poisoning is also relevant but problematic to handle at present. The relationship to species sensitivity distributions needs further clarification. It is probably not necessary when using only a geometric mean for midpoint assessment, but this approach may be needed for damage modelling. It was strongly recommended to also include effects in sediment, soil, terrestrial, and ocean compartments to avoid biases. Aggregation remains an issue for further discussion as to whether the score from the most critical compartment should be retained or if all scores should be summed based on damage modelling.

In addition, a detailed review has been conducted on the fate modules for air, fresh and marine water with the respective sediment compartments, soil, vegetation, human exposure as well as on an optional approach for speciating chemicals. A main issue raised by the reviewers is the "applicability to a very wide variety of substances", because the ability of similar models to adequately describe the environmental behaviour of many types of organic chemicals has not been tested. This means that models need to be significantly adapted and customized for different subsets of chemicals, checking that:

- The process descriptions that are part of the model are adapted to and applicable to the considered subset of chemicals that the model will be used for.
- There are some processes missing in the model, which are important for a subset of substances.

This points to the need for creating a library of process algorithms or even submodels which are well adapted to given sets of substances. Special care should be taken for the gas/particle partitioning of polar substances or for the long term fate of PCBs which cannot be adequately described without considering solid phase diffusion in soils – a process that has not been considered in most multimedia fate models. For single-medium chemicals with extreme partitioning properties, the multimedia modelling approach is not only superfluous, but may also lack the capacity to discriminate among chemicals whose behavior depends primarily on highly uncertain degradation properties.

Additional comments pointed out specific details in the modelling that needed further attention from the OMNIITOX project participants (see enclosed reviewer reports). Many of the statements made by the experts were directly taken into account on a detailed level (processes to incorporate in the model, specific equations for processes, specific data to use for the description of the environment, etc.), leading to changes that were introduced into the base model. However, some of the remarks were of a character deemed impossible to incorporate within the given time frame and have been kept for further research.

Further identified and discussed issues to address were:

- Pros and cons of discounting of future impacts for very persistent substances,
- Identification of the need for data and processes relevant for other climate regions,
- Capacity building in environmental modelling outside regions such as Europe, North America, South Korea and Japan, …,
- Synergy effects helping to identify needs for ERA,
- The possibility of reviewing models and results at the end of OMNIITOX.

Appendix I: The OMNIITOX base model framework

The present framework is described in detail in Rosenbaum et al. (2004).

1 Human Health Impact Characterization

The prediction of the impact of a chemical when released into the environment is vital for decision making based on

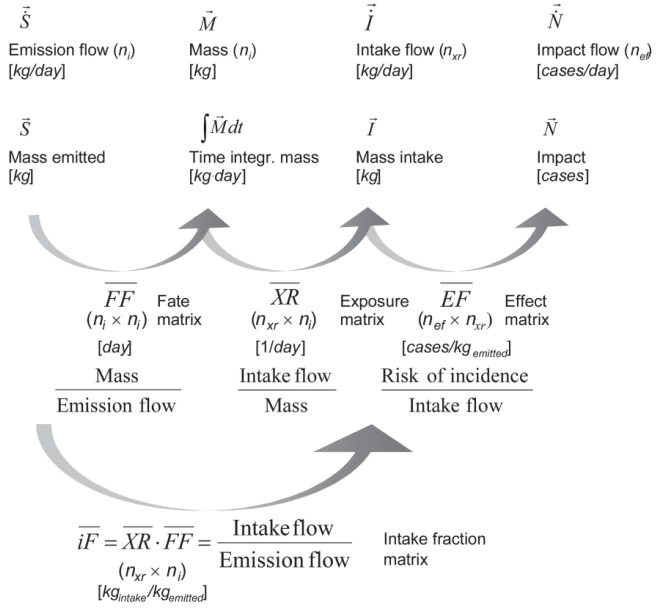

Fig. 1: General scheme for human toxicity

environmental assessments, e.g. 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.

Assessing toxicological effects on human health of a chemical emitted into the environment, whether released on purpose (e.g. pesticides), as a by-product from industrial processes implies a cause-effect-chain assessment (**Fig. 1**): 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 eventually to the resulting number of cases of various morbidity risks (risk vector ($\vec{N}$ in *number of cases*).

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

$$\vec{N} = \overline{EF} \cdot \overline{XR} \cdot \overline{FF} \cdot \vec{S} = \overline{EF} \cdot \overline{XR} \cdot \vec{M} = \overline{EF} \cdot \overline{iF} \cdot \vec{S} = \overline{EF} \cdot \vec{I}$$

expressed in *cases/day*.

The same equation can be written to relate mass emitted, time integrated mass, and time and volume integrated number of cases (at steady state, with linear modeling):

$$N = \overline{EF} \cdot \overline{XR} \cdot \overline{FF} \cdot \vec{S} = \overline{EF} \cdot \overline{XR} \cdot \int \vec{M} dt = \int \overline{EF} \cdot \overline{XR} \cdot \overline{FF} \cdot \vec{S} dt$$
$$= \int \overline{EF} \cdot \overline{iF} \cdot \vec{S} dt = \int \overline{EF} \cdot \vec{I} dt$$

expressed in *cases*, where:

Fate ($\overline{FF}$) 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 $\overline{FF}$; a column entry denotes the initial source compartment *m* and a row entry denotes the final compartment *i*, i.e. where the chemical is transferred into. 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*]. It is equivalent to the effective residence time of the chemical in compartment *i* [*day*] multiplied by the fraction transferred from the source medium *m* to medium *i* [*dimensionless*] (see Fig. 1).

Exposure ($\overline{XR}$) 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) or by dermal contact. It is quantified by the exposure route matrix $\overline{XR}$ that contains exposure factors (or exposure rates); a column entry denotes a final environmental compartment *i* and a row entry denotes the exposure route *xr* (e.g. ingestion, inhalation or dermal). The exposure factor $XR_{xr,i}$ [1/*day*] is the equivalent rate of ingestion of the medium by humans. For intake, for example, it correspond to the fraction of the total mass of drinking water ingested daily by humans. The inverse of this coefficient therefore represents the equivalent time required by the population to inhale or ingest the whole mass in the medium (see Fig. 1).

Effects ($\overline{EF}$) 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 $\overline{EF}$; a column entry denotes an exposure route *xr* (e.g. inhalation, ingestion or demal) and a row entry denotes an effect type *ef* (e.g. cancer, non-cancer). The effect factor $EF_{ef,xr}$ [*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 [dimensionless] in the exposed population per unit mass ingested or inhaled [kg_{intake}] – itself due to an emission source in compartment *m* (see Fig. 1).

The three matrices $\overline{FF}$, $\overline{XR}$, $\overline{EF}$ are combined to estimate the characterization factor matrix called human damage factor matrix:

$$\overline{HDF} = \overline{EF} \cdot \underbrace{\overline{XR} \cdot \overline{FF}}_{\overline{iF}} = \overline{EF} \cdot \overline{iF} \quad .$$

The human damage factor $HDF_{ef,m}$ [*number of cases/*$kg_{emitted}$] can be interpreted as the increase in population risk of a morbidity *ef* due to an emission in compartment *m* [$kg_{emitted}$].

The fate and exposure matrices can also be aggregated into an intermediary matrix: the intake fraction matrix $\overline{iF}$: a column entry denotes an emission compartment *m* and a row entry denotes the exposure route *xr* (e.g. ingestion, inhalation or dermal). The intake fraction $iF_{xr,i}$ [$kg_{intake}/kg_{emitted}$]can be interpreted as the fraction of an emission in an initial com-

partment *m* that is taken in by the overall population through a given intake route *xr* (Bennett et al. 2002).

The framework could also be completed by a damage matrix to distinguish between severities of adverse effects and thus accounting for differences between e.g. a lethal effect and a reversible skin irritation. It could provide results in terms of Years of Life Disabled (YLD) for different illnesses, total Years of Life Lost (YLL) or eventually Disability Adjusted Life Years (DALYs), using for the latter a weighting scheme between the respective severities of the YLD as provided for different morbidities (Murray and Lopes 1996).

2 Ecotoxicological Impact Characterization

In the same way a human population is potentially affected by a chemical release, an ecosystem can be affected. Thus, the link between the chemical emission and its impact on ecosystems can be established and modeled in a similar way. The underlying cause-effect-chain is in principle the same as for human health but with a few differences: the interface between fate and effect is defined at the level of the resulting mass in environmental compartments and the effect matrix therefore reflects directly both exposure and effect (**Fig. 2**).

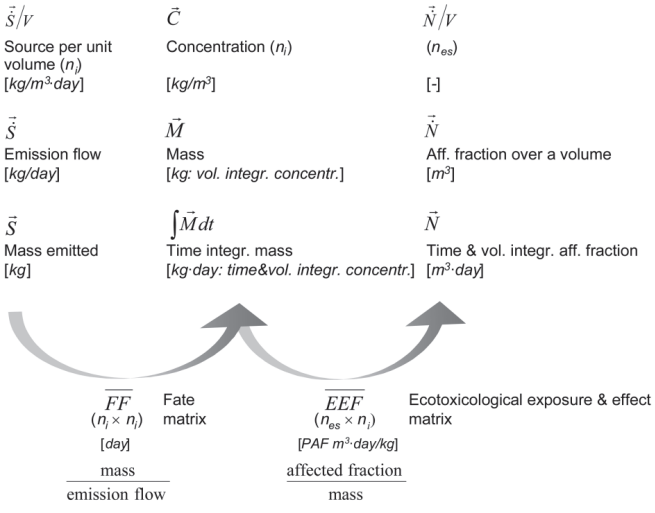

Fig. 2: General scheme for ecotoxicity

The cause effect chain links the emission source (emission flow vector $\vec{S}$ in *kg/day*) to the mass in the environment compartments (mass vector $\vec{M}$ in *kg*) and eventually to the volume integrated increase in the affected fraction of species due to an emission into a compartment *m* (volume integrated affected fraction of species risk $\vec{N}$ in *PAF* m^3):

$$\vec{N} = \overline{EAF} \cdot \vec{M} = \overline{EEF} \cdot \overline{FF} \cdot \vec{S} = \overline{EDF} \cdot \vec{S}$$

The same equation can be written to relate mass emitted, time integrated mass, and time and volume integrated impact in affected fraction:

$$N = \int \overline{EEF} \cdot \vec{M} dt = \int \overline{EEF} \cdot \overline{FF} \cdot \vec{S} dt = \overline{EEF} \cdot \overline{FF} \cdot \int \vec{S} dt$$

expressed in *PAF* $m^3 days$.

Where the cause-effect-chain is reduced to two steps:

Fate. The fate matrix $\overline{FF}$ [*day*] is exactly the same as for human health impact characterization.

Exposure and Effects. The ecotoxicological effect factor (EEF) quantifies the fraction of species in an ecosystem which are affected by a given level of exposure. It is quantified by the ecotoxicological effect matrix $\overline{EEF}$; a row entry denotes the affected ecosystem *es* (e.g. aquatic, marine or terrestrial) and a column entry denotes a final compartment *i*. The ecotox effect factor $EEF_{es,i}$ [$PAF\ m^3/kg$] can be interpreted as the volume integrated increase in affected fraction of species, per unit of chemical mass increase in compartment *i* [*kg*].

The fate and effect matrixes can be multiplied to estimate the characterization factor matrix called ecotoxicological damage matrix: $\overline{EDF} = \overline{EEF} \cdot \overline{FF}$. The $\overline{EDF}$ matrix contains ecotoxicological damage factors; a column entry denotes the initial emission compartment *m* and a row entry denotes the affected ecosystem *es* (e.g. fresh water, marine water, terrestrial, etc.).

References

Bennett DH, McKone TE, Evans JS, Nazaroff WW, Margni MD, Jolliet O, Smith KR (2002): Defining Intake Fraction. Environ Sci Technol 36, 207A–211A

Molander S, Lidholm P, Schowanek D, Recasens M, Fullana i Palmer P, Christensen F, Guinée J B, Hauschild M, Jolliet O, Carlson R, Pennington DW, Bachmann TM (2004): OMNIITOX – Operational Life-Cycle Impact Assessment Models and Information Tools for Practitioners. Int J LCA 9 (5) 282–288 <http://www.omniitox.net>

Murray C, Lopez A (1996): The Global Burden of Disease, a Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factors in 1990 and Projected to 2020. GlobalBurden of Disease and Injury Series, Vol. 1 & 2, Harvard Schoolof Public Health, World Health Organization and World Bank

Rosenbaum R, Pennington D, Margni M, Jolliet O (2004): Development of the OMNIITOX Base Model, OMNIITOX European project deliverable – D32

Received: February 28th, 2006
Accepted: March 1st, 2006
OnlineFirst: March 2nd, 2006

The findings of the Portland Review Workshop 'Dose-Response Modeling for Life Cycle Impact Assessment' (November 2004) have likewise been summarized for Int J LCA. The Workshop Report (Thomas E. McKone, Amy D. Kyle, Olivier Jolliet, Stig Irving Olsen and Michael Hauschild) has been published in Int J LCA No. 2, pp. 137–140 (March issue).