Eidgenössische Technische Hochschule Zürich

SCHWERPUNKTPROGRAMM UMWELT
SCHWEIZ. NATIONALFONDS ZUR FÖRDERUNG DER WISSENSCHAFTLICHEN FORSCHUNG
PROGRAMME PRIORITAIRE ENVIRONNEMENT
FONDS NATIONAL SUISSE DE LA RECHERCHE SCIENTIFIQUE
PRIORITY PROGRAMME ENVIRONMENT
SWISS NATIONAL SCIENCE FOUNDATION

Chemikalienbewertung in Ökobilanzen

Vorbereitende Unterlagen zum 10. Diskussionsforum Ökobilanzen vom 28. April 1999 an der ETH Zürich

Die Diskussionsforen 'Ökobilanz' werden durch das Schwerpunktprogramm Umwelt des Schweizerischen Nationalfonds, Bern, finanziell unterstützt

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ISBN 3-906734-07-2

Zürich, April 1999

Bezugsadresse:

Eidgenössische Technische Hochschule
Laboratorium für Technische Chemie
Gruppe Sicherheit & Umweltschutz
ETH Zentrum UNK
CH - 8092 **Zürich**

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MARTIN SCHERINGER

Das Umweltverhalten von Stoffen – eine Lücke im Life-Cycle Impact Assessment

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Übersicht

1. Problem: wie kann das Umweltverhalten von Stoffen im LCIA erfasst werden?
2. Beispiel: Ökobilanz eines optischen Aufhellers im Vergleich zur Peroxidbleiche
   - Technisches System: Holzschliff, Bleichverfahren
   - Ökobilanz: Produktion des Aufhellers und der Bleich-Chemikalien
   - Umweltchemische Betrachtung: Aufheller und Holz-Inhaltsstoffe im Flusswasser
3. Folgerungen

Problem

Wirkungskategorien im LCIA sind überwiegend

- Für weiträumige oder globale Effekte (GWP, ODP, AP) definiert;
- Mit dem Energieverbrauch korreliert.

Das spezifische Umweltverhalten von Stoffen wird kaum erfasst (Verteilung, Umwandlung, Wirkungen mit spezifischen Mechanismen).

Mögliche Ansätze:

- Fate-Faktoren (O. Jolliet)
- Gebiets-spezifische Charakterisierungsfaktoren (J. Potting)
- Hier: Stoffvergleich mit Persistenz, Reichweite, PEC/PNEC-Quotient

Bleichverfahren

Holzschliff (mechanical pulp):

- Robust, hohe Ausbeute
- Vergilbt schnell (Lignin)

Bleichverfahren:

- Peroxid – wandelt Lignin chemisch um, so dass Chromophore zerstört werden
- Optischer Aufheller – kompensiert gelbe Farbe durch verstärkte Emission von blauem Licht

Ökobilanz

funktionelle Einheit:
1 Tonne gebleichter Holzschliff (mechanical pulp)

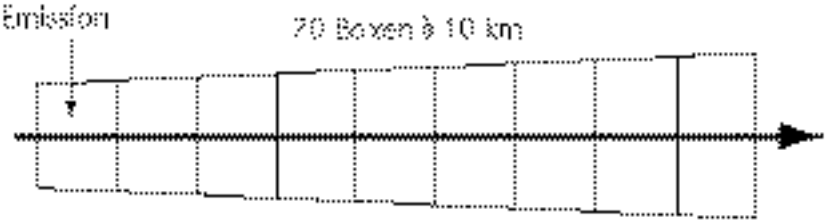
Systemgrenzen:
Produktion u. Anwendung von a) Bleichchemikalien b) opt. Aufheller

LC-Inventory:
Ressourcenverbrauch & Emissionen

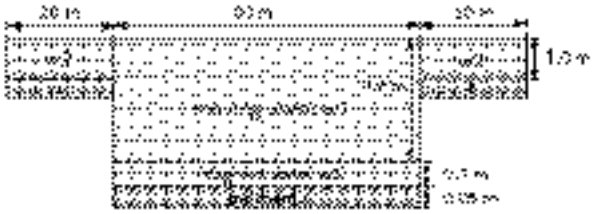
LC-Impact Assessment:
Wirkungskategorien
GWP, AP, NP,
POCP etc.
Eco-indicator 95

Flußmodell

- Prozesse: Strömung, Sedimentation, Abbau
- Gesamtsystem:



- Querschnitt:



ANNEKE WEGENER SLEESWIJK

GLOBOX - an add-on LCA multimedia characterization model including sea compartments

Centre of Environmental Science Leiden University (CML), Leiden, The Netherlands

1 A GLOBAL MULTIMEDIA MODEL FOR LCA-USE WITH SPECIFIC ADAPTATIONS FOR NORTH SEA AND METALS

basis: CML model DYNABOX (based on USES 1.0)

new elements:

- adaptations for use in LCA
- adaptations for the inclusion of sea compartments
- simple upgrading to global level
- some adaptations for the inclusion of metal emissions
- updating of some program elements, according to EUSES/SimpleBox 2.0

Adaptations for use in LCA

1. closing the environmental system

- USES 1.0: open systems, outflows from air and water compartments leave environmental system
  _ not involved in assessment
- solution by Guinée ***et al***. (1996):
  - minimize outflows to seawater and sea-air
  - minimize flows to water compartment to avoid accumulation in freshwater systems (rainwater runoff and leaching)
- this project:
  closed system, existing of four interconnected environmental systems:
  North Sea, ice-free ocean, The Netherlands, continents + sea ice

2. adding exchanges between equivalent compartments of different environmental systems

- advective streams between different air compartments
- advective streams between different sea compartments
- advective streams from freshwater compartments to sea compartments
- advective streams from freshwater suspended matter to sea suspended matter compartments

3. adapting effect factors for LCA-use

- risk assessment models: ecotoxicity effect factors contain `safety factors' to adjust for data uncertainties
- LCA: ecotoxicity effect factors should reflect most probable toxicity ratios between substances

  → pure toxicity data used as effect factors

4. composing equivalency factors from exposure figures and effect data

for each emission compartment:

equivalency factor = $\sum$ time-integrated amount in compartment ***i***
× exposed population
× effect factor

for each substance equivalency factors for the following 4 impact categories:

– human toxicity
– freshwater aquatic ecotoxicity
– seawater aquatic ecotoxicity
– terrestric ecotoxicity

and for the following 18 emission compartments:

– North Sea and ocean: air, seawater, sea suspended matter
– The Netherlands and the world: air, fresh water, fresh water suspended matter, natural soil, agricultural soil, industrial soil

Adaptations for the inclusion of sea compartments

1. gathering sea-specific parameters

2. including exposure to contaminants in sea water

HUMAN FISH INTAKE (NETHERLANDS AND WORLD AVERAGE)

parameter	value
daily intake of fresh fish by Dutch people [kg wet fish day]	0.0005
daily intake of North Sea fish by Dutch people [kg wet fish day]	0.0072
daily intake of ocean fish by Dutch people [kg wet fish day]	0.0018
daily intake of fresh fish by world population [kg wet fish day]	0.0065
daily intake of ocean fish by world population [kg wet fish day]	0.0060

NORTH SEA SYSTEM PARAMETERS

parameter [unit]	value
total area [m^2]	0.55×10^{12}
average total depth [m]	90
average mixing depth [m]	90
average concentration of suspended matter [kg m^3]	0.003
average production rate of suspended matter [kg s^{-1}]	8.7×10^{3}
sum of riverine inflows from The Netherlands [m^3 s^{-1}]	3113
average concentration of suspended matter in riverine inflows from The Netherlands [kg m^{-3}]	0.029
sum of riverine inflows from Western Europe (excluding The Netherlands) [m^3 s^{-1}]	1876
average concentration of suspended matter in riverine inflows from Western Europe (excluding The Netherlands) [kg m^{-3}]	0.056
sum of oceanic inflows [m s^{-1}]	1.84×10^{6}
average discharge of suspended matter [kg s^{-1}]	95
average mixing depth of sediment compartment [m]	0.1
average depth of aerobic top layer of sediment compartment [m]	0.01
average sediment resuspension rate [m^3 s^{-1}]	
average rainrate [m s^{-1}]	1.9×10^{-8}
average surface area of aerosol particles in North Sea air [m^2 m^{-3}]	1.5×10^{-4}
temperature at the air-water interface [K]	282

Simple upgrading to global level

adaptations until now:

- temperature and rainrate continents: values of moderate climate
- seawater temperature and rainrate: North Sea values
- windspeed and –direction: North Sea values
- land and water surface: global values
- ratio land/water of continents: Western-European value
- human intake of different food products: global averages (FAO)

Some adaptations for the inclusion of metal emissions

1. Determination of parameter needs for metals and subsequent model adaption
no model derivations, based on Kow or vapour pressure

→ to be directly collected:

– partition coefficients

– bioconcentration factors

ESTIMATED PARAMETER VALUES, ASSIGNED TO METAL IONS

parameter	value
air-water partition coefficient [–]	
mercury	1.3×10^{-5}
other metals	0
Henry's law coefficient [Pa m^3 mol]	
mercury	0.03
other metals	0
scavenging ratio [–]	1×10^{5}
fraction of chemical in air, associated to aerosol particles ($Fass_{aer}$) [–]	
zinc	
mercury	0.99
other metals	0.05
	0.95
bioconcentration factor from air to plant ($BCF_{air\text{-}plant}$) [$(kg_{chem}\ kg_{wet\ stem}^{-1})/(kg_{chem}\ m_{air}^{-3})$]	0*

* Set to zero by lack of data.

2. Inclusion of activity coefficients

ACTIVITY COEFFICIENTS, CALCULATED FOR DIFFERENT ELECTROVALENCE VALUES OF THE IONS TO BE ASSESSED

electrovalence (+ or -)	activity coefficient in seawater
0	1
1	0.71
2	0.25
3	0.047
4	0.0043

Kp_sedsea = ACTCOEFF × Kp_sed/

(Kp_sed × (1-ACTCOEFF) × (V[sedsea]/V[watersea]) × RHOsolid + 1)

Updating of program elements, according to EUSES

1. **Partition coefficients and degradation rate constants temperature dependent**
2. **Aquatic biota assumed to be in thermodynamic equilibrium with water compartment**

Some results

emission compartment: North Sea water

impact category: aquatic ecotoxicity

substance	***EC_{50} [mg/m^3}***	***DT_{50} [d]***	***eq. factor***
cadmium	553	infinite	1.45E8
fluoranthene	500	290	1.48E5
chromium	5601	infinite	6.86E7
xylene	5716	18	1.10E4
cadmium	***553***	***36500***	***9.71E5***

Future plans

- refine global unit world
  - collection of geographic information
  - collection of exposure information
- add terrestrial vegetation compartment (EUSES)
- investigate useful supplements from other models
- investigate possibility to add module to deal with degradation, conversion and metal speciation
- refinement of effect modelling
- application of model to set of substances

MONIKA HERRCHEN

Möglichkeiten und Notwendigkeiten der Verknüpfung von Elementen der Risikoabschätzung und Produkt-LCA: eine kontroverse Diskussion

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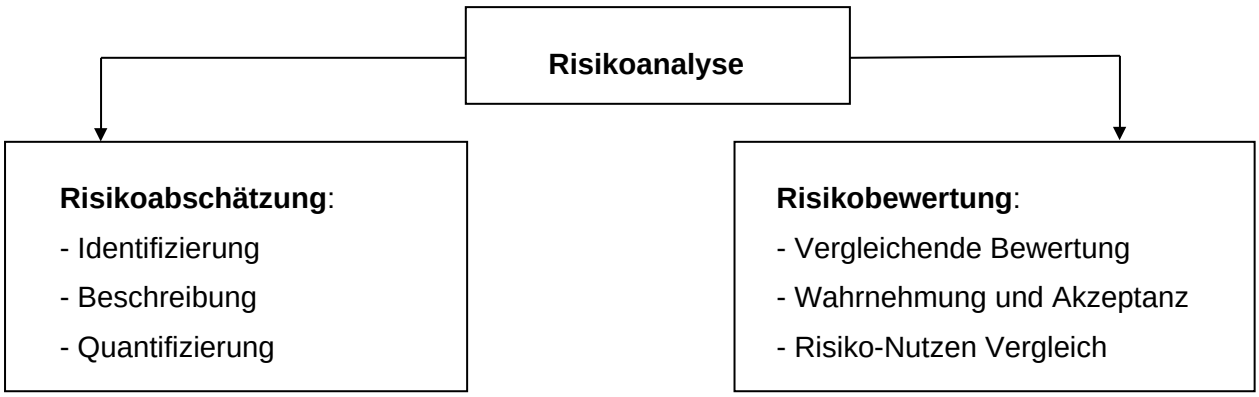
Elemente der Risikoabschätzung in der Produkt-LCA: eine paradoxe Situation

- raum- und zeit**un**abhängige Stoffströme in der Sachbilanz resultieren in Angaben zu (öko-) toxikologischen Potentialen
- Aussagefähigkeit und zielführende Nutzbarkeit der Potential-Angaben ??
- Erfahrung mit verschiedenen Anwendern, auch Ziel-unabhängig:
  a) die Ergebnisdiskussion zielt auf Aussagen zur Schadenssignifikanz und zum Risiko
  b) die Ergebnisdiskussion behält konsequent die Potentialdarstellung bei mit dem Ziel einer Aggregation oder einer Gruppenbildung über mehrere Wirkkategorien sowie anschließender Wichtung

Elemente der Risikoabschätzung in der Produkt-LCA: Ansätze zur Kombination

- getrennte Nutzung der Instrumentarien mit Auswahl entsprechend der Fragestellung
- iterative Nutzung von LCA und Risikoabschätzung
- Integration der Methoden

Elemente der Risikoabschätzung



Definitionen der Risikoabschätzung

- Risk assessment = probabilistische Aussage:
  „the estimation of the probability of clearly defined environmental effects occuring as a result of the exposure to a chemical“ (OECD, 1988)
- Risk assessment = probabilistische Aussage:
  „estimation of the probability or likelihood of undesirable events such as injury, death or the decrease in the mass or productivity of fish, wildlife. Risk is a function of hazard and exposure; ecological risk is a function of (eco)toxicological hazard and environmental exposure“ (U.S.EPA, 1986)
- Risk assessment = deterministische Schätzgröße:
  „Comparing the concentration in the environmental compartments (PEC) with the concentration below which unacceptable effects on organisms will most likely not occur (PNEC)“ (TGDs, 1994)

Ansatz: getrennte Nutzung der Instrumentarien mit Auswahl entsprechend der Fragestellung

- life-cycle thinking wird in den Mittelpunkt gestellt
- auf Basis der Erfahrung mit den betrachteten Produkten (und Prozessen!) werden für die Lebenswegabschnitte entlang der Wertschöpfungskette problemangepaßte Instrumentarien (Stoffstromanalyse, Umweltrisikoabschätzung und -management, techn. Arbeitsplatzsicherheit, Umgang mit Gefahrstoffen...) eingesetzt
- Anwendung: firmeninterne Entscheidungsprozesse, Öko-Design

Ansatz: getrennte Nutzung der Instrumentarien mit Auswahl entsprechend der Fragestellung

Pro:

- keine inhaltliche Überfrachtung und Fehlnutzung von Instrumentarien
- freiwillige Selbstverpflichtungen werden kombiniert mit Befolgung von Auflagen; Konsequenz eventuell Deregulierung
- gezielter Einsatz von finanziellen und personellen Ressourcen

Contra:

- Life-cycle thinking wird nur begrenzt durchgeführt
- „Erfahrung kann täuschen und Problemfelder übersehen“; Betriebsblindheit

Ansatz: iterative Nutzung von LCA und Risikoabschätzung

- Iteration bestehend aus:

1) Anwendung einer LCA-Screening-Methode zur Identifizierung umweltrelevanter, kritischer Emissionen
2) Identifizierung der Emissions-Quellen entlang des Produkt-Lebenszyklus
3) Möglichkeiten: hohe Zahl von Quellen mit geringen Stofffrachten oder geringe Zahl von Quellen mit hohen Stofffrachten (Abschneidekriterien zur Entscheidung)
4) Fokussierung auf die identifizierten kritischen Prozesse (!) und Durchführung einer orts- und/oder zeitabhängigen Risikoanalyse unter Beibehaltung des Produktbezugs (respektive der funktionellen Einheit)

Screening-Methode: Äquivalenzfaktoren für das Kompartiment **Wasser** (Primärkompartiment für die Emission):

Wirkpotentialklasse 0 mit Eigenschafts-kombinationen:	**Wirkpotentialklasse A** mit Eigenschafts-kombinationen:	**Wirkpotentialklasse B** mit Eigenschafts-kombinationen:	**Wirkpotentialklasse C** mit Eigenschafts-kombinationen:	**Wirkpotentialklasse D** mit Eigenschafts-kombinationen:
· NOEC > 1 mg/l oder · LC_{50} > 10 mg/l · abbaubar · nicht akkumulierend	· NOEC > 10 mg/l oder · LC_{50} > 100 mg/l · persistent · und/oder akkumulierend ------- oder ------- · LC_{50} < 10 mg/l · abbaubar · nicht akkumulierend	· NOEC = 1 - 10 mg/l oder · LC_{50} = 10 - 100 mg/l · persistent · und/oder akkumulierend	· NOEC = 0,1 - 1 mg/l oder · LC_{50} = 1 - 10 mg/l · persistent · und/oder akkumulierend	· NOEC < 0,1 mg/l oder · LC_{50} < 1 mg/l · persistent · und/oder akkumulierend
⇩	⇩	⇩	⇩	⇩
Wirkpotentialfaktor: **0**	Wirkpotentialfaktor: **1**	Wirkpotentialfaktor: **10**	Wirkpotentialfaktor: **100**	Wirkpotentialfaktor: **1000** („**Rote Lampe**“, RL)

zunehmend kritische Umweltauswirkungen aufgrund der Stoffeigenschaftskombinationen

Alle anderen Eigenschaftskombinationen werden in die Wirkpotentialklasse 0 gruppiert bzw. mit einem Wirkpotentialfaktor von 0 versehen

Erläuterungen:

abbaubar = „ready degradable“ oder „inherent“ als Ergebnis gemäß OECD-Tests für organische Stoffe

persistent = „non-degradable“ als Ergebnis gemäß OECD-Tests für organische Stoffe

nicht akkumulierend = $logP_{ow} < 3$ ($logP_{ow}$ ist der Logarithmus des Oktanol-Wasser Verteilungskoeffizienten als Maß für die Akkumulierbarkeit eines Stoffes). Die Grenze von 3 beruht auf internationalen Konventionen

akkumulierend = $\log P_{ow} > 3$ bzw. BCF > 100

LC_{50} = die Konzentration, bei der die Hälfte der untersuchten Organismen den betrachteten Endpunkt - in diesem Fall Mortalität - aufweist; ein übliches Maß, „Ökotoxizität“ auszudrücken. Die angegebenen Grenzen sind gemäß Konventionen basierend auf der EU-Richtlinie 67/548/EWG zur Einstufung und Klassifizierung sowie dem Chemikaliengesetz gewählt worden.

Für SimpleTreat-Rechnungen verwendete Daten einer typischen Kläranlage (deutsche Großstadt)

Parameter	Einheit	Wert
Anzahl der Einwohner pro Kläranlage		350000
Abwasser-Input der Kläranlage	m^3/d	51300
Höhe der Luftsäule	m	3.9
Volumen des 1. Absetzbeckens (Primary Settler)	m^3	8000
Tiefe des 1. Absetzbeckens	m	2
Volumen des Belüftungsbeckens (Aeration Tank)	m^3	31000
Tiefe des Belüftungsbeckens	m	4
Volumen des 2. Absetzbeckens (Solid-Liquid-Separator)	m^3	29200
Tiefe des 2. Absetzbeckens	m	3.1
Wassertemperatur	°C	14.7
pH-Wert		8
Art der Belüftung: Oberflächen- oder Blasen-Belüftung		Blasen
Belüftungsrate	m^3/s	5.6
Sauerstoff-Konzentration	kg_{O2}/m^3	1.65
Konzentration von suspendiertem Feststoff im Belüftungsbecken	kg_{DW}/m^3	4.5
Konzentration von Belebt-Schlamm	kg_{DW}/m^3	5
Input an Feststoff im Rohabwasser	kg_{DW}/d	35000
BOD	g_{BOD}/d	17100000
Schlamm-Belastungsrate	$kg_{BOD}/(kg_{DW}*d)$	0.0001

Für SimpleBox-Rechnung verwendete Umweltdaten (deutsche Großstadt)

Parameter	Einheit	Wert
Gesamte Modell-Grundfläche	km^2	891
Anteil Wasser an Grundfläche	%	6
Anteil landwirt. Boden an Grundfläche	%	7
Anteil natürl. Boden an Grundfläche	%	30
Anteil industr. Boden an Grundfläche	%	57
Durchschnittl. Niederschlag	m/s	1.839E-08
Anteil Regenwasser, der in den Boden eindringt	%	25
Mischungshöhe Luft	m	1000
Durchschnittl. Windgeschwindigkeit in 10 m Höhe	m/s	4.04
Durchschnittl. Luft-Temperatur	K	283.15
Mischungstiefe landwirt. Boden	m	0.2
Mischungstiefe natürl. Boden	m	0.05
Mischungstiefe industr. Boden	m	0.05
Gehalt an Wasser in Boden	%	20
Gehalt an Luft in Boden	%	20
Gehalt an org. Kohlenstoff in Boden	%	2
Dichte des Bodens	kg/m^3	2500
Wasserströme in das System hinein	m^3/s	60
Mischungstiefe des Wassers	m	1
Konzentration von suspendierter Materie in Wasser	kg/m^3	0.015
Konzentration von Fischen in Wasser	kg/m^3	0.001
Gehalt an org. Kohlenstoff in suspendierter Materie	%	10
Gehalt an org. Kohlenstoff in Sediment	%	5
Gehalt an Wasser in Fischen	%	95
Gehalt an Wasser in Sediment	%	80
Gehalt an Wasser in suspendierter Materie	%	90
Mischungstiefe des Sediments	m	0.03

Ansatz: iterative Nutzung von LCA und Risikoabschätzung

Pro:

- hohe Flexibilität und Zielorientierung in der Anwendung einschließlich Möglichkeiten des Abbruchs

Contra:

- notwendige Systemsetzungen und Annahmen können unrealistisch sein
- Übergänge zwischen Methoden unvermeidbar (Beispiel: Erhalt des Bezugs auf funktionelle Einheit)

Identifizierung von Trivialitäten bei unflexibler Anwendung

Ansatz: Integration der Methoden

- Ableitung von Äquivalenzfaktoren unter Nutzung von Elementen der Risikoabschätzung

- „Risikoabschätzung“ realisiert als „Gefährdungsanalyse“ bis hin zur „Risikoanalyse gemäß TGDs“
- aktuelle Erweiterungen: Nutzung von probabilistischen Input-Größen
- aktuelle Erweiterungen: raum-zeitliche Beschreibung des Emissionstyps

Ansatz: Integration der Methoden

„Risikoabschätzung“ realisiert als „Gefährdungsanalyse“ (IUCT-Detail Methode)

Impact Score (Wasser) = $I_{Exposition}$ (Wasser) * [Akkumulationsfaktor + $I_{Effekte}$ (Wasser)]
Impact Score (Boden) = $I_{Exposition}$(Boden) * [Akkumulationsfaktor + $I_{Effekte}$ (Boden)]

$I_{Exposition}$ (Wasser bzw. Boden) = 1,37 (log E + 1,301)

E = Emission [kg] x Verteilungsfaktor x Abbaufaktor
$I_{Exposition}$ (Wasser bzw. Boden) ist so normalisiert, daß der Wert zwischen 0 und 10 liegt

$$I_{Effekte}\ \textbf{(Wasser bzw. Boden)} = \frac{\log (PNEC_i / PNEC_{max})}{\log (PNEC_{min} / PNEC_{max})} * 7$$

Referenz: 1,4-Dichlorbenzol

Ansatz: Integration der Methoden

„Risikoabschätzung“ realisiert als „Risikoanalyse in Anlehnung an TGDs“ (CML/RIVM-Methode)

- PEC: Exposition, ermittelt über Verteilungsmodell (EUSES)
- PNEC: Wirkung, auf Basis von No-Effect Levels (NOEL)

$$AETP_{subs,comp} = \frac{\left[\frac{PEC_{water,subs,comp}}{PNEC_{aquatic,ecosystems,subs}}\right]}{\left[\frac{PEC_{water,1,4-dichlorbenzene,water}}{PNEC_{aquatic,ecosystems,1,4dichlorbenzene}}\right]}$$

AETP = Aquatic Ecotoxicity Potential

comp. = das Kompartiment, in das Substanz zu Beginn imittiert wurde Wasser, Luft, industr. Boden, landw. Boden.

subs. = betrachtete Substanz

Ansatz: Integration der Methoden

„Risikoabschätzung“ realisiert als „Risikoanalyse in Anlehnung an TGDs“ (CML/RIVM-Methode)

· Multiplikation der emittierten Mengen mit $\boldsymbol{AETP}_{\boldsymbol{subs}, \text{comp}}$ und Aufsummation

$$\text{Impact Score}_{\text{aquatic ecotoxicity}}\,[\text{kg}] = \Sigma\, \boldsymbol{AETP}_{\text{subs, air}} * m_{\text{subs, air}}\,[\text{kg}]$$

$$\Sigma\, \boldsymbol{AETP}_{\text{subs, water}} * m_{\text{subs, water}}\,[\text{kg}]$$

$$\Sigma\, \boldsymbol{AETP}_{\text{subs, industr. soil}} * m_{\text{subs, industr. soil}}\,[\text{kg}]$$

$$\Sigma\, \boldsymbol{AETP}_{\text{subs, agric. soil}} * m_{\text{subs, agricul. soil}}\,[\text{kg}]$$

Ansatz: Integration der Methoden

Pro:

- elegante Möglichkeit der Kombination von verschiedenen Instrumentarien
- Wissen aus der Stoffbewertung wird soweit wie möglich genutzt

Contra:

- durch großen Bedarf an Informationsdichte für die Input-Daten muß häufig auf Default-Annahmen zurückgegriffen werden, wodurch die Aussageschärfe unkontrollierbar ungenau wird, jedoch in der Ergebnis-Präsentation eine „Scheingenauigkeit“ wiedergegeben wird

Schlußfolgerungen

- es gibt kein eindeutiges Pladoyer für einen der genannten Ansätze
- je nach Anwender- und Entwicklerkreis werden die Ansätze weiterentwickelt und sich dabei voneinander wegentwickeln

GUNTRAM KOLLER

Data Ranges in Aquatic Toxicity of Chemicals – Consequences for Environmental Risk Analysis

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Submitted to Environmental Toxicology and Chemistry, Nov 1998

Keywords

aquatic toxicity, data range, uncertainty, effect assessment, ecological risk assessment

Abstract

A major problem for effect assessment of aquatic ecosystems arises from the large ranges of toxicity data, which can be found in different databases and literature. Here, toxicity ranges are given for 27 high production volume chemicals. Based on these examples and on the current literature on uncertainty in aquatic effect assessment, the implications on the procedure of risk analysis of chemical substances are discussed. Two main requirements for a comprehensive risk assessment are identified, which often play a minor role in current practice (as they are often neglected) as well as in scientific discussion (as they are meant to be trivial). First, data quality must be checked critically before applying any result of a toxicity test. Secondly, experimental data should take into account different species and acute as well as chronic data. If these aspects are considered in risk analysis which is common practice in ecotoxicology but not always in the context of practical applications in risk engineering, a comprehensive picture of the aquatic toxicity of a chemical substance can be obtained.

1 Introduction

The risk posed on aquatic ecosystems by man-made substances is an important part of all environmental risk analysis methods proposed by legal or scientific bodies. In the effect analysis as part of the environmental assessment, it is tried to estimate a concentration which has no undesired effects on the aquatic ecosystem. No Effect Levels (NEL), No Observed Effect Concentrations (NOEC), Predicted No Effect Concentrations (PNEC) or aquatic quality criteria are examples of such concentrations. The aquatic effect assessment in environmental risk analysis is usually based on a set of toxicity data obtained from environmental databases, published toxicological studies or a set of values directly measured in toxicological experiments.

Almost all toxicity data are based on laboratory tests. As these tests have been highly standardized (1), the experimental conditions for each toxicological endpoint and species are clearly defined based on Good Laboratory Praxis (GLP). The effects observed at different concentrations are interpreted using a statistical model in order to obtain the toxic concentration for the endpoint. These statistical models and

their implications on the results of the study are reviewed by Chapman et al. (2). The experimental results obtained are published in a toxicological report or included in some kind of database. Because of this standardization, results of toxicity tests are usually well documented in toxicological studies. However, documentation decreases largely as soon as secondary information sources are considered. Many environmental databases do not include background information about the test conditions such as exact description of the endpoint, pH of system, etc. The quality of documentation of toxicity data in material safety data sheets (MSDS) is even worse, although MSDS are an important data source for environmental risk assessment in industrial praxis. Such background information about the exact test conditions, however, is essential for interpreting the results, as all toxicity data have to be questioned critically before being applied in risk analysis.

A major problem often encountered during risk assessment is the lack of ecotoxicological data covering key species in ecosystems. This holds in particular for chronic toxicity data. This problem of uncertainty because of missing information is addressed by estimating toxicity data via QSAR methods (3) or by estimating safe concentrations using safety factors (uncertainty factors). Most international bodies issued guidelines which factors to apply in order to account for the different sources of uncertainty (4). The scientific community is discussing these factors (5,6) intensively.

In case of chemicals where a sufficient set of toxicity data exists, one major problem of aquatic effect assessment is the large range of data for the same chemical substance. Toxic concentrations can vary by several orders of magnitude depending on experimental conditions, species, endpoint, exposure time (acute – chronic) and aquatic test environment (laboratory – field). Several studies are available reporting ranges of aquatic toxicity data. Especially the difference between acute and chronic data has been studied intensively for a broad field of substances (7). Other studies report the range of aquatic toxicity for a specific group of chemicals especially insecticides and herbicides (8,9). For commodity chemicals produced at high volumes, however, illustrative examples for the ranges of aquatic toxicity data are missing. This is somehow astonishing as a sufficient amount of data exists and as these substances (for instance solvents) play an important role in every-day risk assessment.

If a sufficient amount of reliable and well-documented data is available to the environmental risk manager, the toxicity ranges can be considered and the “safe” concentration for protecting ecosystems can be estimated. Environmental risk analysis is performed not only by “experts” in ecotoxicology knowing the theoretical background of aquatic toxicology. Detailed guidelines for aquatic risk assessment are available at many international bodies in order to simplify and harmonize the methods. These guidelines should provide every user with an easy-to-use “manual” how to perform environmental risk assessment. However, there is not yet consensus reached on a scientifically and politically accepted framework for aquatic effect assessment. Especially the concept of using NOEL values for estimating “safe” concentrations has been criticized (10,11,12,13) and effect concentration at low effect levels (EC5, …) were proposed as alternatives. Despite all guidelines, collection and

interpretation of toxicity data still requires time and background knowledge in order to avoid misinterpretation. As the time available for performing a rigorous study on environmental risk is decreasing continuously at today's economic situation, practitioners are faced with the problem not to have enough time for a comprehensive literature search for the aquatic toxicity of a substance.

Despite all problems of missing data, poor data quality, large toxicity ranges and methodological discussions, practitioners sometimes believe effect assessment to be possible by simply selecting a few single values for the aquatic toxicity of a substance according to published guidelines without any toxicological background knowledge. This would largely simplify and speed-up the risk analysis process, but can lead to misinterpretations and wrong results. Similar practical problems and misunderstandings are described in the literature (14).

The goal of this study is to highlight the problems associated with the application of aquatic toxicity data in risk analysis by giving illustrative examples of 27 selected bulk chemicals. We want to show that in effect assessment, the aquatic toxicity of a compound should be based on a concentration range instead of one or a few single values. After analyzing the different reasons for the toxicity ranges we discuss the current concept of safety factors with respect to the aquatic toxicity of the selected substances. Some recommendations are given, pleading for a critical use of a full set of data when assessing the toxicity of a chemical substance to aquatic ecosystems.

2 Methods

In order to obtain a comprehensive picture of the problems in applying aquatic toxicity data in environmental risk analysis, 27 substances of different chemical classes were selected. Their aquatic toxicity data were presented graphically for different species and endpoints.

2.1 Selection of substances

The basis for selection was a list of High Production Volume Chemicals in the U.S. (production volume > 50,000 t) which contains many important bulk chemicals. The substances were selected, when a sufficient number of toxicity data was available in public databases (at least ten acute values and two chronic values). As an additional criterion, the selected substances should play an important role in fine chemical industry (such as solvents) and they should cover different chemical substance classes. Inorganic acids and bases were not considered, as their toxic effect is usually based on the pH change. The selected 27 substances are listed for each class in Table 1 (note that substances can be mentioned more than once). Most substances exert their toxic effect through narcosis and membrane toxicity and act by an unspecific mode of action. Only some of them have other and specific mechanisms of toxicity such as the cyanides.

2.2 Import and quality of data

Data for the aquatic toxicity of the chemicals were taken from two different databases. ECDIN (Existing Chemicals Data Information Network – http://ecdin.etomep.net/) is a publicly available database of the European Community and includes all substances of the EINECS (European Inventory of Existing Chemical Substances) with varying amount of data. The toxicological information was selected from primary literature by experts. ECDIN is no longer kept up to date for a few years now, as a new database system, IUCLID (International Uniform Chemical Information Database), is being developed. The second database used in this study is the IGS-database (Informationssystem Gefährliche Stoffe) and was built by Swiss Authorities (Nationale Alarm Zentrale, http://www.aac.ch/IGS/root.htm). It contains toxicity data from different other sources (databases) which were selected without further quality control.

For each substance, all toxicity data were exported from the external databases and saved as a text file. After creating a new database (Microsoft Access), all data files were imported. In a first set of calculations, the data were transformed into a standardized format (SI-units, endpoint categories according to chapter 0). Secondly, the following quality criteria were applied on the data:

1. Data were rejected, if no information about species or endpoint was available or if no result was given (10% of data).
2. In a few cases, concentration ranges were given instead of single values. If the range exceeded the factor of 5, data were not used (e.g. effect concentration (growth, 40% increase) of toluene to alga: 0.1-10 mg/l). In the case of smaller ranges, the lower value was used (precautionary principle).

It was not possible to apply additional quality criteria, as the documentation of some data was incomplete (see chapter 0).

2.3 Definition of endpoint categories

Five different endpoint categories were used in this study in order to simplify the graphical representation (LC50, Effect, Chronic, LOEL, NOEL). These categories are based on toxicological endpoints but some of them are defined slightly different. “LC50” contains all acute LC50 values. The category “LOEL” includes all endpoints where a lowest concentration causing toxic effects was described. Therefore, not only Lowest Observed Effect Concentrations according to the toxicological definition were included, but also values extrapolated from a dose response relationship (e.g. EC5). The following endpoint descriptions were collected in the category “LOEL”: EC5, EC10, LC5, LC10 (EC: effect concentration, LC: lethal concentration, number refers to percentage of total effect 100%), LOEL, threshold level. Similarly, the category “NOEL” is used to show all endpoints which in the data source were mentioned as EC0, LC0, no effects, NEL (No Effect Level), NOEL or NOAEL (No Observed Adverse Effect Level). This exceeds the toxicological definition of a NOEL.

All endpoints not included so far were summarized in the categories “Effect” and “Chronic”. If a chronic endpoint could be identified, the category “Chronic” was applied. All remaining acute data or data without sufficient information about the time of the experiment were collected in the “Effect” category. If a LC50 value (e.g. 28 days in fish) was reported, it is shown as “Chronic” and not as “LC50” value in all graphs. All acute lethal concentrations besides the LC50 values (such as LC100, LC25, total mortality) are presented in the category “Effect”.

This classification results in one narrowly defined endpoint category (“LC50”) and four broad categories summarizing similar endpoints. All data were graphically presented using these categories.

3 Results

3.1 Experimental parameters influencing toxicity

The exact experimental conditions are of highest importance for obtaining comparable results in toxicological studies. For acrylonitrile, the time course of toxicity is shown in Figure 1. The LC50 / EC50 values for ***Leuciscus idus*** decrease for 2-3 orders of magnitude when comparing values for 1 and 96 hours. This is a well-known fact of the toxicological response of organisms and only the 96h value will be used in effect assessment. However, if the time information is not included in the data-source, these two values can not be distinguished and the variability of toxicity results can not be explained.

An experiment for measuring the aquatic toxicity of a given substance can be designed as static or flow-through test depending on the mode of adding and controlling the tested substance. As soon as volatile, degradable or adsorbable substances are tested, this can lead to large ranges in results. Figure 2 illustrates this problem using the highly volatile acetone as example. Toxicity data for ***Daphnia magna*** are lower by a factor of 1000, if flow-through tests are compared to static experiments. As acetone evaporates, the effective concentration can largely decrease during static experiments. In flow-through tests, acetone is added throughout the experiment to keep a constant concentration. Therefore a much higher amount of acetone added at the beginning was required in the static test to reach equal toxic effects as in flow-through experiments.

Similar differences in toxicity data can be caused by differences in the pH-value of the experiment, if protonable or deprotonable substances are tested.

3.2 Comparison within related species

Aquatic toxicity strongly depends on the animal or plant species under consideration. As an example, the toxicity of toluene for different fish species is shown in Figure 3. Between the different species, the LC50 values vary by the factor of 200 (interspecies variability). Within one species (intraspecies variability) the range is smaller and does not exceed a factor of 10. Effect-concentrations exhibit higher ranges (factor of 5,000). This fact can mainly be contributed to differences in the measured effect (***Cyprinus***: blood serum concentration; ***Leuciscus***: letal effects; other fish species: behavior,

reproduction). Toluene as an example corresponds quite well with the ranges of intraspecies variability which generally is reported not to exceed a factor of 10 for most substances (5).

3.3 Comparison of different species

To illustrate the species differences, the aquatic toxicity of diethanolamine is shown in Figure 4 as a representative example. Similar graphs were built for all 27 substances but are not shown here. Diethanolamine is known to be toxic to liver and kidney of higher vertebrates. At the cellular level it leads to changes in the phospholipids of the cell membranes (15). Some carcinogenic effects are reported as well, as nitrosamines can be formed during metabolism (15). LC50 values range between 20 and 5,000 mg/l. The ranges within fishes and crustacean span a factor of about 10. For one alga, ***Skeletonema costatum***, the toxic concentration lies two orders of magnitude below that of other algae species (***Scenedesmus, Selenastrum***) (see chronic and NOEL values of Figure 4). High interspecies variabilities and high sensitivities have been reported for algae also for other compounds (16). Crustaceans and algae act most sensitive to diethanolamine, whereas fishes are a factor of 100 less sensitive. If only fish data were used for an effect assessment, the risk would largely be underestimated even if a safety factor of 10 were used.

3.4 Comparison of different substances

Figure 5 gives an overview of the acute aquatic toxicity of all 27 substances (without NOEL and LOEL values). All substances are roughly ordered by decreasing polarity starting with salts at the left-hand side and ending with hexane on the right. On first sight, the large ranges of aquatic toxicity can be seen which cover two to four orders of magnitude for most substances. Higher variabilities (factor of 100,000) can be observed for NaOCl, formaldehyde, acetone, dimethylformamide and methanol. Some single values at the higher end of the concentration range can be explained with inadequate experimental design (static tests: ammonia, NaOCl, $NaNO_2$, acetone; short test periods: acrylonitrile). Applying more restrictive quality criteria would reduce the ranges for the mentioned substances by a factor of 10 to 100. Such strict criteria would, however, remove almost all data for some other compounds.

The largest number of toxicity data was measured for fish and crustacean. Toxicity data for algae and molluscs were available for two thirds and half of the compounds, respectively. A comparison of the toxicity of the different substances to other aquatic organisms was not possible, because data were lacking for most substances. No species can be identified which is most sensitive to all substances studied, which is well known in ecotoxicology (8). General trends of the toxicity results themselves or of the size of the overall variability could not be seen. This fact is not surprising as different modes of toxic action are involved.

3.5 Comparison of different endpoints

One major reason for ranges in toxic concentrations is the difference in the endpoint measured in the experiments. Sublethal effects usually occur at concentrations, which do not cause mortality of the organism. Therefore, lethal concentrations normally have higher values than effect concentrations under comparable experimental conditions. First physiological or chemical changes in the organism can already occur at much lower concentrations, where no macroscopic effect can be observed. Therefore, the exact description of effect concentrations is essential for interpreting the results of the toxicological study.

Figure 6 shows different endpoints of the toxicity to ***Daphnia magna***. The data picture is not completely consistent with theory, partly because quality and quantity of the data was not high enough. NOEL values (except LC0 values) lie at the lower end of the toxic range for most substances. For p-chlorphenol, however, effect concentrations (phototaxis, enzyme inhibition) are reported below the NOEL values (factor of 10). LOEL values can not be found between effect concentration and No Effect Concentrations, but are spread over the whole range of toxicity data. This fact can be explained with the lack of clear documentation and missing of exact definition of most LOEL values. LC50 values usually are above sublethal effect concentrations by factors between 1 and 10.

4 Discussion

4.1 Quality of data

One of the main practical problems of interpreting toxicological data is that the documentation and the quality of the information often is poor, especially in broadly used data sources (such as official databases, MSDS). In this study, no strict quality criteria could be applied, such as minimum testing time, exact description of endpoint and experimental conditions (controlled pH, no static tests) and meaningful citation of data source. Especially the IGS-Data source was quite unsatisfactory in this respect, although it is the official database recommended by Swiss Authorities. The exact documentation of the experimental conditions (pH, temperature, static or flow-through, etc.) was only given for half of the results. A rough description of the endpoint (such as EC50) and species (such as fish) was available for almost all data (95%). However, an exact allocation to chronic or acute tests could only be done in 70% of the results, as for the others no testing time was given. On an additional 15%, data could be ascribed as acute values, as LC50 values commonly are acute endpoints.

In principle, the most common toxicological endpoints (such as NOEL, LC50, LOEL, EC50) are defined. However, a large variety of descriptions and slightly different definitions exist for most endpoints in toxicological information systems. This poses problems for users willing to interpret toxicological data accordingly. In particular, databases show an astonishing and often unclear variety of endpoint descriptions (e.g. TLM, TDLo, LDLo, threshold value, normal effects, increasing mortality, etc.). In most cases, it was possible to attribute standardized endpoints to the verbal descriptions, in some other cases, however, the endpoint description (such as “acute” or “chronic”) were of limited

value. The danger of poor documentation of toxicity data is the tempting possibility of rejecting undesirable data because of low quality, but of accepting suitable data without critical evaluation. Critical questioning of the toxicological data used for risk assessment is a crucial point for avoiding misinterpretation during the whole assessment process.

Figure 1 and 2 show how important the experimental conditions (e.g. time, water flow) are for interpreting the results. It should be emphasized that the experimental conditions must be documented for meaningful interpretation. This criterion is fulfilled for most data measured since toxicological experiments got standardized in the 80's in particular those performed under GLP conditions. As an important quality criterion of toxicological information media such as substance datasheets or databases, the full documentation of all important experimental parameters must be included. If this information is missing, selecting a single value or using statistical methods for interpreting the results can lead to large errors. The aquatic risk can be over- or underestimated by several orders of magnitude. Additionally, the standardization of ecotoxicological endpoints should be further developed and communicated to the public. Every ecotoxicological endpoint which might not be known by all possible users of the results should be defined clearly when passing on toxicological information. This could avoid misinterpretations and misunderstandings of aquatic toxicity data.

4.2 Data ranges and concept of safety factors

Aquatic toxicity data for a substance always cover a certain concentration range. Several reasons are known for this fact such as differences within a species and between species, endpoints, replicates, exposure time, laboratories and between laboratory and field tests (8). Using only a single or a few values can never deliver a reliable picture of all ecotoxicological effects of a substance. Thus, toxicity can be over- and underestimated by several orders of magnitude depending on substance and data quality. Only if a sufficient amount of reliable data is available covering all mentioned reasons for variability, a "safe" concentration for the aquatic ecosystem can be estimated. This condition, however, is fulfilled only for a very small number of substances due to different (especially economic) reasons. Usually, only a much smaller number of data which is at the fingertip of the user will be applied. As only some of the ranges can be covered, the remaining uncertainty of missing information has to be dealt with before estimating the "safe" concentration. A similar problem arises when substances with different levels of information about toxicity are compared. Detailed aquatic toxicity data for one substance can not be used for comparison, if corresponding values are missing for the other substance. To resolve these problems, the concept of safety factors (uncertainty factors) has been proposed.

If no chronic or sublethal effect data, or no NOEL values or field studies are available, the use of safety factors has been recommended for extrapolating "safe" concentrations from LC50 values (4). These

factors are based both on policy and science and try to estimate concentrations that are very probably lying below the real values. The goal of safety factors is to keep the probability of underestimating the risk low, independently on the amount of toxicity data. This pragmatic concept allows effect assessment based on single LC50 values. Usually factors of 10 for extrapolation of lethal to sublethal, acute to chronic, inter- and intraspecies variability and LOEL to NOEL are proposed. A detailed discussion of these safety factors, their background and problems was done by Chapman et al. (5). Some aspects are summarized below and discussed with respect to the results of the present study.

4.2.1 Acute-chronic ratio

Chronic toxicity tests cover a considerable part of the life span of organisms. They are quite time consuming and costly to perform and therefore attempts have been made to develop extrapolation methods to estimate chronic from acute data. The acute-chronic ratio plays an important role in legislation (e.g. water quality criteria in the U.S.). There, it is assumed that the ratio between chronic and acute data of a given substance is equal for all species (17). Using this mean ratio, the chronic quality criterion (Final Chronic Value) can be estimated from the acute criterion (Final Acute Value). The OECD guidelines propose an average factor of 10, if chronic data are missing. This factor was obtained from the 50% percentile of a study of the ratios between 96h LC50 and chronic NOEL values for 72 substances (7). The ratios ranged from values of 0.13 to 1300, which is an indication of the problems associated with this extrapolation.

The use of a constant acute-chronic ratio for all substances has partly been supported (6), but is being increasingly criticized from an ecotoxicological point of view. The extrapolation from acute to chronic toxicity is based on statistical analysis rather than toxicological concepts. In the past, a factor of 10 seemed to be sufficiently protective for most substances and species, as chronic data were quite rare. During the last decade, a number of examples have been reported (5,8), where the ratio between acute and chronic data can not be represented with a constant factor of 10. First, the ratio strongly depends on the species and substance, and second, it can reach much higher values (>1000). This fact is not surprising, as different toxicological mechanisms can be responsible for chronic and acute toxicity. For the 27 substances of this study, conclusions for the acute-chronic ratio could not be drawn, as not enough chronic data of sufficient quality were available.

As this extrapolation is scientifically questionable, it is essential to use chronic data from experiments or substance-specific estimation methods for aquatic effect analysis. The general safety factors for acute-to-chronic extrapolation can neither predict chronic toxicity, nor assure the protection of aquatic ecosystem when trying to extrapolate “safe” concentrations.

4.2.2 Inter- and Intraspecies variability

Considering the enormous evolutionary diversity of aquatic species, it can be easily understood that different sensitivities exist for the same substance. Evolutional, biological, physiological-morphological

and ecological differences between organisms are among the reasons for this diversity. Some earlier studies (18) reported ranges of a factor of 2-50 for LC50 values, whereas in recent studies (5,8) much larger ranges (>10,000) were reported. Similar high ranges of several orders of magnitude can be seen in Figure 5. A statistical evaluation yielding mean and maximal variability strongly depends on the quality criteria applied on the raw data and would therefore not give any additional information. Even within closely related species, a high variability of a factor of 10,000 was shown for some specific substances such as organophosphate pesticides (e.g. disulfoton) (8). These large ranges are desired as the substances are designed to exhibit high selectivity on a specific group of organisms. For most substances, however, aquatic toxicity to similar species does not exceed a range of 10 to 100, especially since detailed guidelines for conducting toxicological experiments are being followed.

From the practical point of view, it would be desirable in risk assessment to identify a most-sensitive species, from which extrapolation to all other species would be possible. This would largely simplify risk assessment of new substances, as only one species would have to be tested and the resulting concentration level would protect all other species. However, such most sensitive species does not exist for several reasons. This can be seen in Figure 5. If crustaceans were assumed to be the most sensitive species, the lowest toxic concentrations would be found for 45% of the substances considered in this study. For 20% of the substances, other species are more sensitive by a factor >100. Applying a safety factor of 10 would not be sufficient for these substances. Thus, for assessing aquatic effects it is essential to have data for a several species of different trophic levels (8,14).

4.2.3 Extrapolation to different endpoints

Figure 6 compares different endpoints. General correlations allowing extrapolation from one endpoint to another (such as lethal to sublethal effects, LOEL to NOEL) could not be observed. Such constant extrapolation factors can be defined with statistical means for ideal data, i.e. data measured in the same laboratory with the same organisms under exactly the same experimental conditions. Applying them on real data from different sources with partly unknown quality can result in large errors and unrealistic values. If such safety factors are used for aquatic effect assessment, the risk can be overestimated for several orders of magnitude. Especially the aggregation of a number of factors often leads to unrealistically low values (19). If NOEL values were extrapolated for the studied substances applying extrapolation factors on LC50 / EC50 values, the results would be lower by a factor between 1 and 1000 than the real NOEL values. Thus, the current system of endpoint extrapolation estimates values, which are protective but often unrealistically low. One exception might be the safety factor of 10 proposed by the European Union to extrapolate from a LOEL to a NOEL for human effect assessment (4). It can only be applied if the quality of the LOEL is without any doubt. Otherwise this extrapolation might underestimate the risk.

General extrapolation factors must not be used to predict toxicity data for other endpoints. For comparison of the aquatic toxicity of two substances (one with a full data set, one with little data), there

is no advantage in using any of those factors. From a legal point of view, it is possible to close data gaps using such factors, as they estimate more or less “safe” concentrations in order to protect the environment. From a scientific point of view, the use of general extrapolation factors for predicting aquatic toxicity is questionable.

4.3 NOEL / LOEL concept in risk analysis

Most existing concepts of risk analysis rely on the No Effect Level (NEL), which is the real concentration not causing any undesired effects in the aquatic environment. This is a hypothetical value, which can not be measured experimentally. Therefore, a NOEL is commonly used to estimate the NEL. In the last decade this concept of NOEL has been criticized (2,10,11,12,13) for the following reasons.

A NOEL is obtained as the highest experimentally measured concentration, where no significantly different effects were observed between the test group and the control group of the experiment. The significant difference is analyzed using one of the statistic hypothesis test procedures usually with a significance interval of 5%. Laskowski showed that this significance level often does not correspond to the desired error probability of underestimating the aquatic risk (13). The error probability of obtaining a (wrong) concentration as result (i.e. as the NOEL), at which toxic effects still occur but simply have not been detected because of pure chance, usually is between 10 and 20% or even higher (13).

Chapman et al. (10) showed different examples how the choice of data interpretation method (hypothesis test) can influence the result of the study (i.e. the NOEL) using the same experimental data. Similarly, a different choice of concentrations used in toxicity experiments can lead to large differences in the resulting NOEL. The main reason for this problem of the NOEL concept is that only one single value of the whole experiment is used for obtaining the result instead of the whole dose-response curve. A small change in experimental data which, for instance, increases the error probability from 4.9 to 5.1% finally leads to a large change in the NOEL, because the next measured (lower) concentration has to be used. This can be the reason why the ranges for NOEL values are reported to be higher than for EC50 values (10).

Several alternatives were proposed instead of the NOEL concept using different kinds of effect concentrations (from EC50 down to EC0) (2,10,11). Problems of hypothesis test selection, dependence on experimental conditions can be avoided by fitting a statistical distribution to all experimental data using regression analysis. From this distribution model, the desired effect level can be calculated. The kind of statistical distribution and regression analysis has no significant influence as long as it is used for interpolation between measured values. However, if a concentration at low effect levels such as EC0 or EC5 shall be extrapolated, the result largely depends on the choice of the model.

The dependence on statistical models can lead to large uncertainties for both EC0 and NOEL values. The endpoint, which has the lowest uncertainty ranges caused by statistical or experimental reasons, is

the EC50 (LC50) value. Therefore, such endpoints should be used for comparing the aquatic toxicity of different substances. The principal problem of estimating a NEL, a concentration at which no effects occur, can be improved but not completely solved by the alternative concepts to the NOEL.

We understand the criticism of the NOEL concept as one which is largely based on mathematical/statistical reasoning. Compared to the data ranges caused by the different sources of variability, these theoretical considerations have to be relativated, especially if a pragmatic approach to aquatic effect assessment is sought.

5 Conclusion

Assessing aquatic effects of chemical substances is a major task in environmental risk assessment. Although a number of guidelines exist, several problems can occur during this procedure especially for non-experts in ecotoxicology. The first important step of successful effect assessment is to question all toxicological data critically before applying them. All background information required for this quality check must be made available in primary and also in secondary information media for toxicological data.

Ecotoxicological data always consist of a range of concentrations depending on species, endpoint, time-scale and experimental conditions. To get a comprehensive impression of the aquatic toxicity of a substance, the whole range must be considered and covered with data. This especially includes data for different species of different trophic levels and acute as well as chronic data. From the legal point of view, safety factors provide a useful and pragmatic means to deal with these uncertainties as they usually (with some exceptions) lead to “safe” concentrations which protect the environment. For predicting toxicity data for instance in order to compare the true aquatic toxicity of two substances, general safety factors should not be used. If the quality and the ranges of toxicity data are not considered adequately, the risk in the aquatic ecosystem can be under- or overestimated by several orders of magnitude.

6 Acknowledgments

The authors thank Martin Scheringer and Oemer Kut (ETH Zürich), Hannes Wasmer and Beate Escher (EAWAG) for reading the manuscript.

7 References

(1) Organization of Economic Cooperation and Development (OECD). 1993. Guidelines for Testing Chemicals. Paris, France.
(2) Chapman PF, Crane M, Wiles J, Noppert F, McIndoe E. 1996. Improving the quality of statistics in regulatory ecotoxicity tests. ***Ecotoxicology*** 5:169-186.
(3) Hansch C, Leo A. 1995. ***Exploring QSAR***. American Chemical Society, Washington, DC.
(4) Commission of the European Communities. 1996. Technical guidance documents in support of the Commission Directive 93 / 67 / EEC on risk assessment for new substances and the Commission Regulation (EC) No 1488 / 94 on risk assessment for existing substances. Brussels, Belgium.
(5) Chapman PM, Fairbrother A, Brown D. 1998. A critical evaluation of safety (uncertainty) factors for ecological risk assessment. ***Environ Toxicol Chem*** 17:99-108.

(6) Fawell JK, Hedgecott S. 1996. Derivation of acceptable concentrations for the protection of aquatic organisms. ***Environ Toxicol Pharmacol*** 2:115-120.
(7) European Centre of Ecotoxicology and Toxicology of Chemicals (ECETOC). 1993. Aquatic Toxicity Data Evaluation. Technical Report 56. Brussels, Belgium.
(8) Fent K. 1998. ***Ökotoxikologie***. Georg Thieme Verlag, Stuttgart, Germany.
(9) Abt. Associates. 1995. Technical basis for recommended ranges of uncertainty factors used in deriving wildlife criteria for the Great Lakes water quality initiative. Final Report. Office of Water, U.S. Environmental Protection Agency, Washington, DC.
(10) Chapman PM, Caldwell RS, Chapman PF. 1996. A Warning: NOECs are inappropriate for regulatory use. ***Environ Toxicol Chem*** 15:77-79.
(11) Hoekstra JA, Van Ewijk PH. 1993. The bounded effect concentration as an alternative to the NOEC. ***Sci Total Environ*** Supplement:705-711.
(12) Hoekstra JA, Van Ewijk PH. 1993. Alternatives for the No-Observed-Effect-Level. ***Environ Toxicol Chem*** 12:187-194.
(13) Laskowski R. 1995. Some good reasons to ban the use of NOEC, LOEC and related concepts in ecotoxicology. ***Oikos*** 73:140-144.
(14) Power M, McCarty LS. 1997. Fallacies in ecological risk assessment practices. ***Environ Sci Technol*** 31:370A-375A.
(15) Criteria group for occupational standards. 1992. Scientific basis for Swedish Occupational Standards XIII, Consensus report for diethanolamin. ***Arbeite och Halsa*** 47:1-4.
(16) Hoffman DJ, Rattner BA, Burton GA, Cairns J. 1995. ***Handbook of Ecotoxicology***. Lewis Publishers, London, United Kingdom.
(17) U.S. Environmental Protection Agency. 1997. Water quality guidance for the great lake system. 40 CFR 132. Washington, DC.
(18) U.S. Environmental Protection Agency. 1984. Estimating concern levels for concentrations of chemical substances in the environment. Environmental Effects Branch, Health Environ. Rev. Div. Washington, DC.
(19) Swartout JC, Price PS, Dourson ML, Carlson-Lynch HL, Keenan RE. 1998. A probabilistic framework for the reference dose. ***Risk Analysis*** 18:271-282.

Table 1: The selected 27 substances

Substance class	Substance
Aliphatic hydrocarbon	Hexane
Halogenated compound	Methylene chloride, p-chlorophenol, dimethylethylhexadecylammoniumbromide
Ether	Diethylether, tetrahydrofuran
Alcohol, phenol	Methanol, ethanol, isopropanol, phenol, p-chlorophenol
Aldehyde, ketone	Formaldehyde, dimethylformamide, acetone
Acid and derivatives	Oleic acid, hydrogen cyanide, ethylacetate, acrylonitrile
Amine	Ammonia, diethanolamine, pyridin, dimethylethylhexadecylammoniumbromide
Long chain compound	Oleic acid, dimethylethylhexadecylammoniumbromide
Aromatic compound	Phenol, toluene, p-chlorophenol
Salt	$NaNO_2$, NaBr, NaOCl, $NiCl_2$, $(NH_4)_2SO_4$, NaCN

Figure 1: Influence of time on LC50 / EC50 values of acrylonitrile

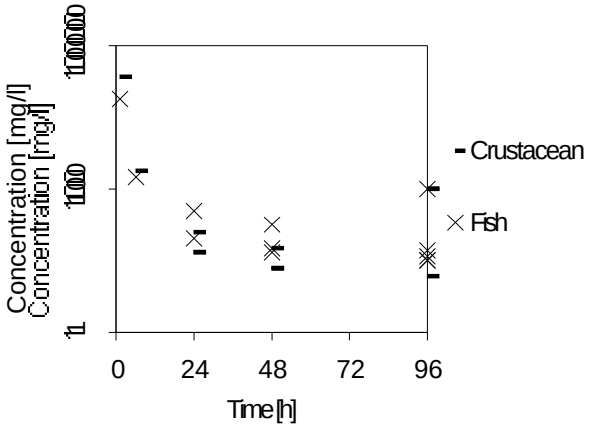


Figure 2: Influence of water flow on toxicity of acetone. s: static, f: flowthrough

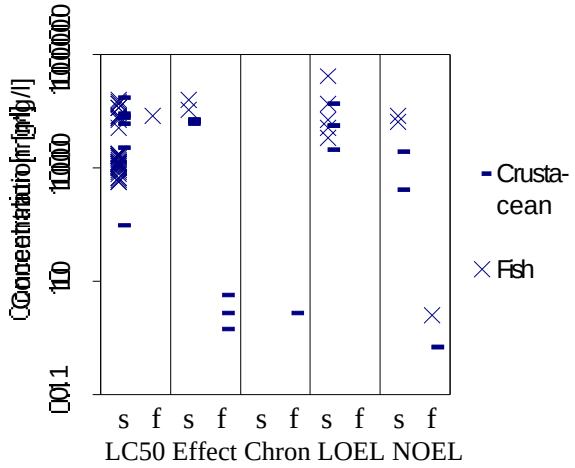


Figure 3: Intra- and interspecies variability in toxicity of toluene to different fish species

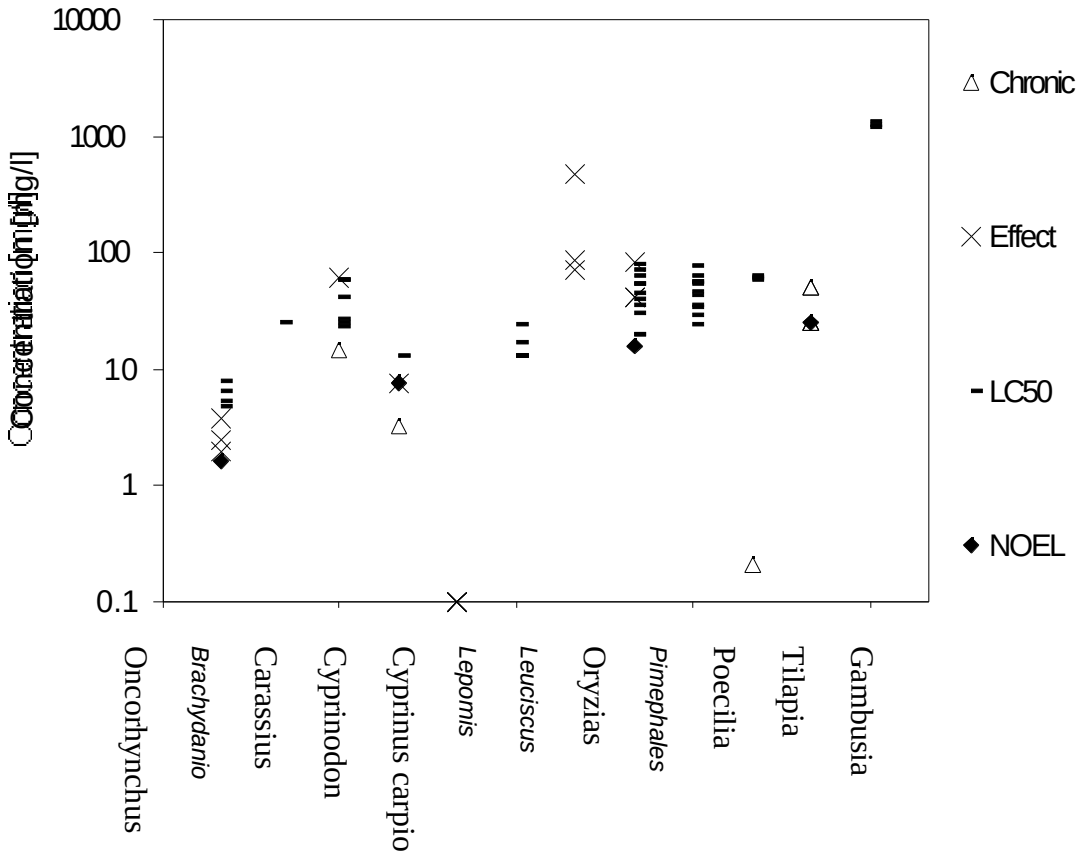


Figure 4: Aquatic toxicity of diethanolamine

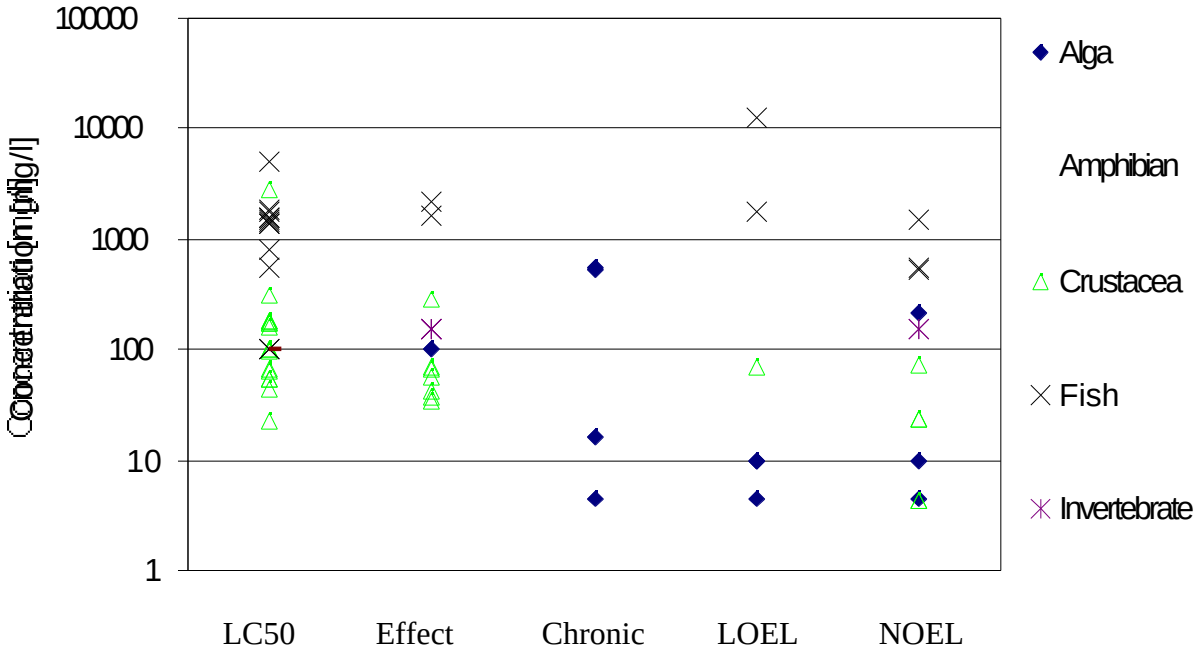


Figure 5: Acute aquatic toxicity (LC20 – LC100, EC20- EC100) of selected substances

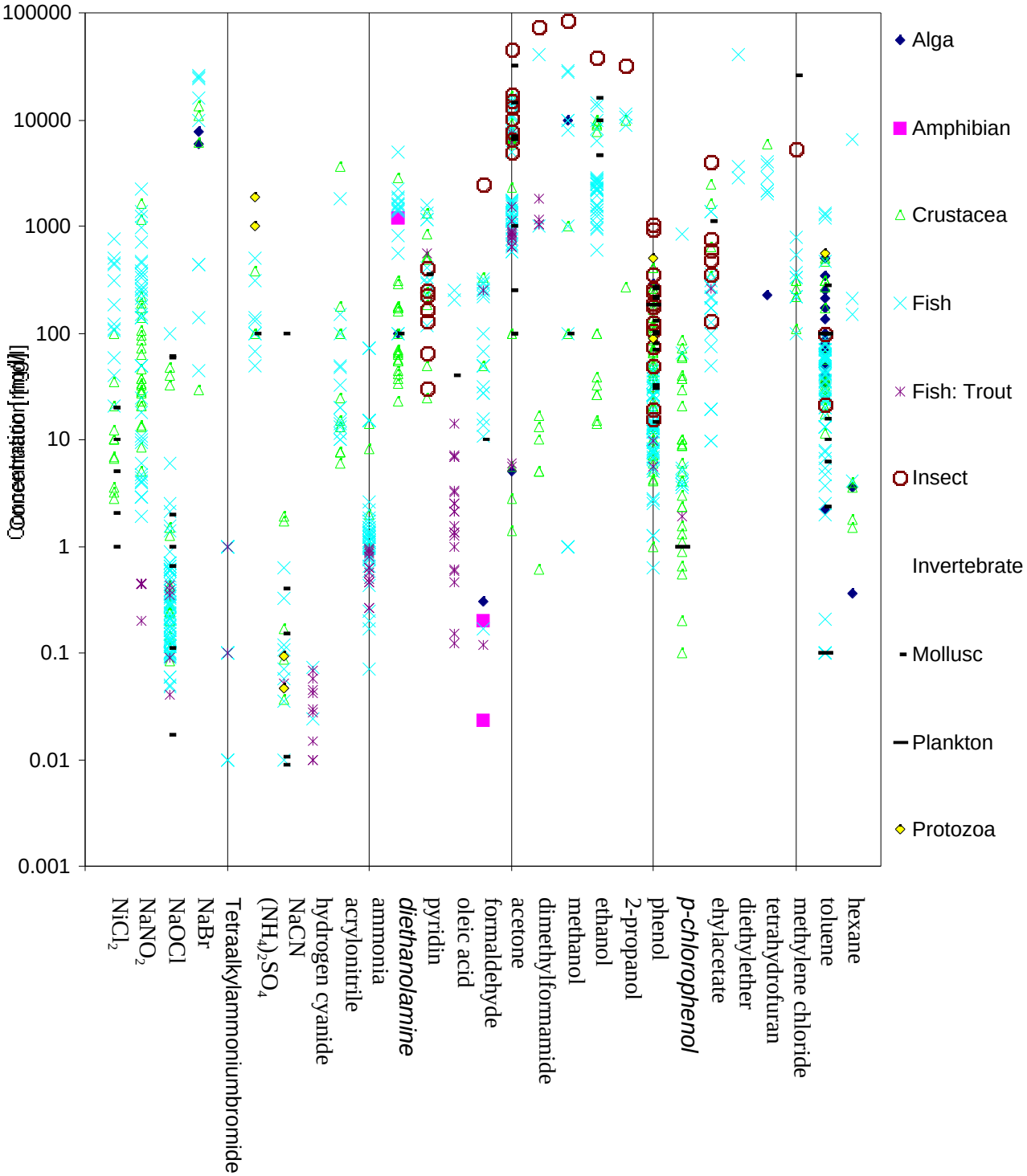
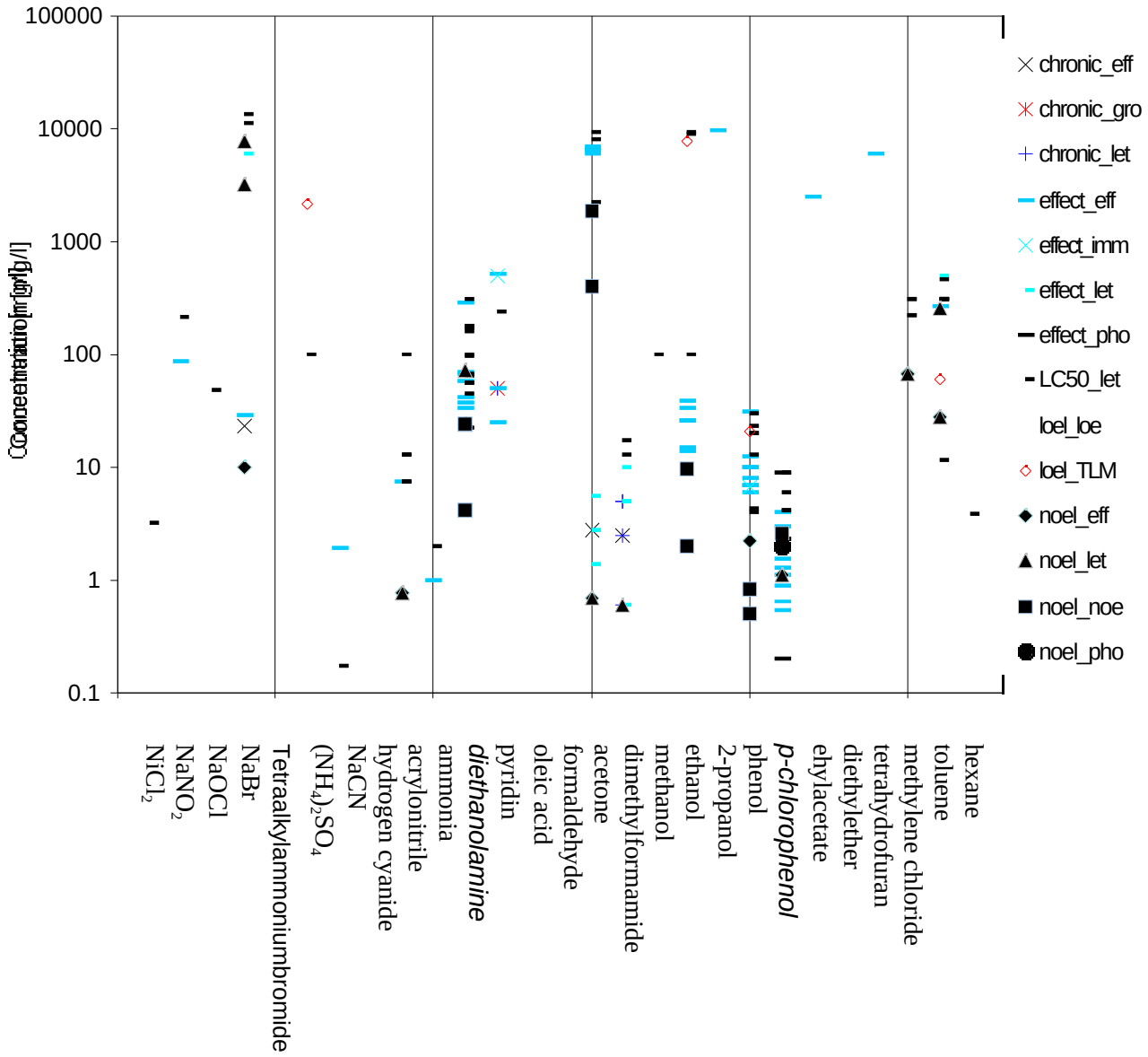


Figure 6: Comparison of endpoints of toxicity to Daphnia magna (_gro: growth _let: mortality, _imm: immobilization, _pho: phototaxis, _eff: other effects, _TLM: probable threshold limit, _loe: LOEL, _noe: NOEL)



MANUELE MARGNI

Life cycle impact assessment of pesticides on human health and ecosystems

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Published in the proceedings of the VITO conference on Life Cycle Assessment in agriculture, agro industry and forestry, Brussels, 3-4 December 98, pp.109-119

Keywords: Pesticides, Life Cycle Assessment (LCA), characterization, impacts, ecotoxicity, human toxicity.

Abstract
This paper presents a life cycle impact assessment method to determine the impact of pesticides on human health and on the ecosystem, considering a full fate and exposure analysis through different pathways, including food residue. The method is applied to the 100 pesticides mostly used in Switzerland. For human toxicity, first estimation of residues shows that food intake causes the highest toxic exposure. Better estimates of pesticides residues are needed in priority. Extrapolation coefficients of 10 commonly applied in risk assessment proved adapted to relate ecotoxicological acute LC50 to chronic NOEC. On the contrary, such factors are not suitable for inter-species extrapolation.
For agricultural management, large impact differences between pesticides with similar function are observed. For wheat, herbicides show impacts variations of a factor up to 10^5 for human health, up to 10^7 for aquatic ecosystem. The method enables the identification of the highest pollution sources and of improvement actions.

1. Introduction

Problem setting

The use of these chemicals in modern farming practices is viewed as an integral part of the success of the agricultural industry. However, most of the pesticides applied to agricultural lands can potentially affect non-target organisms. There is a need to set the real problem concerning the pesticide. On the one hand a chemical are not inevitably bound to a high environmental impact, on the other hand, alternatives methods such as weed control by burning or mechanical processes also generate emissions (Jolliet, 1993[a&b]). Therefore, it is important to go beyond " a-priori " and to be able to quantify the respective impacts of different practices.

Evaluations methods

To determine the pollution potential of pesticides different types of methods have been applied: Transfer models such as those developed by Jury et al. (1987), Leonard (1990) study the fate of the substances. However, these methods concentrate only on the behaviour of pesticides in the environment and they are often not combined with the effect on the receptor population or ecosystem. Ranking methods such as Jouany (1994), and Newman (1995) incorporate different effects, but the

weighting is often made " a priori " without referring to transparent principles. Other methods such as risk assessment take both fate and exposure into account, but they cannot perform a trade off with other types of pollutants. Environmental life cycle assessment LCA enables to assess the environmental impact of products over the whole product life cycle. Methods such as CML 92 (Heijungs et al., 1992) or Ecoindicator (Goedkoop, 1995) incorporated several pesticides, but on a very rough basis, without considering their fate in the environment.

General objectives

The present study answers this need and aims at:

I) The development of a method to evaluate the impact of pesticides on human health and aquatic and terrestrial ecosystem, which enables :
   - To compare different routes of effects (air inhaled, intake in food and water, etc.),
   - To include the modelling of inter-media transfer, especially soil-water, and the intra-media behaviour, determining especially residence times in air and water.
   - To combine fate and effects assessments

II) The application of the method to pesticide management for the most common pesticides used on arable crops in Switzerland.

First the methodological framework is described together with detailed models to calculate the different impact coefficients. Calculations are performed for about 100 pesticides. Finally the impact of practical pesticides application is evaluated and discussed for different crops.

2. Methodology

2.1 Evaluation method of the impact

2.1.1 General framework

This paper proposes to evaluate the impact of pesticides on human health and ecosystems on the basis of semi-empirical method " Critical Surface Time (CST95) " (Jolliet and Crettaz, 1997). This method includes a full fate analysis of different pollutants, referring to the residence time and the dilution volume in each media (air, water and soil) and the effectively absorbed fraction (e.g., in food).

2.1.2 Impact on human health

To compare different substances, in the CST 95 method the following effects are assumed to be equivalent: (1) one person inhales during one year the Human Reference Dose (HRD^{a}) of the substance i or j in air, (2) one person ingests during one year the $HRD^{f\&w}$ of the substance k or l in food or water. According to the equivalency principles, human toxicity can be described as the overall fraction of an emission which is inhaled or ingested by all human beings (the exposure efficiency = the fate factor) divided by the yearly Human Reference Dose (HRD => the effect factor) for both direct

intake and intake through the diet. This Ratio can also be interpreted as the equivalent number of people exposed to the HRD during one year for every kg of substance emitted.

$$F_i^n \,?E_i^n = \varepsilon_i^{mn} \,?\frac{1}{\rho_p \,?N \,?B \,?HRD_i^m} \quad \text{, where} \qquad (1)$$

E_i^n : Effect factor of substance i in medium n (air, water, soil or food chain)

F_i^n : Fate and exposure factor of substance i in the media n.

N : Number of days per year = 365.25 [day/yr]

B : Average body weight = 70 [kg]

HRD_i^m : Human reference dose for inhalation or ingestion [kg/kg-day]

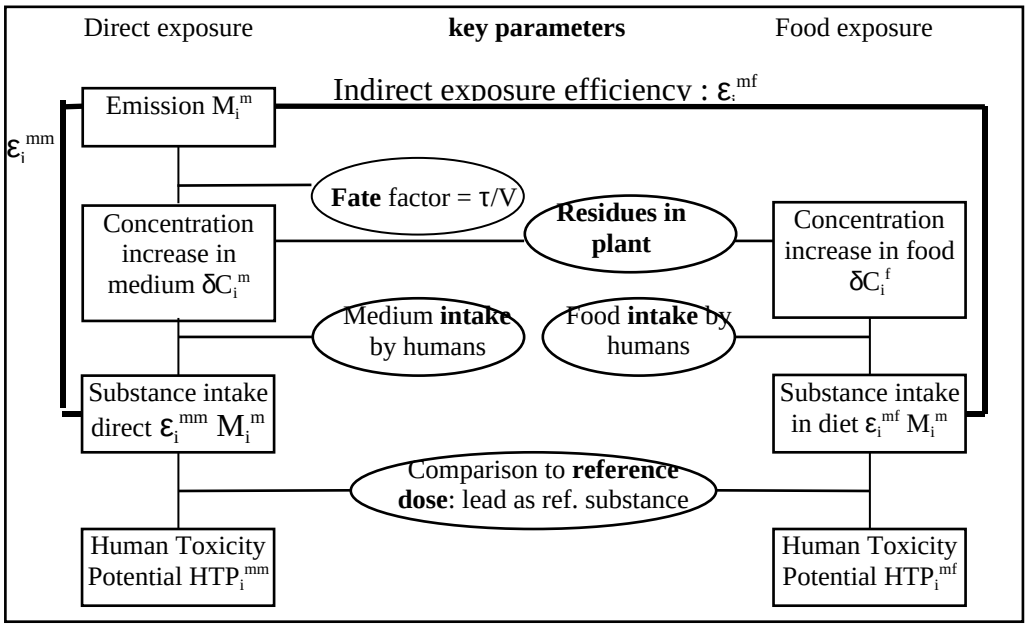

Figure 1 Path of pollutants from emission to human exposure

Jolliet and Crettaz (1998) propose to determine an overall exposure efficiency as a useful concept to characterise fate and exposure - as defined in CST 95 - in a simple and meaningful way. The exposure efficiency ε_i^m can be expressed as the ratio of total human intake to total emissions (fig. 1) and can be calculated as a function of the daily intake of air, water or food multiplied by the residence time and divided by the height of dilution of the pesticide within a given media. To facilitate communication and to use a similar approach to the Global Warming Potential, the Human Toxicity Potential of a substance i (HTP_i^n) is defined by comparing the environmental effect of the substance i with the effect of a reference substance. For human toxicity in CST 95, the effect of the reference substance is arbitrarily chosen as lead (Pb) in air, considering only the inhalation route. Therefore:

$$HTP_i^m = \frac{F_i^m \,?E_i^m}{F_{Pb}^{aa} \,?E_{Pb}^a} \text{, where} \qquad (2)$$

$F_{Pb}^{aa} \,?E_{Pb}^a = 0.13\ m^2\ yr\ kg_{Pb}^{-1}$ for Lead

For pesticides the overall human toxicity potential refer directly to the applied quantities and is given by the summations of the inter-media transfer fraction f_{pn} multiplied by the HTP of the respectively exposure route. The total effect score for the human toxicity is expressed as:

$$S = HTP_i^{tot} \cdot M_i = (f_{pa} \cdot HTP^a + f_{pw} \cdot HTP^w + f_{pf} \cdot HTP^f) \cdot M_i \quad (3)$$

2.1.3 Impact on aquatic and terrestrial ecosystem

Calculations of the ecotoxicity potentials are based on the assumption that two emission are equivalents if they generate their respective No Effect Concentration (NEC) during one year in the entire ecosystem considered. For extrapolation at other concentration levels, the effect is assumed so far to be proportional to the concentration (fig.2). Effect and Fate factors are calculated both for aquatic and terrestrial ecotoxicity similarly. Therefore, one obtains for aquatic ecotoxicity (replace w by s for terrestrial ecotoxicity):

$$F_i^w \cdot E_i^w = \frac{\tau_i^w}{V_i^w} \cdot \frac{1}{NEC_i^w} \quad [\text{m}^2 \text{ yr kg}^{-1}] \quad (4)$$

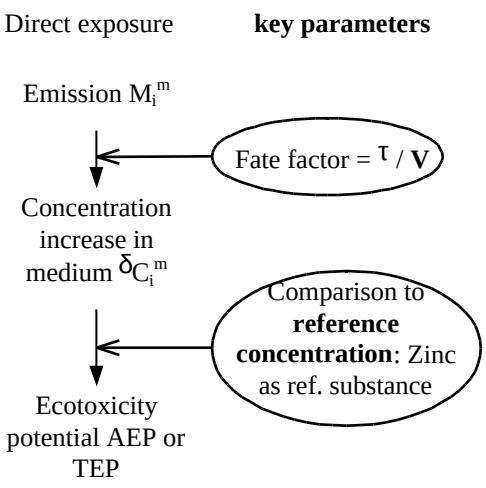

Figure 2 Path of pollutants from emission to concentration increase for ecosystem

The aquatic and the terrestrial ecotoxicity potential of a substance i (AEP res. TEP) are defined by comparing the effect of the substance i with the effect of a reference substance (fig.2). The Zinc (Zn) emitted in water and the zinc (Zn) emitted in soil are arbitrarily chosen.

$$AEP_i^{ww} = \frac{F_i^{ww} \cdot E_i^w}{F_{Zn}^{ww} \cdot E_{Zn}^w} \text{ , where} \quad (5)$$

$F_{Zn}^{ww} \cdot E_{Zn}^w = 5.1\ 10^5$ m^2 yr kg^{-1} for Zinc in water and

$F_{Zn}^{ss} \cdot E_{Zn}^s = 7.1\ 10^6$ m^2 yr kg^{-1} for Zinc in soil

The same inter-media transfer factors as those used for human toxicity can be applied. The total effect score is expressed as: $S = f^{pw} ?AEP^{ww} ?M_i$ (6)

2.2 Determination of the transfer coefficients, residence time and dilution volume (fate factors)

Methodological developments enabled to determine the behaviour of the pesticides in air and the inter-media transfers between soil and surface or groundwater. The selected assumptions and models to calculate these parameters have been detailed for each media by Margni (1997) and Margni et al. (1998) including the values used for all coefficients and the corresponding data sources. Main assumptions are summarised below.

For air, it is assumed that approximately 10% of the applied substances remain in the air or return after the volatilisation processes (DG VI, 1997). The total residence time air is obtained according to the pesticide aerosol-air partition (fraction of sorbed pesticide on the aerosol divided by the substance in gas phase: Finizio et al., 1997), taking into account the residence times of both the aerosol and the substance in the gas phase. The calculated residence times are generally in the order of a few days, with extreme range from a few hours to more than one year.

For soil, the pesticide fraction applied on the soil is estimated to 85% of the total applied quantity. The CST 95 method calculates the ground water emissions according to the model of Jury et al. (1987). The model assume steady water flow, equilibrium linear adsorption, and depth-dependent first-order biodegradation. Margni (1997) defines the parameters that describe the type of soil in which the pesticide ground water transfers are calculated. A new way has been proposed to calculate the pesticide losses to the surface water, taking into account the specific physicochemical characteristics of each pesticide (CREAMS-GLEAMS Model: Leonard et al. 1987). The results show that few substances reach the ground water and the majority of the pesticides runoff losses remain strongly lower than 10 % of the applied dose.

The pesticides residues in food are evaluated according to the tolerance value in food. As a rough estimate, the average residue values in cereals of 5 % of the tolerance value, observed by Elrich (1991) in a case study with chlorothalonil, was generalised to the other pesticides.

2.3 Determination of toxicological and ecotoxicological values (effect factors)

The classical concepts of Human Reference Dose (HRD) as introduced by the US EPA (US Environmental Protection Agency), are used to evaluate the effect of the pesticides on human health for intake through food and drinking water. For the ecosystem "No effect concentration" (NEC) are determined, which establish the quantity of pollutant per unit volume of water or soil which doesn't create damage on the aquatic and terrestrial ecosystem.

No effect concentrations (NEC) for ecosystems and extrapolations factors

The method suggested by Jager et al. (1994), based on risk analysis principles was used in CST 95 to extrapolate the NEC for pesticides in aquatic and terrestrial ecosystems. In this study some indicator species are chosen to characterise the ecosystem sensitivity. For the aquatic ecosystem the present study shows a lack of ecotoxicological data for several pesticides, especially in the case of the algae. In risk assessment a factor of 10 is proposed to extrapolate chronic data on the basis of the acute toxicity data (LC_{50} or EC_{50}). This extrapolation method was tested on 44 pesticides for which both acute and chronic data were available (Margni et al., 1998). This study has shown that the factor of 10 is slightly overestimated but is still representative of the studied pesticides. More than 70% o the values are in the interval 1 to 10 with an average value equal to 7 and a median equal to 4.

When ecotoxicological data for the three species are not available, risk assessment practices assumes additional safety factor of 10 (Jager et al., 1994). This extrapolation was tested on the 53 data where the three species where available (Margin et al., 1998). It showed that for pesticides there is no direct correlation between data for two species and these for three. Taking into account the algae value can decrease the NEC down to a factor 10^4. For substances where the variation of data found in the literature for the same group exceeds a factor 5, both the maximal and the minimal values are taken in account in a sensitivity study (⋖ in figure 3 to 4).

3. Characterization factors

Results are presented in the form of characterization factors used by the CST 95 method. Values of all parameters, which influence directly the fate and effect factors, are presented in details in Margni (1997). The human, aquatic, and terrestrial toxicity potentials are presented in table 1 for more than 100 pesticides. The unit are expressed respectively in equivalent kg of lead emitted in the air, Zinc emitted in the water and Zinc emitted in soil (reference substances), referred to the kg applied pesticide.

For instance, effects of chlorothalonil through residues in food are about 50 times higher than those induced by air inhalation and five orders of magnitude higher than those through consumption of drinking water. The AEP^w of 0.75 $kg_{Zn}{}^w$ /$kg_{chlorothalonil}$ means that the aquatic ecotoxicological impact of 1 kg chlorothalonil applied on the field is equivalent to the impact of 0.75 kg Zn emitted to water.

For human toxicity, it is possible to compare the characterization factors of emissions into air, water and food, which are calculated on the basis of the same reference doses (Table 1). The effects through residues in food are approximately between 100 and 10'000 times higher than those generated by air inhalation and approximately between 1’000 and 10 million times higher than the effects caused by drinking water. This emphasises the importance of improving the assessment of food residues in order to get more reliable results. This also shows the importance of the fate factor in the final result. For the

aquatic ecotoxicity both the runoff losses f^{pw} and the NEC^{w} influence the finals results. For terrestrial ecosystem the NEC^{s} is mostly responsible for the impact variations.

Large variations occur in the human toxicity potentials (per kg of applied pesticide), with range between 118 $kg_{equ.Pb\ in\ water}/kg_{applied\ active\ substance}$ (monolinuron) down to 10'000 smaller value with fluoroglycofen-ethyl. Variation is even large for aquatic ecotoxicity, approximately a factor 100 million between the highest value (chlorpyrifos) and the smallest (teflubenzuron).

Table 1 : Human toxicity potential (HTP), aquatic (AEP) and terrestrial (TEP) ecotoxicity potential. The units are expressed as kg equivalents reference substance per kg active substance applied. For AEP the number of available species used to the NEC extrapolation is specified (Nr. Sp.). The sum of the toxicity potentials via air and water ($HTP_{air+water}$) have to be used for the assessment of non-edible crops (biomass production). They are preceded by the sign ">" if the results of only one of the two pathways are available. Food and total HTP are preceded by the sign "<" if they are based on non detectable value.

		Human health				***Ecosystems***			
						aquatic	***terrestrial***	
Active substance		**HTPa+w** [kg equ. Pb_air/ kg applied]	**HTPf** [Kg equ. P_air/ kg applied]		**HTP** [Kg equ. P_air/ kg applied]	**Nr sp.**	**AEPw->w** [kg equ. Zn_w/ kg applied]	**TEPa->s** [kg equ. Zn_s/ kg applied]	**Application active subst.** [kg appl./ ha]
2,4-D		1.20E-03	< 4.09E-01	<	4.09E-01	3	2.46E-04	3.35E-05	1.08
Aclonifen (Aclofen)		3.43E-05	4.38E-02		4.38E-02	3	3.83E-02	2.39E-05	2.7
Amidosulfuron	>	2.36E-06	6.55E-01		6.55E-01	2	1.39E-04	4.13E-05	0.0225
Asulam		1.59E-05				2	1.54E-05	1.01E-05	2
Atrazin		8.34E-03	< 1.68E+01	<	1.68E+01	3	4.17E-03	7.50E-03	1
Benazolin	>	1.29E-04	< 9.17E-01	<	9.17E-01	2	1.97E-04	1.16E-04	0.45
Bentazone		4.85E-05	< 4.60E-02	<	4.60E-02	3	5.37E-06	1.31E-04	1.92
Bifenox		1.91E-05	1.35E-03		1.35E-03	2	8.37E-05	5.53E-05	0.75
Bifenthrin	>	1.30E-07	< 2.21E+00	<	2.21E+00	3	6.34E-02	1.16E-05	0.02
Bromoxynil als Ester		4.53E-04	< 2.21E-01	<	2.21E-01	2	4.11E-03	6.81E-03	0.4
Carbendazim (L)		1.87E-04	2.08E+00		2.08E+00	2	6.08E-02	1.74E-01	0.2475
Carbendazim (S)							0.00E+00		?
Carbetamide	>	3.42E-06	4.30E-02		4.30E-02	3	6.06E-04	9.85E-04	2
Chloridazon		1.03E-04	< 1.83E-01	<	1.83E-01	3	3.41E-04	1.50E-05	2.58
Chlormequat chloride		3.58E-04	3.84E+00		3.84E+00	2	4.28E-05	5.48E-05	0.92
Chlorothalonil		7.64E-03	3.93E-01		3.93E-01	3	7.47E-01	2.27E-03	1.5
Chlorpyrifos		3.87E-06	3.14E+00		3.14E+00	2	2.43E+00	1.23E-04	0.375
Chlortoluron		4.77E-04	3.27E-01		3.27E-01	2	1.04E-04	4.09E-05	1.8
Clodinafop-propargyl	>	0.00E+00				3	1.87E-08	9.66E-06	0.072
Clomazone		3.78E-05	3.26E-02		3.26E-02	2	4.87E-04	3.01E-04	0.0918
Cloquintocet-mexyl	>	1.15E-10				3	1.07E-06	2.41E-05	0.01785
Cymoxanil	>	2.45E-07	8.43E+00		8.43E+00	3	9.54E-06	3.13E-05	0.1572
Cypermethrin		2.57E-04	< 3.54E-01	<	3.54E-01	3	1.54E-01	7.30E-05	0.05
Cyproconazole	>	3.44E-05	8.24E-01		8.24E-01	3	1.18E-01	1.11E-03	0.07
Cyprodinil	>	2.38E-05	1.18E+01		1.18E+01	2	4.16E-03	7.00E-04	0.75
Deltamethrin		5.13E-04	< 1.57E+02	<	1.57E+02	3	1.95E-01	2.96E-03	0.0075
Desmedipham		1.06E-02	< 1.85E+02	<	1.85E+02	3	5.05E-03	2.17E-04	0.102
Dicamba		1.56E-02	< 1.84E+01	<	1.84E+01	3	9.09E-05	9.03E-05	0.24
Dichlobenil		1.29E-06				3	2.06E-04	2.19E-04	3
Difenoconazol	>	1.78E-07	2.43E-01		2.43E-01	3	3.08E-04	7.76E-04	0.125
Diflufenican		6.15E-04	4.53E-01		4.53E-01	3	6.01E-04	4.06E-04	0.15625

Dimefuron		8.33E-04	1.29E-01		1.29E-01	3	1.03E+00	3.34E-01	1
Dimethenamid		8.79E-05	1.79E-02		1.79E-02	3	2.19E-03	5.06E-04	1.225
Dimethomorph		1.47E-06	2.72E-02		2.72E-02	3	3.74E-04	2.01E-04	0.15
Dinoseb (DNBP)		1.31E-02	< 6.42E+00	<	6.42E+00	2	2.99E-02	4.21E-02	5.1625
Diquat (dibromide)		6.47E-03				2	1.71E-01	2.29E-02	1.6
DNOC		8.17E-03	< 1.68E+00	<	1.68E+00	3	4.19E-05	2.07E-03	3.94
Epoxiconazole	>	1.48E-03	1.16E+01		1.16E+01				0.09375
Ethephon		2.75E-04	7.37E-01		7.37E-01	2	1.01E-05	5.70E-05	0.48
Ethofumesate		9.74E-06	< 5.24E-02	<	5.24E-02	3	2.53E-03	2.02E-03	0.9
Fenpiclonil		9.33E-06	1.18E-01		1.18E-01	3	3.49E-02	1.22E-02	0.03
Fenpropidin	>	5.83E-07				3	1.39E-01	3.75E-04	0.3
Fenpropimorphe	>	1.09E-04	< 3.93E+00	<	3.93E+00	3	1.88E-03	3.90E-04	0.75
Fentin acetate	>	7.05E-05	< 2.01E+02	<	2.01E+02	3	1.52E-01	6.02E-03	0.33
Fentin hydroxide	>	4.16E-05	< 2.56E+02	<	2.56E+02	3	8.99E-02	7.83E-02	0.25875
Fluazifop-P-Butyl	>	4.54E-06	1.41E+02		1.41E+02	3	2.03E-04	5.64E-05	0.28125
Fluazinam (L)						3	1.06E-02	3.62E-04	0.25
Fluazinam (S)						3	0.00E+00	4.25E-04	2.625
Fluorochloridon		1.82E-04	< 1.18E-01	<	1.18E-01	2	4.06E-04	2.30E-04	0.75
Fluoroglycofen-ethyl	>	1.07E-08	4.60E-03		4.60E-03	2	2.18E-05	1.21E-05	0.03
Fluroxypyr	>	4.02E-06	3.41E-01		3.41E-01	3	5.37E-05	4.82E-03	0.1295
Fluroxypyr-als Ester	>	8.81E-11	8.53E-02		8.53E-02	2	8.36E-07	1.23E-04	0.2072
Flusilazole	>	1.11E-02	1.77E+01		1.77E+01	3	7.29E-03	2.71E-03	0.25
Glufosinate-ammon.	>	2.80E-06	< 2.76E-01	<	2.76E-01	3	4.46E-06	2.36E-04	0.8
Glyphosate		4.34E-05	2.76E-01		2.76E-01	3	7.17E-05	1.17E-04	0.8
Hexaconazole	>	3.62E-04	9.43E+00		9.43E+00	3	2.13E-03	8.73E-04	0.1875
Ioxynil		7.39E-03	< 6.45E+00	<	6.45E+00	3	4.87E-05	1.16E-04	0.274175
Isoproturon		1.37E-03	< 4.75E-01	<	4.75E-01	3	1.24E-01	1.47E-05	1.5
Lamda-cyhalothrin		1.16E-03	4.71E+01		4.71E+01	3	7.87E-02	1.73E-04	0.0075
Linuron		3.87E-03	< 4.42E+00	<	4.42E+00	3	7.44E-02	1.75E-03	0.75
Mancozeb	>	3.28E-06	4.60E-01		4.60E-01	1	9.93E-05	5.23E-05	2.4
Maneb		9.57E-05	4.60E-01		4.60E-01	3	4.46E-03	2.31E-03	2.4
MCPA		5.88E-02	< 2.55E+01	<	2.55E+01	3	4.69E-06	5.09E-06	1.155
MCPB		1.61E-06	< 1.47E-03	<	1.47E-03	1	6.73E-06	1.64E-05	1.5
Mecoprop-P	>	1.66E-04	< 3.27E-01	<	3.27E-01	3	9.10E-06	7.81E-05	1.08
Mecoprop (MCPP)		7.38E-03				2	2.38E-05	4.77E-05	0.962
Metalaxyl		2.49E-04	5.52E+00		5.52E+00	3	1.91E-04	1.38E-04	0.2
Metaldehyde	>	2.12E-06				1	1.05E-07	1.14E-05	0.375
Metamitrone	>	1.51E-05	< 1.92E-01	<	1.92E-01	3	1.22E-02	1.01E-04	2.45
Metconazole	>	2.56E-05							0.09
Methabenzthiazuron	>	4.66E-05	< 4.78E-02	<	4.78E-02	3	1.05E-01	3.21E-02	1.54
Methiocarbe		1.10E-05				2	5.33E-03	1.05E-01	0.1
Metolachlor		5.51E-03	< 1.49E+00	<	1.49E+00	3	1.91E-01	4.07E-03	2.64
Metribuzin		3.09E-04	< 4.74E+00	<	4.74E+00	3	1.09E-02	9.37E-04	0.56
Metsulfuron-methyl		8.10E-05	< 8.84E-01	<	8.84E-01	2	1.07E-04	9.46E-05	0.008
Monolinuron		8.52E-03	1.18E+02		1.18E+02	2	1.82E-04	2.16E-04	1.125
Napropamid		3.75E-04	< 4.17E-01	<	4.17E-01	2	1.42E-04	3.13E-04	1.2375
Nicosulfuron	>	5.52E-09				2	4.92E-06	7.10E-05	0.05
Orbencarb	>	8.55E-04	< 1.72E-01	<	1.72E-01				3.718
Oxadixyl	>	1.65E-03	< 1.33E+01	<	1.33E+01	2	4.27E-05	2.53E-04	0.2
Pendimethalin		1.19E-03	< 5.52E-01	<	5.52E-01	3	2.79E-02	2.74E-04	1.6
Phenmedipham		4.28E-04	9.10E-01		9.10E-01	3	1.42E-04	2.57E-05	0.8635
Pirimicarb		1.52E-04	5.89E-01		5.89E-01	3	9.39E-02	9.31E-04	0.075
Prochloraz		4.39E-04	1.96E+00		1.96E+00	3	9.77E-04	1.58E-05	0.45
Propamocarb (HCL)	>	3.17E-05	1.34E+01		1.34E+01	3	9.31E-06	5.80E-06	0.992
Propaquizafop		2.56E-05	4.12E-02		4.12E-02	3	1.21E-04	3.38E-05	0.125
Propiconazole		1.75E-04	8.84E-01		8.84E-01	3	7.35E-03	3.12E-03	0.125

Prosulfocarb		1.07E-03	2.76E-01		2.76E-01	3	5.53E-02	5.63E-04	3.2
Pyridate	>	8.36E-08	< 1.03E+00	<	1.03E+00	3	1.35E-05	2.11E-05	0.675
Rimsulfuron	>	4.49E-08	1.35E+00		1.35E+00	3	1.29E-04	1.01E-05	0.00875
Simazine		1.84E-03	2.36E+00		2.36E+00	3	1.49E-02	3.01E-04	1
Tebuconazole		7.24E-03	5.89E+00		5.89E+00	3	1.15E-01	1.60E-03	0.25
Tebutam	>	1.78E-06	< 1.95E-03	<	1.95E-03	1	1.67E-04	4.77E-05	3.3
Teflubenzuron		1.29E-03	7.37E+00		7.37E+00	1	4.95E-08	2.04E-08	0.06
Terbufos		1.93E-04	< 1.18E+02	<	1.18E+02	3	1.00E+00	1.61E-04	0.25
Terbuthylazin		2.63E-03	5.94E+00		5.94E+00	2	1.91E-04	2.51E-04	0.425
Thifensulfuron-methyl		1.04E-03	2.51E+01		2.51E+01	2	1.88E-05	4.06E-04	0.0075
Triasulfuron	>	2.80E-06	1.37E-01		1.37E-01	2	3.23E-05	3.21E-05	0.075
Tridemorph	>	4.47E-06	9.35E-01		9.35E-01	3	3.71E-03	1.27E-03	0.2625
Trifluralin		7.54E-05	< 7.67E-02	<	7.67E-02	2	5.66E-04	3.04E-05	1.2
Triflusulfuron	>	6.07E-08	1.24E-03		1.24E-03	2	1.14E-07	1.01E-05	12.5
Trinexapac-ethyl						2	1.60E-05	1.02E-03	0.175

4. Application to agricultural practices

If characterization factors are suitable to estimate the impact per kg of applied substances, a high characterisation factor does not always implies a high level of pollution. In fact, the applied quantities to ensure a given function (weed control) can also vary of several orders of magnitude between different pesticides and need to be combined to the indicators.

Damages are calculated on the basis of the applied quantity to achieve a similar function per hectare (table 1 last column). For the aquatic ecosystems, a maximal variation interval of four orders of magnitude is added to the potential damage generated by the active substance, when these is based on insufficient qualitatively or quantitatively data (see chapter 2.3.).

Human health

Damages on human health are calculated assuming that the yield is eaten by humans. Figure 3 shows the damages of herbicides on human health. Differences in the impact are high, up to five orders of magnitude (MCPA - Fluoroglycofen-ethyl) for treatments on wheat and up to three for potatoes, corn and beets. This range is smaller for fungicides on wheat (two orders of magnitude) and it is clearly reduced for insecticides.

Aquatic ecosystems

Striking variations occur for the damages of herbicides applications on aquatic ecosystems: more than eight orders of magnitude for wheat (figure 4), six for corn, and al least five for rapes, beets, potatoes and green land. For fungicides the chlorothalonil is the active substance which has the largest impact. Two types of interval are used for the impact comparison in aquatic ecosystem. The first is linked to the variations in ecological data to determine the NEC (↔), the second is a fixed 10^4 interval of variation that take in account the missing ecotoxicological data (¾). For some pesticides, with a variation interval, their applications remain in any case less dangerous for the environment compared to other substances without variation interval (e.g.: fluroxypyr compared to isoproturon in wheat, figure 4). On

the contrary, other active substances have already a high potential damage, even without considering the variation interval. Impacts on terrestrial ecosystems are presented and discussed by Margni et al. (1998)

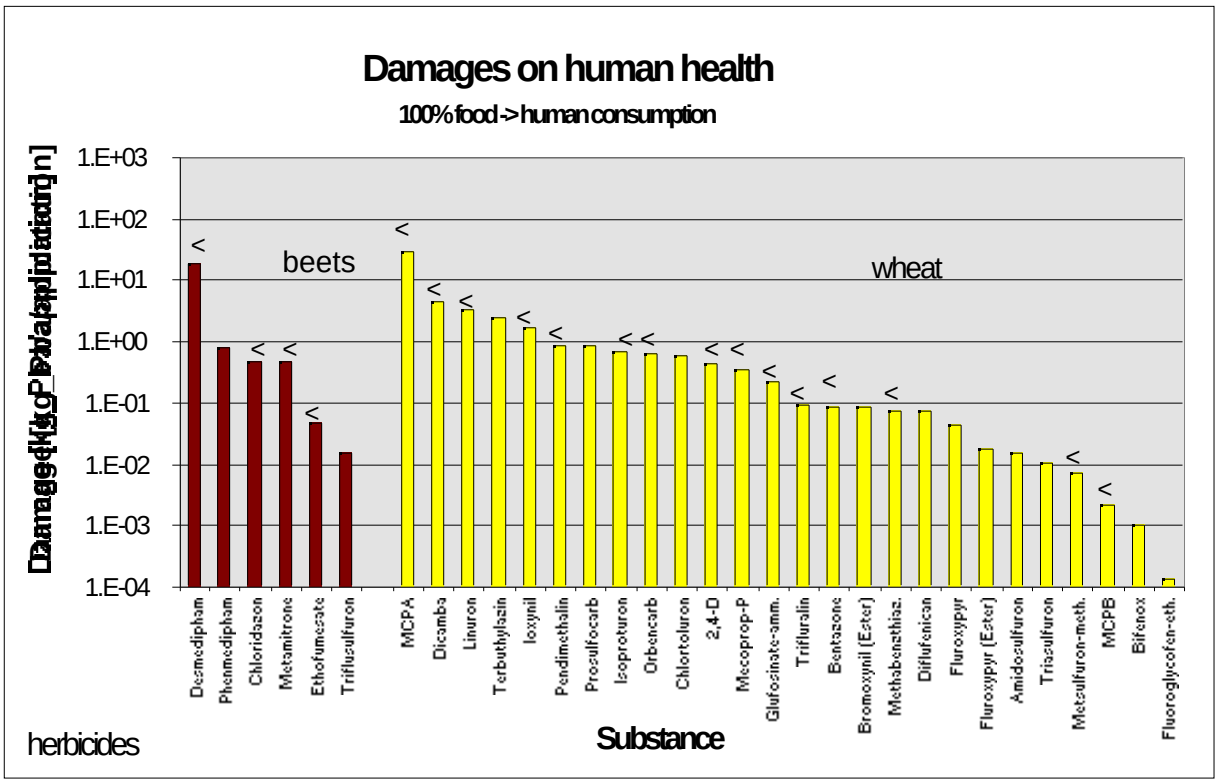

Fig. 3: Damages on human health: herbicide applications on beets and wheat.

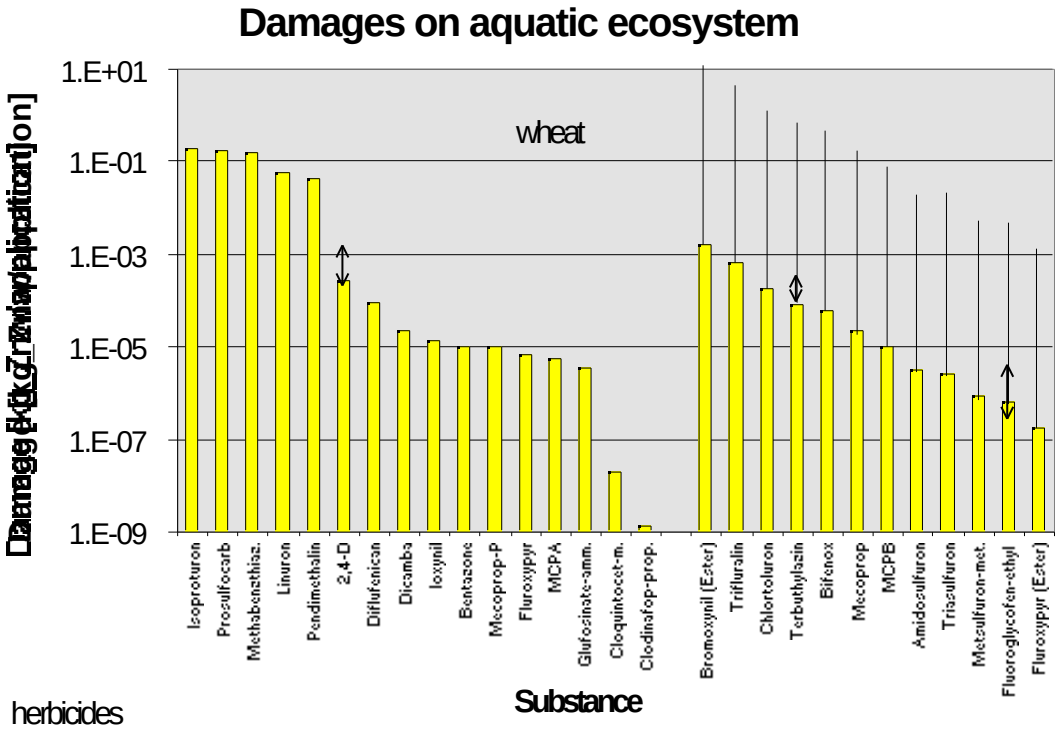

Fig. 4: Damages on aquatic ecosystem: herbicide applications on wheat.

To evaluate active substances in a global way, theirs damages have to be compared for the three impact category (human health, aquatic and terrestrial ecosystem). If the substance causes more damage simultaneously in human health in aquatic and in terrestrial ecosystems, it can clearly deduced that this substance produce more impacts compared to another, which follows in the classifications of the three respective impact categories.

5. Conclusion and perspectives

The present work has provided a consistent method to assess the environmental impacts of pesticides, enabling the coherent combination of fate and effect. Nevertheless, the current state of the LCA methods allows only a rough impact assessment, aiming at orders of magnitude. The obtained results must first be considered as a mean to compare pesticides. If required detailed assumptions (soil characterisation, water dilution volumes, hydrological system, etc.) could be adapted to the specific conditions of the studied system to approach as much as possible the real situation.

Here, only the applied active substances are evaluated. For some substances the principal metabolites are as toxic as the original molecules. To determine the total score the impacts of the toxic metabolites should be evaluated in a similar manner and added to that of the applied substance. Basic scientific studies should be undertaken in priority to evaluate better the pesticides residues in food, that potentially represent the most important exposure pathway for human. To evaluate the damage on ecosystems, lacks on ecotoxicological data are obvious, especially for soil organisms.

Uncertainties are mainly dominated by the toxicological data with possible variation that range from factor 10 to 100. For aquatic ecotoxicity, the impact variations are up to 8 orders of magnitude, so the comparisons can be supposed valid in case of large differences even if a detailed error analysis were not carried out. It would be necessary for further studies to use probabilistic methods, at the example of the Monte Carlo method. Combined with a sensitivity analysis that should allow to measure the uncertainty inherent to the data and to identify the parameters with have the largest influences on the final results.

In the light of the obtained results, it can be affirmed that we dispose at present a tool for the environmental evaluation of pesticide impacts, which provide a global evaluation of the damage caused by their use in arable crops. The results of this study can start to be used: on the one hand the pesticides impacts can be compared to other agricultural practices; and to the other hand the results of this study can be used to the agricultural consulting to justify and promote management systems and pesticide choices that respect at most the environment, without penalising the culture itself.

ACKNOWLEDGMENTS

The following institutions and people are sincerely to acknowledge for supporting this study : service romand de vulgarisation agricole (srva) and foundation NESTEC for financing respectively the study and the publication. Special thanks to Raphael Charles at the Swiss Federal Research Station for Plant Production (RAC) and G.

Gaillard at the Swiss Federal Research Station for Agricultural Economics and Engineering, (FAT) for their support in data search.

6. Bibliography

DG VI, European Commission Agriculture. 1997.Harmonisation of environmental life cycle assessment for DFG. 1990. Pflanzenschutzmittel im Trinkwasser. DFG, Mit teilung XVI der Kommission für Pflanzenschutz-, Pflanzenbehandlungs- und Vorratsschutz mittel, VCH Verlag, D-6940 Weinheim

agriculture, 3rd Draft of Final Report Concerted Action AIR3-CT94-2028.

Eilrich, G. L. 1991. Tracking the fate of residues from the farm gate to the table, a case study. In Pesticides residues and food safety, American Chemical Society, pp.203-212

Finizio, A., D. Mackay, T. Bidleman and T. Harner. 1997. Octanol-air partition coefficient as a predictor of partitioning of semi-volatile organic chemicals to aerosols. Atm. Environment, Vol.31, PP. 2289-2296.

Goedkoop, M. 1995. Eco-indicator 95, weightening method for environmental effects that damage ecosystems or human health on a European scale, Final report, RIVM.

Hauschild and Wenzel, 1997. Environmental Assessment of Products. Volume 2: scientific background, Chapman & Hall, London. P. 257.

Heijungs, R. et al.. 1992. Environmental Life Cycle Assesment of products, Background and Guide. Centre of Environmental Science (CML), NL-2300 RA Leiden.

IPCS, 1996. Inventory of International Programme on Chemical Safety and other WHO pesticide evaluations and summary of toxicological evaluations performed by the Joint Meeting on Pesticide Residues (JMPR). WHO, Geneve, not a formal pubblication, 57

Jager, D.T., T.G. Vermeire, W. Slooff, H. Roelfzema. 1994. Uniform System for the evaluation of substances II Effet Assessment. Chemosphere 29, p. 319-335.

Jolliet, O., 1993[a]. ***Ökobilanz von thermischer, mechanischer und chemischer Kartoffelkrautbeseitigung***.

Landwirtschaft Schweiz, Band 6 (11-12).

Jolliet, O., 1993[b]. ***Life cycle analysis in agriculture: comparison of thermal, mechanical and chemical processes to destroy potato haulm***. Expert Seminar on Life cycle Assessment of Food products, Technical University of denmark, 22-23 November 1993.

Jolliet, O. and P. Crettaz. 1998. Assessment of Human Toxicity in Life Cycle Assessment, including fate and exposure: the critical surface-time method. Submitted to Risk Analysis, an international Journal

Jolliet, O. and P.Crettaz. 1997a. Critical surface-Time 95. A life cycle impact assessment Methodology including fate and exposure, Swiss Federal Institute of Technology, Institute of Soil and Water Management, Lausanne.

Jouany, J.M. 1995. Etablisssement d'une liste de substances phytosanitaires utilisées en agriculture à surveiller dans le bassin lémanique: Méthodologie. 1994. Rapp. Comm. Int. Prot. eaux Léman contre pollution, campagne 1994, 217-233.

Jury, W.A., D.D. Focht and W.J. Spencer. 1987. Evaluation of pesticide groundwater pollution from standard indices of soil-chemical adsorption and biodegradation. J. Environ. Qual. 10 (4), 422-428.

Leonard, R.A. 1990. Movement of Pesticides into Surface Waters. In:Pesticides in the Soil Environment: Processes, Impacts, and Modeling-SSSA Book Series, no.2, Segoe Rd., Madison, WI.

Leonard, R.A., E.G. Kinsel and D.A. Still. 1987. GLEAMS : Groundwater Loading Effects of Agricultural Management Systems. Transactions of the ASAE, Vol. 30(5), pp. 1403-1418.

Margni, M. 1997. Impact des pesticides sur la santé humaine et les écosystèmes: évaluation par la méthode des écobilans. Mémoire de recherche pour l'obtention du diplôme d'études postgrades en ingegnerie et management de l'environnement. Superviseur: Dr. O. Jolliet, EPFL-DGR/IATE/HYDRAM, Lausanne.

Margni et al., 1998. Life cycle impact assessment of pesticides on human health and aquatic and terretrial ecosystems. In preparation

Newman, A. 1995.Ranking Pesticides by Environmental Impact. Env. Science & Technology, vol.29, No 7.

Nowell, L.H. and E.A. Resek. 1994. National Standards and Guidelines for Pesticides in Water, Sediment, and Aquatic Organisms : Application to Water-Quality Assessments. Reviews of Environmental Contamination and Toxicology, Vol. 40.

WHO. 1990. Principles for the Toxicological Assessment of Pesticide Residues in Food. Environmental Healt Criteria 104, Geneva.

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Von der Extrapolation von Nicht-Risiken zur Abschätzung potenzieller Schäden

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Präambel

Dieser Beitrag fokussiert ausschliesslich auf die Beeinträchtigung der menschlichen Gesundheit durch Umweltchemikalien und klammert damit alle weiteren Effekte, insbesondere jene auf die Ökosphäre, aus. Die Ökobilanz der Chemikalien selbst bleibt ebenfalls unbeachtet, da nur bereits emittierte Chemikalien diskutiert werden. Im weiteren wird davon ausgegangen, dass die Dispersion, der Abbau und die Kompartimentswechsel (also das Stoffschicksal) und die Exposition des Menschen bereits berücksichtigt werden, wie dies heute state-of-the-art ist (Guinée ***et al.*** 1996, Jolliet 1996, Goedkoop ***et al.*** 1998, Wegener Sleeswijk und Jolliet in diesem Dokument).

Zusammenfassung

Die heutige Praxis der Bewertung von humantoxischen Schadstoffen in Ökobilanzen entspricht der Extrapolation von Nicht-Risiken. Dieser Beitrag stellt dagegen die Abschätzung potenzieller Schäden in den Vordergrund da diese entscheidungsrelevant und mit anderen Gesundheitsbeeinträchtigungen vergleichbar sind. Resultate epidemiologischer Studien verbessern die Möglichkeit der Abschätzung potenzieller Schäden bereits deutlich. Solche Studien liefern jedoch nur für wenige Substanzen Dosis-Wirkungsbeziehungen. Ein hier vorgestelltes Konzept kombiniert die Resultate epidemiologischer Studien mit Informationen aus der Arbeitsplatzhygiene, die teilweise auf toxikologischen Tests beruhen. Die Ausarbeitung des vorgestellten Konzeptes würde den vorgezeichneten Übergang von der Extrapolation von Nicht-Risiken hin zur Abschätzung potenzieller Schäden weiter stützen, da hiermit die Zahl bewertbarer Schadstoffe kaum zurückgehen würde.

1. Ausgangslage: Die Extrapolation von Nicht-Risiken

Die Methode der kritischen Volumina hat in der Ökobilanzierung und gerade auch in der Schweiz eine lange Tradition (Jansen ***et al.*** 1972, Basler & Hofmann 1974, BUWAL 1984, BUWAL 1991). In dieser Methode wird angenommen, dass die Höhe der verordneten Immissionsgrenzwerte ***G***[1] [kg/m3] der Substanz ***i*** emittiert ins Kompartiment ***k*** gerade einem guten Indikator für die reziproke

$$U_k = \frac{I_{ik}}{{}_i G_{ik}} \qquad [\mathrm{m}^3/\text{funktionelle Einheit}] \qquad (1)$$

Umweltgefährdung ***U*** entspricht:

wobei ***Ii,k*** für die Emission der Substanz ***i*** ins Kompartiment ***k*** steht und in kg pro funktionelle Einheit gemessen wird. Analog haben Heijungs ***et al.*** (1992) je nach Verfügbarkeit den TCL (acceptable concentration in air), AQC (Immissionsgrenzwert der air quality guideline), den TDI (tolerable daily intake) und den ADI (acceptable daily intake) vorgeschlagen.

[1] Diese Methode wird analog auch mit maximalen Arbeitsplatzkonzentration (MAK) oder noch akzeptablen täglichen Aufnahmeraten (ADI) verwendet.

Bemühungen zur Addition dieser kompartimentsweisen Umweltgefährdungsindikatoren und erste Ansätze zum Einbezug des Stoffschicksals folgten diesen frühen Entwicklungen (Thalmann-Graf 1991, Hofstetter 1991/1993, Schaltegger ***et al.*** 1992, Heijungs ***et al.*** 1992, Gebler 1992, Guinée ***et al.*** 1993, Jolliet 1993).

Neuere operable Methoden zur Bewertung von toxischen Substanzen beruhen noch immer auf dem Prinzip von Formel (1), wobei zum Teil umfassende Stoffschicksal- und Expositionsmodellierungen vorgeschaltet werden (Guinée ***et al.*** 1996, Jolliet ***et al.*** 1997, Wenzel ***et al.*** 1997, Huijbregts 1999).

Diese Arbeiten und Methoden extrapolieren im wesentlichen deklarierte 'Nicht-Risiken', um einen Indikator für die Gefährdung der menschlichen Gesundheit zu berechnen. In Abbildung 1 sind drei Dosis-Effekt-Punkte aus Formaldehyd-Begasungsversuchen mit Ratten eingezeichnet. Bei diesem Beispiel geht es nun nicht darum, dass gerade im Bereich der Kanzerogenität davon ausgegangen wird, dass es keine sicheren Expositionskonzentrationen gibt, sondern um die Frage, welche Information aus solchen Toxizitätsdaten zur Verfügung stehen. Falls es einen Grenzwert gibt, unterhalb dessen keine Effekte (in diesem Fall Nasaltumore) beobachtet werden können, so liegt dieser im gezeigten Beispiel irgendwo zwischen 0 und 5.59 ppm. Aufgrund der vorhandenen Information kann dabei nicht abgeschätzt werden, ob eine solche NOEC (no observable effect concentration) näher bei null oder 5.6 ppm liegt. Um Resultate von Tierversuchen auf den Menschen zu übertragen und um der grossen Variationsbreite der Sensitivität von Lebewesen Rechnung zu tragen, werden Unsicherheitsfaktoren eingeführt (z.T. in der Höhe von Faktor 1000!). Eine NOEC für den vorliegenden Fall würde daher nahe bei Null zu liegen kommen (siehe Abb. 1). Dieser Punkt ***(X)*** wird nun in den genannten 'kritische Volumina'-Methoden für die Effektabschätzung verwendet. Wie Abbildung 1 zweifelsfrei zeigt, enthält ***X*** keinerlei Effektinformation. Es ist daher zumindest fraglich, wie entscheidungsrelevant Bewertungsmethoden sind, die auf der Extrapolation von 'Nicht-Risiken' beruhen.

Es ist wichtig hier anzufügen, dass selbst NOEC in der Umwelt selten überschritten werden, d.h., nach toxikologischen Gesichtspunkten keine Gesundheitseffekte zu erwarten sind. Da die Ökobilanz ***sensu stricto*** der Maxime folgt, dass eine tiefere Belastung immer besser ist (***less is better***), werden auch Emissionen, die zu keiner Schwellenwertüberschreitung führen, gleich behandelt wie solche, die zu einer Überschreitung führen.

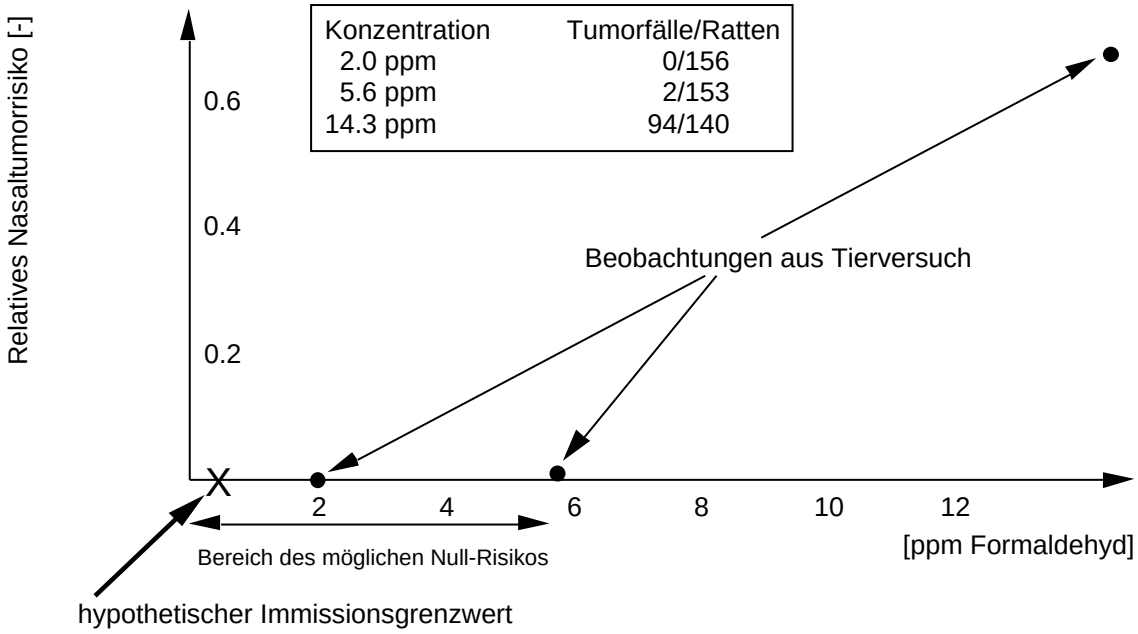

Abb. 1: Typische Information aus Tierversuchen am Beispiel von Nasaltumorbildung aufgrund Formaldehydexposition bei Ratten (Daten aus Lutz 1997)

2. Ziel: Abschätzung potenzieller Schäden

In Entscheidungen ist es wenig hilfreich zu wissen, wieviel m^3 Luft oder Liter Wasser während einem Jahr bis zu einem Schwellenwert belastet werden und damit gerade noch mit einer gewissen Sicherheit unschädlich sind. Während der alleinige relative Vergleich von toxischen Substanzen mit der Extrapolation von 'Nicht-Risiken' in gewissen Fällen Sinn macht, verunmöglicht dieser Weg das Einbeziehen anderer Umwelteinwirkungen, die ebenfalls die menschliche Gesundheit schädigen und meist separat ausgewiesen werden (z.B., Sommersmog, Lärm, Ozonschichtabbau, Treibhauseffekt).

Um die Entscheidungsrelevanz zu erhöhen und gleichzeitig die Vergleichbarkeit mit anderen Umwelteinwirkungen auf die Gesundheit zu ermöglichen, wird hier postuliert, dass der Pfad der 'Nicht-Risiken'-Extrapolation verlassen werden muss und stattdessen Schädigungen an der menschlichen Gesundheit direkt abgeschätzt werden sollen.

Da in Ökobilanzen oftmals weder Ort und Zeitpunkt der Emission noch die Hintergrundbelastung am Ort der Exposition bekannt sind, ist die Expositionsabschätzung aufgrund der Emissionen eine Durchschnittsbetrachtung, die den immer individuellen Emissionssituationen nur in seltenen Fällen gerecht wird. Aufgrund dieser Ausgangslage muss das formulierte Ziel daher relativiert werden; es sollen lediglich potenzielle Schädigungen der menschlichen Gesundheit berücksichtigt werden.

3. Stellenwert von epidemiologischen Studien

Umweltepidemiologische Studien kommen diesem formulierten Ziel recht nahe. Die Umweltepidemiologie arbeitet nicht mit Tieren sondern Menschen, macht keine Versuche sondern beobachtet besonders stark und besonders schwach exponierte Bevölkerungsgruppen (Kontrollgruppe) und

versucht alle relevanten Kofaktoren mitzuerfassen. Aus der Vielzahl verschiedener Studiendesigns sind vor allem die Kohortenstudien über lange Zeiträume sehr wertvoll, da damit eine gute Kenntnis der Kofaktoren erreicht werden kann und die viel relevanteren chronischen Wirkungen von Schadstoffen untersucht werden können.

Typischerweise liefern epidemiologische Studien Zusammenhänge zwischen der relativen Zunahme einer Krankheit oder von Todesfällen und einem Indikatorschadstoff, welcher den signifikantesten Zusammenhang mit den untersuchten Endpunkten zeigt. Epidemiologische Studien können die Kausalität solcher Zusammenhänge nicht zeigen, sondern sind auf Erklärungen und Versuche der Toxikologie angewiesen.

Da die einzelnen Substanzen in der Umwelt oft in tiefen Konzentrationen auftreten und die Toxikologie chronische Effekte noch wenig erforscht hat, klafft hier oft eine grosse Erklärungslücke. Obschon synergistisches Verhalten von nur ganz wenigen Substanzen toxikologisch gezeigt werden konnte, wird davon ausgegangen, dass das Zusammenwirken der unzähligen Umweltschadstoffe zu den beobachteten Wirkungen führt. In diesem Zusammenhang wird auch oft auf den Umweltschadstoff***cocktail*** verwiesen, welcher deutlich schädlicher ist als die Summe aller erwarteten Einzelwirkungen. Aufgrund dieser Erkenntnisse ist es gerade für Bewertungsmethoden wichtig, Schadstoffe als Teil dieser Cocktails zu bewerten und, z.B., bei der Bewertung von Gesundheitsschäden durch Partikel nicht auf toxikologischen Studien mit künstlich hergestellten Partikel aus Teflon abzustellen. Epidemiologische Studien sind geeignet, Effekte in realen Systemen zu studieren.

Diese optimistische Bewertung von epidemiologischen Studien muss nun jedoch besonders für umweltepidemiologische Studien wiefolgt relativiert werden:

- Epidemiologische Studien können keine kausalen Zusammenhänge belegen.
- Es wird meist angenommen, dass die individuelle Exposition dem gewichteten Mittel aller Messstationen entspricht.
- Damit wird auch meist nicht berücksichtigt, dass sich Menschen zu mehr als 22 Std. in Gebäuden aufhalten, wobei die erwarteten Konzentrationen in Innenräumen für jene Substanzen, die auch im Innenraum emittiert werden meist höher sind und für die anderen durchwegs tiefer.
- Bei chronischen Effekten wird meist angenommen, dass der Konzentrationsverlauf über viele Jahre nicht relevant ist und lediglich die integrierte Aufnahmemenge relevant ist.
- Es wird angenommen, dass sich die Studienbevölkerung ständig im Studiengebiet aufhält und die Exposition immer derjenigen Konzentration entspricht, die der dem Wohnort am nächsten gelegenen Messstation entspricht.

- Studien zu chronischen Effekten sind viel seltener und auch deutlich aufwendiger in der Durchführung.
- Obschon unterschiedliche Lebensstilfaktoren wie Rauchen und Klimavariablen wie Temperatur, Feuchtigkeit und z.T. Wind als Kofaktoren meist berücksichtigt werden, bleiben viele andere Kofaktoren ausgeklammert. Jenkins ***et al.*** (1996) fordern z.B. den Einbezug der körperlichen Aktivität der Individuen als Kofaktor.
- Unexponierte Kontrollbevölkerungen können kaum noch gefunden werden, womit immer grössere Studienpopulationen benötigt werden, um signifikante Zusammenhänge finden zu können. Unfälle, Katastrophen und erhöhte Belastungen am Arbeitsplatz ermöglichen Studien auch mit kleineren Personengruppen.

Ausserdem – und dies ist gerade für die Ökobilanzbewertung von zentraler Bedeutung – ist gerade der letzte Punkt dafür verantwortlich, dass epidemiologische Studien nur für wenige Schadstoffe verfügbar sind (siehe nächster Abschnitt). Nur für wenige der über 100'000 Chemikalien werden je epidemiologischen Studien mit signifikanten Resultaten zur Verfügung stehen.

Aufgrund der realen Randbedingungen und den entscheidungsrelevanten Effektinformationen sollten die verfügbaren epidemiologischen Studien unbedingt in die Bewertungen einfliessen. Gleichzeitig muss realistischerweise eingestanden werden, dass für die in Ökobilanzen relevanten Schadstoffe niemals alle nötigen Studien zur Verfügung stehen werden.

4. Gibt es sichere Schadstoffkonzentrationen?

Für karzinogene Stoffe und ionisierende Strahlung wird schon lange postuliert, dass es wohl keine Konzentrationen mit Null-Risiko gibt. Diese Vermutung hat einerseits mit den bisher bekannten Erklärungen für die ablaufenden Mechanismen zu tun, andererseits sind für diese Belastungen schon früh epidemiologische Studien gemacht worden, welche ganze Populationen erfassen.

Neuere Resultate epidemiologischer Studien legen nun nahe, dass es wohl in den meisten Fällen auf der Ebene von Populationen keine sicheren Grenzwerte gibt, unterhalb derer keine Effekte zu beobachten wären. Dies schliesst nun nicht aus, dass gesunde Individuen bei heutigen Umweltbelastungen keinerlei Symptome zeigen, sondern sagt lediglich, dass in der Bevölkerung immer vorgeschädigte Personen und Risikogruppen leben, welche auch bei sehr kleinen Zusatzexpositionen Effekte zeigen.

Diese Erkenntnis legt es nahe, dass die Vorstellung eines Schwellenwertes, unterhalb jenes keine Beeinträchtigungen entstehen, aufgegeben wird und stattdessen lediglich eine allenfalls unterproportionale Wirkung angenommen wird. Diese Erkenntnis stützt ausserdem die oben erwähnte, in Ökobilanzen häufig gemachte Annahme des ***less is better***.

5. Berechnung potenzieller Schäden mit Hilfe epidemiologischer Studien

Steen ***et al.*** (1992) waren unter den ersten, die Informationen aus epidemiologischen Studien für Ökobilanzbewertungsmethoden genutzt hatten. Studien zur Berechnung externer Kosten durch Umwelteinwirkungen basierend auf dem Schadenskostenansatz leisteten als Erste die breite Aufarbeitung vorhandener epidemiologischer Informationen (ESEERCO 1995, ExternE 1995, IER 1997).

Hofstetter (1998) schlägt eine an die Ökobilanzierung angepasste generische Modellierung von Gesundheitsschäden aufgrund von Emissionen vor. Die Stoffschicksals- und Expositionsanalysen wurden dabei mit modifizierten Multimediamodellen, Resultaten aus aufwändigen Dispersionsmodellen und Modellen zur Populationsdichte erstellt. Die Effektanalyse basiert hauptsächlich auf Resultaten epidemiologischer Studien und die abschliessende Gewichtung der verschiedenen Erkrankungen und vorzeitigen Todesfälle erfolgte mit Hilfe der ***Disability Adjusted Life Years*** (DALYs), eine von der Weltbank und der Weltgesundheitsorganisation WHO verwendete Methode (Murray ***et al.*** 1996a/b). Die neue Ökobilanzbewertungsmethode EcoIndicator'98 (Goedkoop ***et al.*** 1998) übernimmt diese Methodik und wird bzgl. Schädigung der menschlichen Gesundheit mindestens folgende Elemente enthalten:

- Potenzielle Schäden in DALYs pro kg Emission ins Wasser oder in die Luft werden für 55 karzinogene Schadstoffe berechnet (Hofstetter 1998:195ff). In den meisten Fällen konnte die Effektinformation aus IRIS (1996) übernommen werden. Für die IARC Gruppen 1 und 2A (hohe Evidenz) liegen diesen Angaben meist epidemiologische Studien von Arbeitsplätzen zu Grunde. Toxikologische Studien ergänzen hier die Datenbasis.
- Die Zusammenstellung von epidemiologischen Studien zu respiratorischen Effekten von Pilkington ***et al.*** (1997) bildete die Grundlage für die Abschätzung von über 120 Schadensfaktoren für direkt oder indirekt respiratorisch wirksame Schadstoffe (Hofstetter 1998:289ff). mehr als 100 dieser Schadstoffe tragen zur Ozonbildung in der Troposphäre bei und wurden mit Hilfe des Modells von Jenkin et ***al.*** (1997) berücksichtigt. Im Gegensatz zu GVF (1996), wo alle Effekte der Luftbelastung der Indikatorgrösse 'Partikelexposition' zugerechnet wurden, werden in Pilkington ***et al.*** (1997) verschiedene Studienresultate für diverse Schadstoffe kombiniert.
- Studien an den Überlebenden der Atombombenabwürfe von Hiroshima und Nagasaki erlauben die Quantifizierung von Gesundheitseffekten aufgrund von niedrigen Dosen ionisierender Strahlung (ICRP 1990, UNSCEAR 1993). Frischknecht ***et al.*** (1999) berechnet hiervon mit Hilfe von ExternE (1995) für rund 30 Radionuklide Schadensfaktoren für Emissionen in die Luft und ins Wasser.
- Auch Teilaspekte der erwarteten Gesundheitsschäden aufgrund des Klimawandels lassen sich aufgrund epidemiologischer Studien über Wärme- und Kältestress quantifizieren (Kalkstein ***et al.***

1997, The Eurowinter Group 1997, Martens ***et al.*** 1998). In Hofstetter (1999) werden basierend auf Tol (1999) solche Schadensfaktoren für über 30 Treibhausgase berechnet.

Diese Zusammenstellung soll zeigen, dass vorhandene epidemiologische Studien durchaus schon heute die Quantifizierung zahlreicher Gesundheitseffekte für eine Vielzahl von Schadstoffen erlaubt. Weitere Schadensfaktoren für ozonschichtabbauende Stoffe und einige gut untersuchte Umweltschadstoffe (z.B. Blei) liessen sich mit beschränktem Aufwand hinzufügen.

Und trotzdem zeigt diese Zusammenstellung deutlich, dass viele Effekte unberücksichtigt bleiben und für einige der in Ökobilanzen relevanten Chemikalien keine Bewertung vorgenommen werden kann. Aus diesem Grund wird hier eine Kombination des epidemiologischen und toxikologischen Ansatzes vorgeschlagen.

6. Vorschlag zur Kombination von toxikologischen und epidemiologischen Erkenntnissen

Diese Ausgangslage und die Forderung, dass informierte Entscheidungen wenn immer möglich auf dem gesamten relevanten Wissen basieren sollten, legen es nahe, dass epidemiologische ***und*** toxikologische Techniken zu einer Methode zusammengefügt werden. Das Ziel ist dabei, die Stärken der beiden Ansätze zu vereinen. Es sollen also weiterhin direkte Gesundheitsbeeinträchtigungen anvisiert und unter so realen Umständen wie möglich untersucht werden, aber entsprechende Zusammenhänge sollen für mehr als nur Leitindikatoren zur Verfügung gestellt werden.

Das hier beschriebene Konzept wurde in Hofstetter (1998:292ff) als Alternative vorgeschlagen, aber dort nicht weiterverfolgt und operationalisiert. Die tatsächliche Machbarkeit muss deshalb noch gezeigt werden.

Am Beispiel respiratorischer Effekte soll das Konzept im folgenden dargelegt werden. Aus der toxikologischen Literatur zur Arbeitsplatzbelastung sind Angaben zu Konzentrationen bekannt, die einen bestimmten Gesundheitseffekt hervorrufen. Tabelle 1 listet diese Informationen für die respiratorischen Effekte auf, wie sie in Schweden bestimmt wurden (Hansson 1997). Diese Zusammenstellung war das Ergebnis einer Untersuchung an 278 Substanzen, die von der ***Swedish Criteria Group*** für die Festlegung maximaler Arbeitsplatzkonzentrationen reviewt wurden. In Anonymous (1994) sind alle untersuchten Substanzen aufgelistet und die jeweilige Zeitschriftenausgabe von ***Arbete och Hälsa*** mit den entsprechenden Resultaten referenziert.

Zusätzlich zu den 15 in Tabelle 1 erwähnten Substanzen/-gemischen wurden weitere 6 Substanzen bisher ohne Angabe von Effektschwellenwerten dieser Gruppe zugeteilt. Die Zusammenstellung ist zudem insofern unvollständig, dass nur 278 Substanzen bis 1996 untersucht wurden und jeweils nur derjenige Effekt einer Substanz ausgewertet wurde, welcher bei der tiefsten Konzentration sichtbar wird. Eine Ausweitung der untersuchten Substanzen und die Auswertung der Schwellenwerte für alle

Effekte (wenn möglich sogar innerhalb der Kategorie der respiratorischen Effekte) ist daher eine Vorbedingung für einen soliden Datensatz.

Substanz	Evaluationsjahr	No-Effect Level (NEL)	Effect Level (EL)
Aluminium	1982	0.1-2.7 mg/m^3	
Cobalt	1983	0.03 mg/m^3	
Piperazine	1985	0.3 mg/m^3	
Vanadium	1983		0.06 mg/m^3
Sulphur dioxide	1985		1 ppm (2.7 mg/m^3)
Coal dust	1986		2 mg/m^3
Nitrogen dioxide	1986		0.5 mg/m^3
Cotton dust	1986		0.3-0.5 mg/m^3
Terpenes	1987		125 mg/m^3
Ozone	1987		0.24 mg/m^3
Diisocyanates and polyisocyanates	1988		1 ppb
Titanium dioxide	1989		0.2-2.8 mg/m^3
Paper dust	1990		<5 mg/m^3
Attapulgite	1991		2.7 mg/m^3
Talc, asbestos-free and with low quartz content	1992		1 mg/m^3

Tab. 1: No-Effect und Effect Levels von Substanzen dessen kritische Effekte das respiratorische System betreffen (Hansson 1997)

Der hier gewählte Grundansatz entspricht dem in Hofstetter (1998:311) eingeführten ***Umbrella-Principle***. Die vorhandenen epidemiologischen Studien für Partikel, Ozon, SO2, NO2 und CO bilden den Haltegriff während alle weiteren Substanzen, welche im toxikologischen Versuch ebenfalls respiratorische Effekte zeigen, den Schirm bilden.

Unter der Voraussetzung, dass die in Tabelle 1 ausgewiesenen Effektkonzentrationen gute Stellvertreter für die relative Potenz, respiratorische Schäden hervorzurufen, sind, dass diese Substanzen im Gemisch also synergistisch wirken und gerade proportional ihrer Effektschwellenwerte hierzu beitragen, kann folgende Allokation der beobachteten Gesundheitsschäden vorgenommen werden:

Gesundheitseffekte ***g*** der Substanz ***i*** per kg =

$$\frac{1}{\text{totale Emission von i}} \cdot \text{totaler Gesundheitseffekt g} \cdot \frac{\frac{\text{Konzentration von i}}{\text{effect level g von i}}}{\sum_i \frac{\text{Konzentration von i}}{\text{effect level g von i}}} \qquad (2)$$

wobei sich ***total*** immer auf dieselbe Studienregion bezieht, ***Emission*** steht hier für die Luftemissionen, welche für die gemessenen Konzentrationen im Studiengebiet verantwortlich sind (Advektion zu berücksichtigen!), bei den ***Konzentrationen*** handelt es sich um inhalierte bodennahe Luft (allenfalls Indoor/Outdoor korrigiert), ***i*** steht für jene Substanzen, welche den Gesundheitseffekt ***g*** zeigen und schliesslich ***effect level*** entspricht den in Tabelle 1 beispielshaft aufgelisteten Werten.

Sind, wie in diesem Beispiel, aus epidemiologischen Studien Effektschätzer für verschiedene Substanzen bereits vorhanden, sollen die relativen Verhältnisse dieser Effektschätzer anstatt der viel spekulativeren Intrapolation von Effektschwellenwerten verwendet werden (Abb. 2).

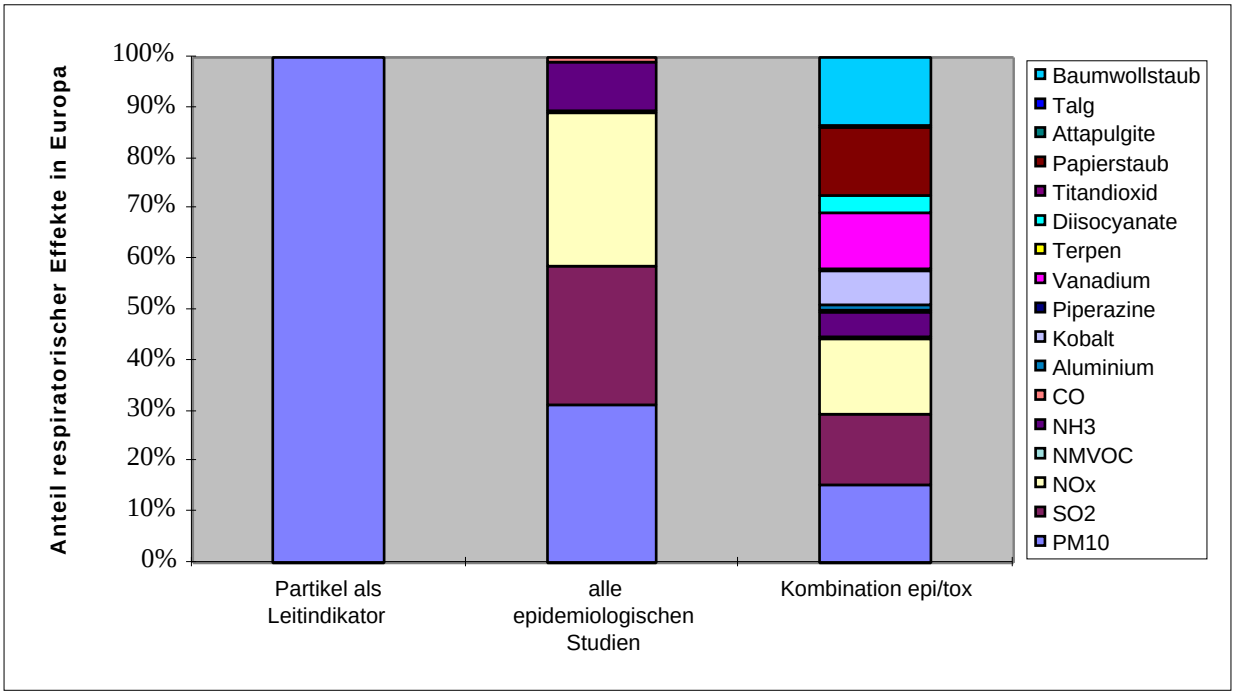

Abb. 2: Illustration der drei möglichen Allokationsmethoden zwischen den beobachteten respiratorischen Effekten aufgrund der Luftbelastung (100% = ca. 9000 DALYs/Mio.Einwohner und Jahr, vgl. Hofstetter (1998:342)) und einzelnen Schadstoffen. Links: Partikel als Leitindikator wie vorgeschlagen in GVF (1996); Mitte: Zurechnung der Gesamtschäden auf die in epidemiologischen Luftverschmutzungs-Studien berücksichtigten Schadstoffe (Hofstetter 1998); Rechts: Fiktives Beispiel für das hier präsentierte Konzept, welches toxikologische und epidemiologische Erkenntnisse kombiniert.

Kritische LeserInnen mögen nun einwenden, dass der Ansatz wiederum auf toxikologische Daten zurückgreift und hiermit gegen die Kritik in Abschnitt 1 kein Gegenrezept liefert. Dieser Kritik sei entgegnet,

- dass bewusst Effektschwellenwerte gewählt werden, bei denen eine gewisse Anzahl von Individuen den beschriebenen Effekt zeigen,
- dass die in Tabelle 1 aufgelisteten Konzentrationen oftmals nicht oder nicht alleine auf Tierversuchen basieren und damit die Übertragung auf den Menschen einfacher ist und
- dass diese Schwellenwerte lediglich verwendet werden, um die real beobachteten Gesundheits-effekte spezifischen Verursachern zuzuordnen und damit durchaus potenzielle Schäden abgeschätzt werden können.

Aber selbstverständlich hat auch dieses Konzept wichtige Schwachstellen. Die Voraussetzungen, dass dieser Ansatz Sinn macht, sind:

- die Vorbedingung, dass Effektschwellenwerte gute Stellvertreter sind für die relative Wirkung in einem nicht vordefinierten Schadstoffcocktail, tatsächlich zutrifft (zu testen)
- für eine Vielzahl von Substanzen Effektschwellenwerte für verschiedene Effekte vorhanden sind, und
- die relevanten bodennahen Umgebungskonzentrationen aller Substanzen die zu einem Effekt beitragen entweder gemessen werden oder zuverlässig berechnet werden können.

Diese Bedingungen sind heute nur teilweise oder nicht erfüllt und behindern daher eine rasche Umsetzung des vorgeschlagenen Konzeptes. Trotz dieser noch beschränkten Praktikabilität würde dieses Konzept auch die Epidemiologie weiterbringen, könnten doch epidemiologische Studien neu nicht nur nach einem Leitindikator ausgewertet werden, sondern in Konkurrenz auch dieser Summenindikator in die multivariate Faktorenanalyse aufgenommen werden.

Eine wichtige Einschränkung der Praktikabilität des vorgestellten Konzeptes ist die Erweiterbarkeit auf die Schadstoffaufnahme via Trinkwasser und Nahrung. Gerade der letztere Aufnahmepfad ist äusserst wichtig für persistente Stoffe und in epidemiologischen Studien besonders schwer fassbar. Möglicherweise müssen hier Analogien durch Zusatzwissen über die interne Exposition und den Metabolismus im Körper gesucht werden.

7. Schlussfolgerungen

Die heutige Praxis in der Bewertung von Gesundheitsrisiken von Schadstoffen in Ökobilanzen entspricht noch meist einer Extrapolation von 'Nicht-Risiken', d.h., dass Informationen über (toxikologisch) sichere Konzentrationen verwendet werden, um Aussagen über potenzielle Risiken zu machen. Dieses Vorgehen ist nicht nur in der Sache selbst fraglich und revisionsbedürftig, sondern stellt auch grosse Probleme, wenn solche Phantomindikatoren mit realen potenziellen Schädigungen der menschlichen Gesundheit durch andere Umweltauswirkungen, z.B. erhöhte UV-Strahlung durch den Ozonschichtabbau, verglichen werden müssen.

Epidemiologische Studien zur Effektabschätzung leisten gute Dienste, wenn es darum geht, reale und spezifische Gesundheitsveränderungen den Unterschieden in den Cocktails von Umweltschadstoffen zuzuordnen. Es wurde gezeigt, dass bereits für eine beachtliche Zahl von Wirkungspfaden potenzielle Schäden pro kg Emission beitragender Schadstoffe ermittelt werden konnten. Die Verwendung weiterer Original- und Metastudien sollen diese Datenbasis weiter verbreitern und abstützen.

Da einerseits die oft ubiquitäre Verteilung von Schadstoffen und die globale Industrialisierung das Vorhandensein geeigneter Kontrollgruppen stark einschränkt und andererseits sehr kleine Effekte aufgrund tiefer Umweltkonzentrationen gezeigt werden sollen, führen umweltepidemiologische Studien nur bei grosser Studienpopulation zu signifikanten Resultaten und können nur wenige der vermuteten Wirkungszusammenhänge untersucht werden. Die Allokation beobachteter Effekte auf einzelne Schadstoffe ist zudem meist nicht möglich.

Der vorgängige Abschnitt 6 stellt ein Konzept vor, welches die Kombination von Resultaten epidemiologischer Studien und Effektschwellenwerten aus der Arbeitsplatzhygiene präsentiert. Die neu berechneten Indikatoren pro Gesundheitseffekt können wiederum in epidemiologischen Studien benützt werden, um die beobachteten Schadstoffcocktails besser zu charakterisieren.

Das vorgestellte Konzept soll als Diskussionsbeitrag verstanden werden. Seine Praktikabilität muss mit weiteren Arbeiten noch gezeigt werden. Die Erweiterbarkeit auf Expositionen via Trinkwasser und Nahrungsmittel erscheint zwar möglich, ist aber momentan nicht realistisch.

Bis zur Operationalisierung dieses Konzeptes wird vorgeschlagen, die Informationen aus epidemiologischen Studien für die Ökobilanzbewertung ausgiebig zu nutzen und spezifische Gesundheitsgefährdungen durch nichtabgedeckte Einzelsubstanzen fallspezifisch einer Risikoanalyse zu unterziehen.

Verdankung

Ich danke Thomas Mettier (UNS-ETH) für die Durchsicht eines Entwurfes dieses Manuskripts und dem Schwerpunkt Programm Umwelt (Schweizerischer Nationalfonds) für die Finanzierung des Projektes 'Environmental Prioritizing', im Rahmen dessen dieser Beitrag entstanden ist.

Literatur

Anonymous 1994. Concensus Reports in Previous Volumes, ***Arbete och Hälsa*** 30: 68-72 (1994)

Basler & Hofmann 1974. Basler & Hofmann Ingenieure und Planer, ***Studie Umwelt und Volkswirtschaft, Vergleich der Umweltbelastung von Behältern aus PVC, Glas, Blech und Karton***, Eidgenössisches Amt für Umweltschutz (BUS), Bern 1974

BUWAL 1984. BUWAL (Hrsg.), ***Oekobilanzen von Packstoffen***, Schriftenreihe Umweltschutz Nr.24, Bern 1984

BUWAL 1991. Habersatter K., Widmer F., ***Ökobilanz von Packstoffen - Stand 1990***, BUWAL-Schriftenreihe Umwelt Nr. 132, Bern 1991

ESEERCO 1995. Empire State Electric Energy Research Corporation (ESEERCO), ***New York State Environmetal Externalities Cost Study, Volume 1: Introduction and Methods***, Oceana Publications Inc., New York 1995

ExternE 1995. ExternE, ***Externalities of Energy***, European Commission EUR 16520 EN, Volume 1-6, Luxembourg 1995

ExternE 1997. ExternE Core Project, ***Extension of the Accounting Framework; Final Report***, compiled by Mayerhofer P., Krewitt W., Friedrich R., The European Commission, Contract No. JOS3-CT95-0002, Stuttgart 1997 (available on http://www.ExternE.jrc.es)

Frischknecht ***et al.*** 1999. R. Frischknecht, A. Braunschweig, P. Hofstetter, P. Suter, Human Health Effects of Ionising Radiation in Life Cycle Impact Assessment, in preparation

Gebler 1992. Gebler W., ***Ökobilanzen in der Abfallwirtschaft***, Stuttgarter Berichte zur Abfallwirtschaft, Bericht 41, 2. Auflage, Bielefeld 1992

Goedkoop 1995. Goedkoop M., ***The Eco-indicator 95, Final Report and Manual for Designers***, Amersfoort 1995

Goedkoop ***et al.*** 1998. Goedkoop, M., P. Hofstetter, R. Müller-Wenk R. and R. Spriensma. 1998. The Eco-Indicator'98 Explained, ***International Journal on Life Cycle Assessment*** 6 (3) 352-360 (1998)

Guinée ***et al.*** 1993. Guinée J., Heijungs R., A proposal for the Classification of Toxic Substances within the Framework of Life Cycle Assessment of Products, ***Chemosphere*** 26 (10): 1925-44 (1993)

Guinée ***et al.*** 1996. Guinée J., Heijungs R., van Oers L., van de Meent D., Vermeire Th., Rikken M., ***LCA Impact Assessment of Toxic Releases***, CML/RIVM, VROM The Hague 1996

GVF 1996. GVF, ***Monetarisierung der verkehrsbedingten externen Gesundheitskosten, Synthesebericht***, GVF-Auftrag Nr.272, GS EVED, 1996

Hansson 1997. Hansson S.O., Critical Effects and Exposure Limits, ***Risk Analysis*** 17 (2): 227-36 (1997)

Heijungs ***et al.*** 1992. Heijungs R., Guinée J.B., Huppes G., Lankreijer R.M., Udo de Haes H.A., Wegener Sleeswijk A., Ansems A.M.M., Eggels P.G., van Diun R., de Goede H.P., ***Environmental Life Cycle Assessment of Products; Backgrounds & Guide***, Leiden 1992

Hofstetter 1991. Hofstetter P., Bewertungsmodelle für Ökobilanzen in ***Tagungsband Energie- und Schadstoffbilanzen im Bauwesen***, 7.3.91 an der ETH Zürich (Bestellung HBT, ETH-Hönggerberg, 8093 Zürich)

Hofstetter 1993. Hofstetter P., ***Weighing of Different Environmental Problems***, paper presented at the SETAC/UNEP-Workshop, held in Amsterdam 9-10 June 93

Hofstetter 1998. Hofstetter, P. 1998. ***Perspectives in Life Cycle Impact Assessment; A structured approach to combine models of the technosphere, ecosphere , and valuesphere***, Boston: Kluwer Academic Publishers

Hofstetter 1999. Hofstetter P., Damages to Human Health due to Climate Change, Draft March 1999

Huijbregts 1999. Huijbregts M.A.J., Priority Assessment of Toxic Substances in LCA; Application of the Uniform System for the Evaluation of Substances 2.0, Draft, IDES, Amsterdam 1999

ICRP 1990. Internatinal Commission on Radiological Protection, 1990, Reccomendations of the Internatinal Commission on Radiological Protection, Publication 60, ***Annals of the ICRP, 21 (1-3)***

IER 1997. IER (ed.), ISET, ZEW, DIW, ***ExternE National Implementation Germany***, Contracts JOS3-CT95-0010, JOU2-CT-0264, EC, Draft Final Report, Stuttgart 1997

IRIS 1996. U.S. EPA, Office of Health and Environmental Assessment, ***Integrated Risk Information System (IRIS)*** (on-line data base http://www.epa.gov/ngispgm3/iris/), Cincinnati October 1996

Jansen ***et al.*** 1972. Jansen P., Jordan S, Schikarski W., ***Vergleichende Modelltheorie der atmosphärischen Schadstoffbelastung durch Kernkraftwerke***, Vortrag gehalten am 10. Colloque IRCHA sur les atmosphères polluées, 3.-5. Mai 1972 in Paris, zitiert in Unger H. Atomkraftwerke. Eine umweltfreundliche Energieerzeugung in Alt C., Weber F. (Hrsg.) ***Reinhaltung der Luft***, Karlsruhe 1973

Jenkin ***et al.*** 1997. Jenkin M.E., Hayman G.D., Derwnt R.D., Saunders S.M., Pilling M.J., ***Tropospheric Chemistry Modelling: Improvements to Current Models and Application to Policy Issues***, AEA/RAMP/20150/R001 Issue 1, AEA Culham 1997

Jenkins ***et al.*** 1996. Jenkins S., Hay D., Air Poillution, Health and Exercise: a Review, ***Energy & Environment*** 7 (1): 51-5 (1996)

Jolliet 1993. Jolliet O., Ökobilanz thermischer, mechanischer und chemischer Kartoffelkrautbeseitigung, ***Landwirtschaft Schweiz*** 6 (11-12): 675-82 (1993)

Jolliet 1996. Jolliet O. (ed), Impact Assessment of Human and Eco-Toxicity in Life Cycle Assessment, in, Udo de Haes (ed.), ***Towards a Methodology for Life Cycle Impact Assessment***, SETAC-Europe Working Group on Impact Assessment, Brussels 1996

Jolliet ***et al.*** 1997. Jolliet O., Crettaz P., Fate Coefficients for the Toxicity Assessment of Air Pollutants, Int J LCA 2 (2): 104-10 (1997)

Kalkstein ***et al.*** 1997. Kalkstein L.S., Greene J.S. (1997), An evaluation of cliamte/mortality relationship in large U.S. cities and the possible impacts of a climate change, Environmental health Perspectives, Vol. 105, No.1, January 1997, 84-93

Lutz 1997. LutzW., Krebsrisiko, in Cercl'Air (Hrsg.), Kanzerogene Luftschadstoffe, Übersicht zur Fachtagung vom 30./31.1.1997, Bern

Martens ***et al.*** 1998. Martens, W.J.M., (1998) Climate Change, Thermal Stress and Mortality Changes, Social Science and Medicine, Vol. 46, No. 3, 331-344

Murray ***et al.*** 1996a. Murray Ch.J.L., Lopez A.D. (Eds.), The Global Burden of Disease, ***Volume I of Global Burden of Disease and Injury Series***, WHO / Harvad School of Public Health / World Bank, Harvard University Press, Boston 1996

Murray ***et al.*** 1996b. Murray Ch.J.L., Lopez A.D., Global Health Statistics, ***Volume II of Global Burden of Disease and Injury Series***, WHO/Harvard School of Public Health/World Bank, Harvard University Press, Boston 1996

Pilkington ***et al.*** 1997. Pilkington A., Hurley F., Donnan P., ***Health Effects in ExternE Transport: Assessment and Exposure-Response Functions, Institute of Occupational Medicine***, Edinburgh, Draft July 1997

Schaltegger ***et al.*** 1992 Schaltegger St., Sturm A., ***Ökologieorientierte Entscheidungen in Unternehmen***, Schriftenreihe des Instituts für Betriebswirtschaft, WWZ Basel, Bern 1992

Steen ***et al.*** 1992. Steen B., Ryding S.-O., Swedish Environmental Research Institute, Federation of Swedish Industries, ***The EPS Enviro-accounting Method***, Göteborg 1992

Thalmann-Graf 1991. Thalmann-Graf W.R., ***Ökobilanzen im Verpackungsbereich***, European Packaging Federation, Berlin 1991

The Eurowinter Group 1997. The Eurowinter Group (1997), Cold exposure and winter mortality from ischaematic heart disease, cerebrovascular disease, respiratory disease, and all causes in warm and cold regions of Europe, The Lancet, Vol. 349, May 10, 1341-1346

Tol 1999. Tol R.S.J. (1999), `The Marginal Costs of Greenhouse Gas Emissions', Energy Journal, 20 (1), 61-81.

UNSCEAR 1993. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, ed.), 1993. ***Sources and Effects of Ionizing Radiation; UNSCEAR 1993 Report to the General Assembly, with Scientific Annexes***, United Nations, New York

Wenzel ***et al.*** 1997. Wenzel H., Hauschild M., Alting L. (eds.), ***Environmental Assessment of Industrial Products***, Institute for Product Development, Technical University of Denmark, Chapman & Hall 1997

Anhang A. Referentinnen und Referenten des 10. Diskussionsforums Ökobilanzen

Dr. Martin Scheringer, Studium der Chemie und theoretischen Physik an derJohannes-Gutenberg Universität Mainz; 1990 Abschluß als Diplom-Chemiker. 1996 Promotion an der Abteilung für Umweltnaturwissenschaften der ETH Zürich, seit 1996 wissenschaftlicher Mitarbeiter in der Gruppe für Sicherheit und Umweltschutz in der Chemie am Laboratorium für technische Chemie der ETH Zürich. Forschungsprojekte im Bereich Umweltchemie, Chemikalienbewertung und Oekobilanzen von chemischen Produkten. Buchpublikation "Persistenz und Reichweite von Umweltchemikalien", Wiley-VCH 1999.

Anneke Wegener Sleeswijk graduated as a biologist in 1987. Since 1991, she is working in the field of environmental science at the Centre of Environmental Science of Leiden Universty (CML). Between 1991 and 1995, she worked on several projects concerning methodological issues as well as case studies. She specialized in the subject of methodological aspects of LCA of agricultural products. Since 1995, she is working on a PhD subject concerning environmental fate modelling of substances for the purpose of LCA.

Dr. Monika Herrchen graduated as a chemist in 1981 and obtained her PhD degree in biochemistry in 1983. From 1983 to 1985 she passed a postdoctoral fellowship in biochemistry at the University of Bristol, England. Since 1985, she has been working at Fraunhofer-Institute for Environmental Chemistry and Ecotoxicology (Fh-IUCT) at Schmallenberg: Between 1985 and 1994, she was a research scientist at the departments of water and soil protection (7 years) and of applied ecology (2 years). Since 1994 she is head of the Department of Environmental Information and Assessment Systems. Her actual research fields are hazard and risk assessment of industrial chemicals and pesticides (desk work), impact assessment for technical products, processes and industrial sites, expertises, and scientific management. She is member of several national and international scientific committees on LCA and chemical assessment.

Guntram Koller wurde am 4. September 1970 in Linz, Österreich geboren. Im Juni 1996 schloss er das Studium der Technischen Chemie – Studienzweig Chemieingenieurwesen an der Technischen Hochschule in Graz mit Auszeichnung ab. Seit Oktober 1996 arbeitet er an seiner Doktorarbeit bei Professor Konrad Hungerbühler in der Gruppe für Sicherheit und Umweltschutz, ETH Zürich. In diesem Rahmen beschäftigt er sich mit der Bewertung von Gesundheit-, Sicherheit und Umweltschutzaspekten während den frühen Phasen der Entwicklung industrieller chemischer Prozesse.

Manuele Margni graduated as a rural engineer with environmental specialisation in 1996. From 1996 to 1997 he attended a European master course in environmental engineering and management where he dealt with tools like LCA, eco-audits and risk analysis as well as with environmental politics and decision making. In summer 1997 he developed a life cycle analysis method to evaluate pesticides' impact on human health and ecosystems for "srva" (service romand de vulgarisation agricole) at Lausanne. 1998 he worked in the domain of environmental and safety management (audits, pre-audit, organisation of emergency cases) for several enterprises. At present he is elaborating an integrated quality, environment and safety management system for an ISO 9002, ISO 14001 and SCC (Security Certificate for Contractors) certification in a fuel and oil commercialising company, Benoil SA, Rancate.

Dr. Patrick Hofstetter schloss 1989 das Studium zum Dipl. Maschineningenieur ETH ab. Seit 1989 ist er selbständiger Berater in Energie- und Umweltfragen (Büro für Analyse & Ökologie). Seit 1990 ist er teilzeitlicher wissenschaftlicher Mitarbeiter an der ETH Zürich (seit 1.7.97 bei Umweltnatur- und Umweltsozialwissenschaften); Forschung im Bereich der Ökoinventar- und Bewertungsmethodik für Ökobilanzen, Sommer 1998 Dissertation mit dem Titel "Perspectives in Life Cycle Impact Assessment; A structured approach to combine models of the technosphere, ecosphere, and valuesphere".

Anhang B. Tagungsprogramm

Programm des 10. Diskussionsforums an der ETH Zürich[2]

Chemikalienbewertung in Ökobilanzen

Beginn um 9:45, am 28. April 1999

Ort: Auditorium Maximum (Hauptgebäude F 30, Rämistrasse 101, ETH-Zentrum)

ab 9:45	Kaffee/Tee und Gipfeli
10:10	Begrüssung (Stephanie Mössner, Martin Scheringer)
Teil 1	
10:15	**Das Umweltverhalten von Stoffen - eine Lücke im LCIA**
	Martin Scheringer, Safety and Environmental Technology Group, Laboratory of Chemical Engineering, ETH, Zurich, Switzerland
11:00	**GLOBOX: an add-on LCA multimedia characterization model including sea compartments**
	Anneke Wegener Sleeswijk, Centre of Environmental Science Leiden University (CML), Leiden, The Netherlands
11:45	**Möglichkeiten und Notwendigkeiten der Verknüpfung von Elementen der Risiko-abschätzung und Produkt-LCA: eine kontroverse Diskussion**
	Monika Herrchen, Fraunhofer-Institut für Umweltchemie und Ökotoxikologie, Schmallenberg, Germany
12:30	Mittagessen
Teil 2	
13:45	**Schwankungsbereiche aquatischer Toxizitätsdaten - Schlussfolgerungen für die Risikoanalyse von Chemikalien**
	Guntram Koller, Safety and Environmental Technology Group, Laboratory of Chemical Engineering, ETH, Zurich, Switzerland
14:30	**Life cycle impact assessment of pesticides on human health and ecosystems**
	Manuele Margni, Institute of soil and water, EPFL, Lausanne, Switzerland
15:15	**Von der Extrapolation von Nicht-Risiken zur Abschätzung potenzieller Schäden**
	P. Hofstetter, Environmental Sciences: Natural and Social Science Interface (UNS), ETH, Zurich, Switzerland
16:00	Schlusswort

[2] Die Diskussionsforen 'Ökobilanzen' sind Bestandteil des Schwerpunktprogramms Umwelt des Schweizerischen Nationalfonds und werden durch diesen finanziell unterstützt.