Journal of Hazardous Materials, 18 (1988) 1–16
Elsevier Science Publishers B.V., Amsterdam — Printed in The Netherlands

ASSESSING ECOSYSTEM IMPACTS FROM SIMULANT AND DECONTAMINANT USE

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(Received May 13, 1987; accepted in revised form September 30, 1987)

Summary

Environmental effects from chemical release depend on factors which include the routes and rates of release, the modes of dissemination and degradation, and toxicities to local organisms. Data used for ecosystem risk characterization varies in quality and quantity, making it difficult to detemine the relative ecohazards of compounds. The problem addressed in this paper is: "Given particular chemicals, use scenarios and natural communities, how can the toxicological effects of future chemical use be projected using available data?" A method which uses available qualitative and quantitative data on physical and chemical properties, toxicity, and usage was devised to rank the ecological hazard of compounds. The ecohazard of a compound is determined using a decision key and a risk rank matrix which can initially use qualitative data. We regard the risk matrix as an algorithm for quantitatively expressing ecosystem risk. The risk characterization is refined by incorporating quantitative data in successive iterations through the process. The method was used to rank the anticipated ecosystem risk for over 40 chemicals used in military training.

Introduction

Chemicals may enter ecosystems through air, water, and soil routes. Once released, contaminants may be assimilated by individual organisms [1], transformed among biotic connections [2], and dispersed and magnified depending on ecosystem characteristics. Assessing the environmental effects of chemical release requires different levels of sophistication and integration depending on

0304-3894/88/$03.50

the objective of the assessment. If the objective is assessment or prediction of the response of an ecosystem to contamination, integrated testing [1,2] following carefully developed procedures is required. The outstanding characteristic of toxicological assessments in ecosystems is the need to evaluate subtle, complex effects by toxicologists. Responses suitable for the assessment of ecosystem damage are difficult to define clearly and unambiguously. Measures that are available often have poor precision (high measurement variance) and unknown accuracy. While many health related responses (e.g. acute, chronic and reproductive toxicity) are components of ecosystem effects (abundance and biomass, population structure), alone they are not predictive of ecosystem level endpoints. System complexity and data scarcity therefore limit the toxicologist's ability to predict the magnitude and significance of toxic effects in ecosystems.

The study of toxicological response of ecosystems to environmental exposures of contaminants is termed "ecoepidemiology" [3]. Ecoepidemiological studies are concerned with describing effects, identifying causes, and determining links and pathways between populations, communities and ecosystems. Ecoepidemiological analysis uses many types of test systems [1,4] which are integrated with other data to provide an assessment of the expected damage to the ecological system.

Developing a probabilistic estimate of the effect of chemical release on an ecosystem is an ecological risk assessment. We define the elements in an ecosystem risk assessment (hazard identification, exposure assessment, exposure–response assessment, risk characterization, risk management) analogously to those for humans [5]. Although the importance of a single species to an ecosystem may be mitigated by homeostatic processes in the ecosystem [6], a reasonable starting point for risk assessment is identification of toxicological effects having a high probability of causing appreciable acute or chronic toxicity [7–12] to at least one major ecological system component [1,2,13].

Rank matrix for assessing ecological risk

Our ecological risk assessment makes several assumptions which are commonly accepted by toxicologists. First, most complex toxicological responses arise through a series of identifiable (if unknown) steps. Some of these steps are highly predictive of the overall toxicological response. As a simple example, blood lead concentrations are predictive of lead-induced loss of neurological function or decreased population growth due to toxicity may reduce ecosystem productivity. Second, a response in one species often indicates similar responses in other species. If a compound is acutely toxic to laboratory mammals, we assume that it is also acutely toxic to mammals in the ecological system of concern. In general, it is not possible to project across broader taxonomic boundaries from standard toxicity data. Third, the ability to predict toxic re-

TABLE 1

Risk rank assignment matrix[a]

Toxicity	Low exposure			Medium exposure			High exposure		
	Low EBCA	Medium EBCA	High EBCA	Low EBCA	Medium EBCA	High EBCA	Low EBCA	Medium EBCA	High EBCA
Low	1	2	3	2	4	5	3	5	7
Medium	2	4	5	4	6	8	5	8	9
High	3	5	7	5	8	9	7	9	10

[a]Body of table gives the risk rank corresponding to the product of the toxicity, exposure and EBCA scores:

Product of scores	1	2	3	4	6	8	9	12	18	27
Rank	1	2	3	4	5	6	7	8	9	10.

sponses improves as the number of species exhibiting similar toxic responses to a given chemical increases. For example, a chemical which is a carcinogen in mice, rats, rabbits, and chimpanzees is more likely to be a carcinogen in man than is a chemical which is carcinogenic only in male rats. Fourth, no prediction is perfect since unusual species differences occur. The teratogenicity of thalidomide in humans, but not in other mammals, is an example. Fifth, in the absence of experimental data, the toxicity of mixtures is predicted best by dose additivity.

Data used for ecosystem risk characterization varies in quality and quantity, making it difficult to determine the relative ecohazards of compounds. The risk-rank matrix described here is not limited by the availability of quantitative data. The ecohazard of a compound is determined using a decision key which can initially use thoughtful subjective data. Successive stages of risk characterization refinement use data from models and test batteries. We regard the risk matrix as an algorithm for qualitatively determining a level of ecosystem risk based on the best information available.

Our evaluation of ecosystem response to chemical release depends primarily on the toxicity of the compound(s), the exposure level(s), and the areal extent of contamination which we term the "effective biological contact area" (EBCA). In our scheme, each of these can be "low", "medium" or "high", or numerically, 1, 2 or 3. The product of the three scores is a relative measure of risk to the ecological system (Table 1). (Similar reasoning has led to the equally crude 'MITRE' ranking system for hazardous waste sites [14]).

Toxicity

The traditional concern of hazard identification for acute toxicity has led to several assessment approaches [14–19]. Usually, one-dose acute toxicity data (mg/kg) for a species (D_{MAMMAL}) of mass W_{MAMMAL} (kg) [20] is expressed

(eqn. 1) as exposure to a 70 kg human (D_{HUMAN}). In our scheme, the estimated dose for *any* mammalian target species, when expressed in human equivalents, is scored [2, 21]: 1(Low) > 15,000 mg/kg; 2 (Medium) ≥ 500–15,000 mg/kg; 3 (High) < 500 mg/kg. Alternatively, a hazard-equivalent exposure for a target species can be estimated from an acute toxicity value for a laboratory species by comparing the acute value with environmental levels estimated from models [22–24]. If the expected environmental levels exceed D_{HUMAN}, a score of 3 is assigned, otherwise the score is 2 or 1, as appropriate. Algorithms for interspecies conversion of acute toxicity can be used for compounds which are not cholinesterase inhibitors [25].

$$D_{HUMAN} = D_{ANIMAL} \ (W_{ANIMAL}/70)^{1/4} \quad (1)$$

Other types of toxicity may modify the acute toxicity scores. For example, if the compound is mutagenic, teratogenic, tumorigenic, phytotoxic, or ecotoxic, the score derived from acute toxicity data is increased one unit. Alternatively, we have used data for the distribution of doubling doses (μg/plate) [26] in the Ames assay [27] to devise hazard scores for mutagenicity (Table 2) analogous to those for acute toxicity [21]. If the estimated environmental load of the mutagen(s) [28,29] exceeds the estimated doubling dose, the score is 3.

Exposure–response

Studies of processes in an ecosystem show that the numbers of pathways and possible target species increase with area [30]. Hence, the probability that release will produce an ecologically significant effect increases as the effective biological contact area (EBCA) increases. Proposed EBCA scores are given in Table 2.

Exposure

For an ecosystem, exposure is the quantity of the toxic substance in a specified volume of the ecosystem; this may incorporate water, air or soil. Exposure assessment for ecological systems is difficult because quantification of exposure must consider the quantity of product used, the concentrations of toxic compounds in the product, the area or volume the toxic material is distributed in, and persistence. Exposure (mg/m^2) is scored here as a multiple of the human-equivalent acute toxicity (mg/kg) if the compound does not bioaccumulate or the chronic toxicity if it does (Table 2).

Risk characterization

The relative risk for a compound is the product of the three scores (Table 1). For example, an ecosystem exposed to a highly toxic compound used at low exposures over a medium EBCA, or to a low toxicity compound used at a moderate level over a large EBCA, is at the same risk (rank = 5). As described below, entry to the matrix can be refined using the decision key and models.

TABLE 2

Decision key example for simulants/decontaminants

Exposure-response: effective biological contact area (EBCA)

(1) Is compound part of personal use packet, used only indoors, or used only at prepared chemical training ranges?
Yes - small quantities properly disposed of, EBCA = 0.
small quantities improperly disposed of, EBCA = 1.
No - Uncontrolled use on the training site, EBCA scores are: 1 (Low) < 4072 m^2 (1 acre); 2 (Medium) 4072-40,702 m^2 (1-10 acres); 3 (High) > 40,720 m^2 (> 10 acres)

Toxicity

(2) Is compound a biocide (other than a bactericide)?
Yes - use published toxicity data (Battery 1)
perform laboratory bioassays (Battery 2)
No - use laboratory toxicity data (Battery 1 or 2)
or estimated values (Battery 2)
No biocidal properties, score 0.
Biocidal properties (other than bactericide), score 3.

(3) Is product acutely toxic to native fauna?
Computed as an acute lethal exposure standardized to a 70 kg man, scores are:
1 ≥ 15,000 mg/kg; 2 > 500 - 15,000 mg/kg; 3 < 500 mg/kg.
(Battery 2 [39]. May require Battery 3 testing).

(4) Is the compound genotoxic?
Genotoxic effects may be important if the ecosystem includes species of low reproductive potential, such as large mammals [22,24].
For mutagenicity determined or estimated [36] for the Ames [27] assay, the doubling doses (μg/plate) [26,27] and {scores} are: > 100.0 {0}, > 10.0 {1}, > 1.0 {2}; < 1.0 {3}.
(Battery 1 literature review or Battery 2 bioassays).

(5) Is the compound phytotoxic?
This question cannot be answered presently for most products. There is little systematic phytotoxicity data [41,51], and excepting agrichemicals, there are no models for phytotoxicity that would be generally useful for chemical products. Phytotoxicity is not well established (Battery 1-2), score 2. Evidence (Battery (1-3) suggests significant damage, score 3.

Exposure

For compounds which do not bioaccumulate (below), exposure (mg/m^2) is a multiple of the acute toxicity expressed in human equivalents. If the compound bioaccumulates, the chronic toxicity, or the estimate given by 0.01 (LD_{50} or LC_{50}), is used.
1 (low) < 7 (LD_{50} or LC_{50}) mg/m^2
2 (Medium) 7 (LD_{50} of LC_{50}) mg/m^2 to 70 (LD_{50} or LC_{50}) mg/m^2
3 (High) ≥ 70 (LD_{50} or LC_{50}) mg/m^2

(6) Is the compound likely to bioaccumulate?
If log K_{ow} (octanol-water partition coefficient) ≥ 3.5 (Battery 1-2), assume a bioaccumulation risk, score 3.
Given S, the water solubility (mg/l), $\log S = -0.922 \log K_{OW} + 4.184$ [31].
If the material is rapidly degraded [52], score 1.
Equations to estimate bioaccumulation and half life are in [31].

(7) Is soil accumulation and consequent chronic exposure a concern? Soil accumulation levels can be estimated from the octanol-water partition coefficient [31] and breakdown data [52] (Battery 2). If degradation half-life is < 4 days score 1, else score 3.

(8) Has the product or its hydrolysate a pH outside the range of 5.0-9.0?
No, score 1, else score 3 (Battery 1-2; see [31]).

Decomposition of products, media transfer and accumulation, persistence and bioaccumulation are modifying factors which can be included (Table 2) if they are known or can be estimated.

Implementing the risk matrix

The dearth of release, distribution, and toxicological data, and the complexity of ecological systems, limit forecasting the magnitude and significance of ecotoxicological effects. Although some models of ecosystem effects have been proposed, they have large data requirements and interpretation of model results may be difficult and ambiguous. To avoid the problems associated with inherently complex models, we consider three hierarchical batteries of simple models to predict effects of toxic chemicals on ecological systems. Most decisions regarding the acceptability of products will be made inexpensively with the first battery. The remainder of this paper uses the terms "assay" and "model" interchangeably.

Battery 1 is literature reviews and simple models to estimate water solubility, partition and adsorption coefficients, acute toxicity, vapor pressure and volatilization rates, allometric relationships, and other properties [31]. Battery 2 is simple, single-species, short term bioassay screening assays and chemical analyses. Included here are quantitative structure–activity (QSAR) models for narcotic toxicity [32] and genotoxicity [33–35], methods to estimate Ames assay mutagenicity [36]; carcinogenicity [37], teratogenicity [38], acute toxicity [39] and metabolic pathways and metabolites [40] in mammals, interspecies conversions; bioconcentration factors for aquatic organisms and other species; phytotoxicity [41,42] and equations to estimate the total mass of active compounds in complex mixtures [28,29]. Batteries 1 and 2 predict the character of effects which may be explored through ecological system studies in the third battery.

Models in Battery 3 combine Battery 2 results with ecosystem state (population or trophic level sizes), and/or ecosystem process variables. Because most ecosystem field studies are multispecies, expensive, and lengthy, the battery approach minimizes expense by dropping harmless chemicals early in the procedure. The statistics necessary for estimating the correlation between two or more batteries have been developed [43–46]. Error rates can be estimated for a particular substance out of several substances assayed and for the set of chemicals assayed. This allows the set of assays within a battery to be adjusted to produce a desired error rate.

Ecosystem variables which lead or accompany ecosystem damage from pollutants, and which can be estimated inexpensively, were identified using impact case-studies [47–49]. For example, the size of the nutrient pool tends to increase in aquatic, and decrease in terrestrial, ecosystems. Those changes are easily determined from chemical and volume measurements on streams. Primary productivity, not as easily measured, displays the same responses as the

nutrient pool. Species diversity tends to decrease in pollutant-impacted communities, as does size variability among community members. The reduction in size variability is often from loss of the larger members (large fish or trees). Pollutant-stressed ecosystems are often invaded by species from earlier successional stages, i.e. the system appears to regress successionally. Regression is easily monitored by scoring the relative importance of organisms common in the preceding successional stage.

Decision key for products

Table 2 is a protocol for projecting effects on unspecified ecosystems, processes and components in the absence of knowledge of which effects on which aspects of ecosystems are to be considered significant. The process for characterization and management of risk, which includes data collection, review and interpretation, can be systematized as a decision key [2,50]. The open-ended nature of the set of targets and the set of effects necessarily increases both the number of substances surviving the initial battery and the number of possible effects found for each. The ecohazard can then be refined using results from models which include specific features critical to the exposed ecosystem.

The first step of the decision key is exposure–response (EBCA) assessment. If exposure is other than minimal, sequentially arranged binary choice questions are used to eliminate action pathways. Next, toxicity is considered. If the compound is designed or used as a biocide (other than a bactericide), the key requires that toxicological data come from experiment. Data for non-biocides may be estimated using physical and chemical properties such as water solubility and vapor pressure [31]. One can iteratively move from models with limited predictive ability and data requirements to more comprehensive ones.

Although a decision key allows for systematic decision-making, risks associated with the use of specific products may still be underestimated. Final judgments of ecosystem risk should be made by a group of experts. Such judgments must take into account: (1) best consensus judgment of toxicological hazard; (2) duration of use experience; (3) quantity used; (4) application method; (5) area exposed; (6) probability of a toxic response at that exposure; (7) expected severity of a toxic response at that exposure; and (8) availability of less hazardous materials for the same purpose. Other formal evaluations (e.g. US EPA, FDA) can be used with the caveat that such evaluations may be for totally different conditions of exposure.

Application of risk rank matrix to compounds being evaluated for use

This section applies these methods to rank decontaminants, simulants, diluents, additives, compounds used as thickeners or dyes, and chemicals included for comparative purposes because of chemical similarities to agents (e.g. malathion, parathion) (Table 3). Simulants are chemicals having chemical or

TABLE 3

Summary of data and final ranking of chemical agent simulants, decontaminants, and other compounds

Compound	CAS No.	Rat oral LD_{50}	Solubility ($\log K_{OW}$)	Half life	Breakdown prod. toxicity	Environmental effects	Toxicity	Exposure	EBCA	Total
Simulant										
BIS-EPH[a]	126-63-6	15000	insoluble	years[c,d]	NS[b]	low	2	3	2	12
BIS	3568-48-8	<4950	(2.28)	weeks[c,d]	toxic	moderate	2	3	2	12
BUSH	109-79-5	1500	(1.96)	5 h[d,e]	N/S	moderate	3	1	3	9
CEES	693-07-2	252	(4.3)	6.75[c,d]	N/S	mutagen	2	3	2	12
Diethyl adipate	141-28-6	16000	low	3 d	N/S	low	2		2	4
DEHP	762-04-9	5190	soluble	40 d[d]	N/S	moderate	2	3	2	12
DEM	105-53-3	1500	(0.18)	6 d[d]	N/S	moderate	2	2	3	12
DEP	84-66-2	9000	slight	short	N/S	low	3	2	2	12
Diethyl pinelate	2050-20-6	NA[e]	(2.34)	mod-long	N/S	low	1	1	1	1
DES	110-40-7	14470	slight	mod-long	N/S	low	2	2	2	8
Diethyl succinate	123-25-1	8530	insoluble	moderate	N/S	low	2	2	2	8
DFP	55-91-4	6	(1.92)	72 h[c]	N/S (pH > 7)	high	3	2	2	12
DIMP	1445-75-6	503	(1.2)	long[c,d]	N/S	low	2	3	2	12
DMA	627-93-0	1809	(0.72)	short	N/S	repro	3	3	2	18
DMHP	868-85-9	4250	soluble	10 d[d]	N/S	low	2	1	2	4
DMHP	756-79-6	4000	miscible	13.2[c] 12 d[d]	N/S	moderate	3	3	3	27
DOP	117-81-7	31000	slight	3 weeks	N/S	low (mutagen)	3[h]	2	2	12
DPGME (DPM)	34590-94-8	4900	soluble	moderate	N/S	moderate	2	2	3	12
Ethanol	64-17-5	7060	soluble	short	N/S	low	3	1	2	6
ECA	105-39-5	50	insoluble	74 d[d]	toxic	moderate	3	3	2	18
Malathion	121-75-5	370	(2.89)	moderate	N/S	low	3	3	2	18
Methyl salicylate	119-36-8	887	(2.4)	22 d[d,e] 5.7 d (air)	N/S	low	3	2	2	12
Paraoxon	311-45-5	1.8	1%	weeks	N/S	high	3	2	2	12
Parathion	56-38-2	2-6	(3.81)	> > 62 d	toxic	moderate	3	2	2	12
FEP 200	25322-68-3	28900	soluble	weeks (?)	N/S	low	1	1	3	3
TBP	126-73-8	3000	(4.00)	years (?)	N/S	low	2	2	2	8
TEP	78-40-0	1600	(−3.7)	years[c,d]	N/S	low	3	2	2	12
TMP	512-56-1	840	soluble	short	N/S	low	3	1	2	6

Decontaminant										
CaO	1305-78-8		soluble	mineralizes	N/S	low	1	1	3	3
Citric, acid, anhyd.	77-92-9	11700	soluble	$<<2$ h	N/S	none	2	1	3	6
DETA	111-40-0	1080	soluble	weeks (?)	low	low	2	1	3	6
EGM	109-86-4	3380	soluble	long	N/S	low	2	1	3	6
NaOH	1310-73-2	40	500 g/l	fast	N/S	low	3	1	3	9
$C(OCl)_2$	7778-54-3	850	decomposes	fast	toxic (Cl_2)	low	3	2	3	18
Na_2CO_3	497-19-8	<4000	soluble	fast	N/S	low	2	1	3	6
NaOCl	64-17-5	12	soluble	fast	low	low	3	1	3	9
Other compounds										
Acrylate, butyl	141-32-2	3700	insoluble	slow (?)	NA	low	2	1	1	2
Acrylate, ethyl	140-88-5	1020	slight	slow (?)	NA	moderate	2	1	1	2
Acryloid K1252 (polymethyl methacrylate + ethyl butyl acrtylate	25608-33-7			slow(?)	NA	low	2	1	2	4
Acryloid All	NA	2910		slow (?)	NA	low	1	1	2	2
Chlorobenzene	108-90-7	12600	(2.71)	slow	low (?)	moderate	3	2	0[f]	0
Glycerine	56-81-5	nontoxic	soluble	fast	none	low	1	1	3	3
Gua arabic	9000-01-5	nontoxic	insoluble	slow	none (?)	low	1	1	3	3
Methacrylate, isobutyl	97-86-9	>6400	insoluble	slow (?)	none	low	1	1	1	1
Methylacrylate, methyl	80-62-6	8400	slight	days	low	low	1	1	1	1
p-Nitrophenol	100-02-7	350	slight	days-months	toxic	moderate	3	2	3	18
Perchloroethylene	127-18-4	8850	(0.41)		NA	low	3	1	0[f,g]	0
Phenol	108-95-2	414	soluble	days	NA (low?)	moderate	3	1	0[f]	0
Phenophthalein	77-09-8	>>500	soluble	NA	NA	NA (low?)	1	1	3	3

[a]BIS-EPH = Bis (2-ethylhexyl)-2-ethylhexyl phosphonate; BIS = Bis (2-ethylhexyl) phosphonate; BUSH = n-butyl mercaptan; CEES = 2-chloroethyl sulfide; DEHP = Diethyl hydrogen phosphonate; DEM = Diethyl malonate; DES = Diethyl sebacate; DETA = Diethylenetriamine; DFP = Diisopropyl fluorophosphate; DIMP = Diisopropyl methylphosphonate; DMA = Diemethyl adipate; DMHP = Dimethyl hydrogen phosphonate; DMMP = Dimethyl methyl phosphonate; DOP = Di(2-ethylhexyl) phthalate; DPGME (DPM) = Dipropylene glycol monomethyl ether; ECA = Ethyl chloroacetate; EGM = Ethylene glycol monomethyl ether; TEP = Triethyl phosphate; TBP = Tributyl phosphate; TMP = Triethyl phosphate; PEC = Perchloroethylene.

[b]N/S = not significant.

[c]Soil.

[d]Water.

[e]No available information.

[f]Compounds scored 0 because they are governed under hazardous waste regulations.

[g]Data limited for hazard determination.

[h]Toxicity score based on acute toxicity increased by +1 due to possible mutagenicity of compound/breakdown products.

physical properties resembling those of agents but which are toxicologically safer to use. Decontaminants are compounds having high reactivity, such as oxidizing capacity, which are used to neutralize or decompose agents or simulants. Due to their toxicity or reactivity, simulants and decontaminants may be an environmental hazard.

Use scenarios

Ecosystem effects from simulants depend on the use scenario and the area of the intended release. The amount of simulant needed to simulate low (2 g/m^2) and high 10 g/m^2) site concentrations was estimated using generalized dispersal patterns for several sizes of release areas. Current training use of most compounds in Table 3 is restricted, although some have been used in open-air testing. Decontamination studies and equipment tests have used DEM and DMMP (often with PEG-200 or polymethyl methacrylate and Sudan Red No. 7), methyl salicylate (MS), BUSH, TEP, and BIS. (Abbreviations are defined in the footnote to Table 3).

Hazard ranking

A preliminary hazard ranking was developed based on a qualitative evaluation of the data in Table 3. Final scores were developed using the decision key. Not all answers in the decision key were obtainable from available environmental data. In Table 2, the responses to element 1 in combination with known use scenarios provide the EBCA score. Elements 2–5 contributed to assignment of the score for compound toxicity. Elements 6–8 were used to define the exposure score. Compounds were assigned scores based on literature data, [20, 51–53] and actual or anticipated usage. Toxicity scores were assigned using acute toxicity according to the procedures identified earlier. Most LD_{50} values were for rats, and these values were scaled to a 70 kg human using eqn. (1). If the compound bioaccumulated or had a long half-life, the chronic toxicity dose was used. If the available information suggested that the compound was mutagenic, teratogenic, tumorigenic, phytotoxic, or ecotoxic, the score was increased one unit. Exposure level scores were based mainly on degradability of the compound and its expected half-life. EBCA scores were based on the maximum actual or anticipated dispersion for each compound. Hence, a compound used only in a personal kit was assigned an EBCA score of 1. Compounds now used in the field and sprayed over large areas were assigned an EBCA score of 3. Based on our assessment of the proposed or likely used scenarios of compounds not presently in use, an EBCA score of 2 was assigned.

One product (DMMP) attained the highest score (27), five (DMA, ECA, Malathion, HTH, *p*-nitrophenol) scored 18 and thirteen (BIS, BIS-EPH, CEES, DEHP, DEM, DIMP, DOP, DEP, DFP, DPM, Paraxon, Parathion, TEP) scored 12. All other chemicals scored 9 or below. Risk management considers that the 18 compounds scoring ≥ 12 require additional evaluation. Be-

cause an increase in the use of a compound not in this list could increase its EBCA score, additional compounds could become concerns in the future.

Discussion

Hakanson [54] has proposed a conceptual framework for evaluating relationships between aquatic contamination and ecosystem risk. He formalizes the relationship between exposure, recipient sensitivity and potential effect as: $E=f(D, T, W_i)+R$. In this way, the potential ecological effect (E) is a function of the exposure (D, which could be given as a concentration or a load of a substance or a waste water), the toxicity of the contaminant (T), the sensitivity of the i-th recipient to this given substance or effluent water ($W_i=1, 2,...,n$). The residual term (R) expresses the fact that it is practically impossible to establish a 100% explanatory model in ecological contexts from a limited number of variables. He stresses that the crucial point with this approach, like ours, is to quantitatively express normative E-values from a limited number of readily available, inexpensive and representative integrating variables.

The models considered so far identify the type of effect and its relative magnitude, on a target species. However, the target organism in these models is not necessarily a single species but may, and usually will, be a group of organisms with similar characteristics. For example, if deer is the target species, in the broader ecological sense deer are surrogates for the guild comprised of large mammalian herbivores, such as sheep and cattle, using the same food sources. As another example, if substances causing deoxygenation of a stream are of concern, the appropriate target is all freshwater invertebrates demanding high (>5 mg/l) dissolved oxygen levels, not the daphnia used in a bioassay. Aggregation of species into trophic levels must be done cautiously; e.g. estimates of risk (to a lake) that include population-specific toxicities can be several times the risks estimated from trophic toxicity data [24]. Aggregation into guilds or trophic levels may obscure deleterious effects on particular members of the aggregate that are of concern.

Leaving aside the issue of aggregation, which must be dealt with in the context of particular ecosystems, the task is to translate qualitative or quantitative estimates of hazard to selected target organisms to significant effects in the ecosystems. To do this, we must move away from the one-link direct effects models generally considered in bioassay testing and by population-theoretic ecologists to the systems analyses and network interactions appropriate to actual ecological situations. The ecologist needs to qualitatively describe, as concisely as possible, the ecosystem stressed by products. We suggest that these descriptions should be expressed as connectivity models [55–63] so that the propagation effects through ecological system connectance can be determined. These models can be used in at least two ways. First, the pattern of measured population swings may make it possible to identify through use of the models

the entry point(s) of the stress, and the signs of the most significant correlations between species populations. Second, when population interactions are well known, the models can be used to project the points of damage, even if damage is indirect.

Conclusions and recommendations

The final hazard ranking, like any ranking dependent on consolidation of a large data base into a simplified quantitative system, is both useful and useless. The ranking is useful because by following carefully the decision key, missing data can be identified and the absolute level of support for any ranking can be developed. The ranking is flawed because discrimination in relationships between environmental concentration and effect are smoothed in the simplified structure of the ranking scheme. Nonetheless, the proposed ranking does identify from simple data the compounds posing the greatest environmental risk and shows when a more careful analysis is needed to establish actual environmental hazard.

The results from the hazard ranking procedures, based upon environmental fate estimates and available data, prioritize the environmental research needs for simulants. As additional environmental data are generated, or the use of that simulant changes, the ranking will be re-evaluated. This dynamic ranking system provides flexibility to meet the changing needs of the personnel using the hazard ranking system.

Acknowledgments

We thank the reviewers, Dr. P. Fraleigh and Dr. C. Olson.

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Appendix: Examples demonstrating the ecosystem risk ranking method

Example 1: Sodium N-chlorobenzenesulfonamide (Chloramine B; CAS 127-52-6)

1. Exposure: The most likely route of ecosystem exposure is ingestion by mammals of disposed towelettes. No significant exposure of plants or fish is possible. Exposure score = 0.
2. Biocidal properties (other than a bactericide): Compound is bactericide.
3. Acute toxicity: Given LD_{50} = 15 mg/kg (pulmonary edema in rat); Hence, D_{HUMAN} = 0.251 (15 mg/kg) = 3.77 mg/kg). Toxicity score = 3.
4. Genotoxicity: No mutagenicity data [20].
5. Phytoxicity: No data [51].
6. Bioaccumulation: Chloramine B is water soluble and does not decompose.

Hazard ranking: Toxicity score * exposure score = 0.

Conclusion: It is unlikely Chloramine B poses a toxic hazard to mammals, fish or plants. Exposures to mammals can be eliminated by rewrapping used towelettes in foil and discarding them in appropriate containers.

Example 2: n-Butyl mercaptan (BUSH; CAS 109-79-5)

1. EBCA: Ecohazard is from spray application during field training. EBCA score = 3.
2. Is the compound a biocide? No; weak anticholinesterase activity.

3. Acute toxicity:
   rat: oral LD_{50} = *1500 mg/kg, inhalation* LC_{50} = 4020 ppm/4 h;
   mouse: inhalation LC_{50} = 2500 ppm/4h;
   rabbit: eye irritation = 83 mg/72 h (standard Draize test);
   other: nonmutagenic, nonteratogenic, cholinesterase antagonist slightly soluble in water, highly volatile.

   The compound's high volatility (170,000 mg/m^3, 20°C) and low water solubility protect fish from it. Expected native mammal acute toxicity is nominally medium, but native mammals tend to avoid acute exposure during training exercises. Birds are more likely to experience exposure. Because of its high volatility and low mammalian toxicity, only acute inhalation and contact exposure routes need be considered. Chronic native mammal toxicity is zero since the high volatility prevents chronic exposure. The rat acute LD_{50} (1500 mg/kg) gives D_{MAN} = 0.251 (1500 mg/kg)-376.5 mg/kg, corresponding to asn acute toxicity score of 3.
4. Genotoxicity: BUSH is not genotoxic. It is nonmutagenic to bacteria (Ames test) and insects (*Drosophila melanogastor*) (B.P. McNamara 1979, unpublished data); it is nonteratogenic when given orally to rats in doses of 100 mg/kg/day for 10 days (days 6–15 of gestation) [63].
5. Phytotoxicity: Mercaptans interfere with plant energy assimilation, which is of concern in agriculture but is not important for natural systems, which tend to be limited by other factors.
6. Bioaccumulation: High volatility prevents bioaccumulation.
8. Soil accumulation: High volatility prevents soil accumulation.
9. pH: Very weak acid.

Hazard ranking: Since only vapor and direct contact are available as exposure routes, we treat this conservatively as the low exposure, high EBCA and high toxicity case in Table 3, yielding a total score of 9 (rank = 7).

Conclusion: *N*-butyl mercaptan presents a moderate to low danger to ecosystems, largely because of expected direct application to natural communities. Birds are probably the only community elements at significant risk.