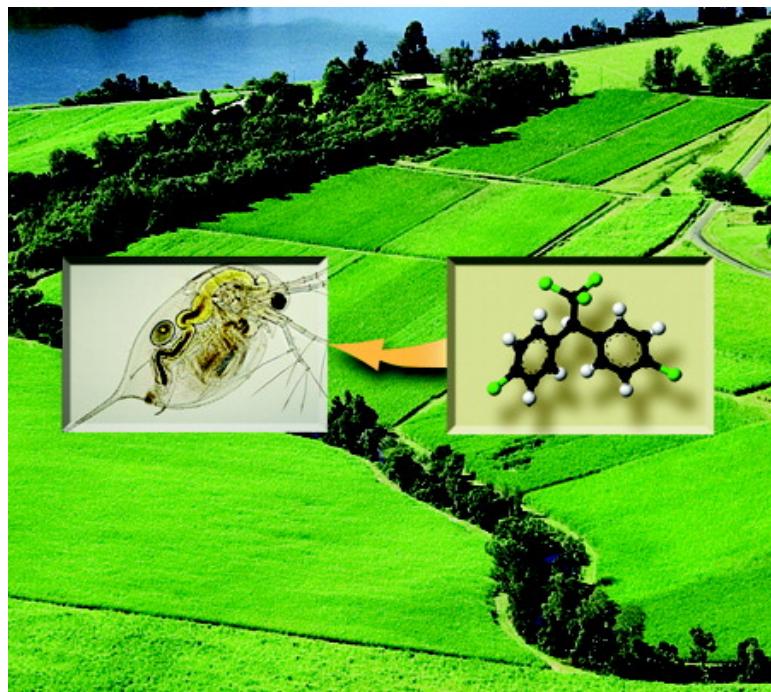


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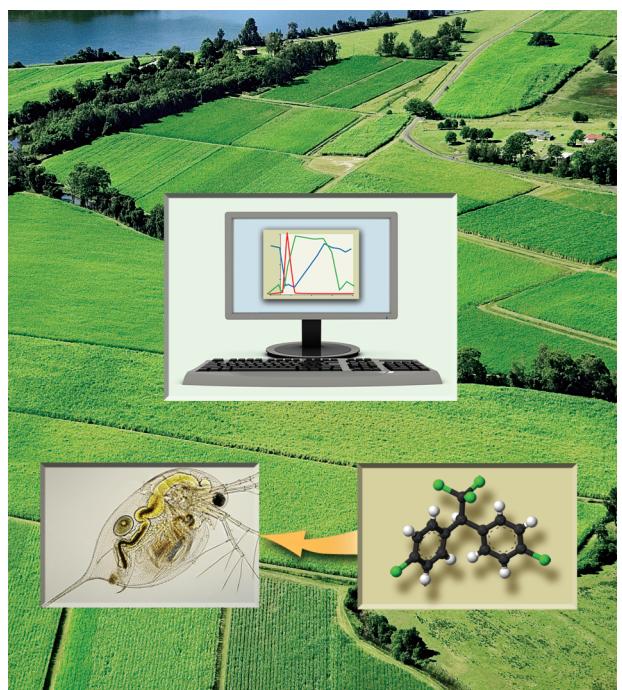
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Ecological Risk Assessment: From Book-Keeping to Chemical Stress Ecology

PAUL J. VAN DEN BRINK

*Alterra & Aquatic Ecology and Water Quality Management,
Wageningen University, The Netherlands*

To best address the effect of chemicals in the environment, extrapolation from single species to ecosystems must be understood and modeled.



Ecotoxicology emerged in the 1970s as the environmental branch of the field of toxicology. As a consequence, its major focus was on investigating the impacts of chemicals on individuals, rather than populations, communities, or ecosystems. Typical ecotoxicity experiments involve testing the effects of a chemical under standard laboratory conditions on individuals of a standard test species. This has yielded a massive amount of historical data, compiled in databases (e.g., www.epa.gov/ecotox) that are used in the ecological risk assessment (ERA) to estimate the environmental consequences of anthropogenic use of chemicals. Because of its focus on standardization, single-species tests, prescribed risk assessment methodologies, and flowcharts, ERA often seems more based on book-keeping than on science. Ecotoxicology, as the science

underpinning ERA, should permit itself to grow out of its “single-species” shell because it should focus on the protection of populations and communities in the field (1, 2). The discrepancy between the question posed in ERA and the answer provided by single-species tests is concealed by the use of assessment factors (1).

This historical background explains why only a very limited amount of ecological theory has become integrated into the field of ecotoxicology and ERA. As a result, science-based, ecosystem-level risk assessment methodologies have hardly been developed. To counteract this ecological deficiency in ERA, frameworks have been proposed to integrate some level of ecology into decision making (e.g., 3, 4). During the past decade, great progress toward this integration has been made on the experimental side (e.g., 5, 6) and some also on the modeling side (e.g., 7, 8). The understanding of how populations, communities, and ecosystems are affected by chemicals could be increased by integrating the fields of toxicology, chemistry, ecology, and bioinformatics at different levels of biological organization. The development of methods to extrapolate this improved understanding to untested situations would then greatly improve the ERA of chemicals (9); Figure 1.

I designate this integrative field as the scientific area of “chemical stress ecology”, which I regard as a subdomain of the recognized field of stress ecology. The term “stress ecology” was used occasionally in the 1970s and 1980s (10, 11) but became institutionalized in the field of ecotoxicology and ERA because of the 2003 paper by Van Straalen (12). Contemporary definitions of ecology and stress are combined in chemical stress ecology, which is the study of the consequences of chemically induced changes in a biological

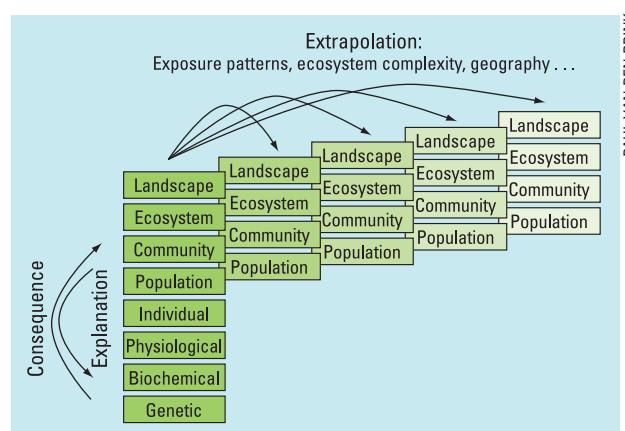


FIGURE 1. Conceptual framework for the propagation of effects across different levels of organization and spatiotemporal extrapolation. The vertical axis denotes the propagation of effects to higher levels of biological organization while explanation of higher-order effects can be found at the lower levels. The horizontal axis indicates that effects recorded for a certain level of biological organization often need extrapolation to other circumstances to be useful for the risk assessment process.

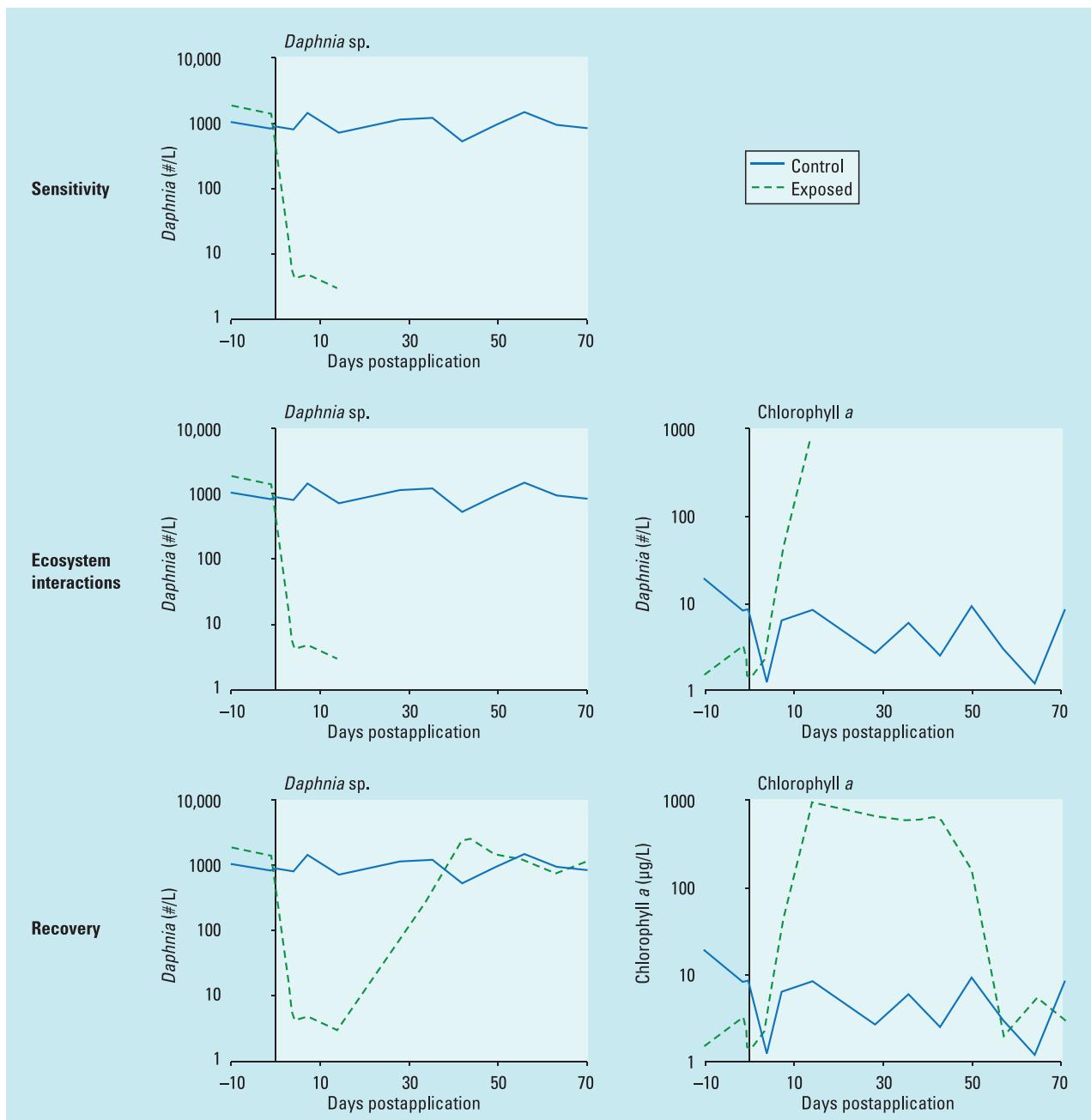


FIGURE 2. The three basic eco(toxico)logical processes determining the response of ecosystems to stressors. The “sensitivity” plot shows the decline of a sensitive species (here, *Daphnia* sp.) after introduction of the stressor at day 0 (direct effect). The “ecosystem interactions” plots show that as a result of the decline of this grazer the chlorophyll *a* of the phytoplankton increases (indirect effect). The “recovery” plots show that because of dissipation of the stressor and the resilience of the ecosystem, a recovery to the levels of an unstressed control can occur.

system and the resultant effects on organisms, such as their abundance, distribution, and interactions with other organisms and the environment.

Levels of biological organization

In this section, I will argue that three aspects are of key importance for the way species and ecosystems react to chemical stress, viz., intrinsic species sensitivity, ecosystem interactions, and recovery (Figure 2). Below, I present how a better understanding of these aspects could improve the scientific foundation and ecological justification of the chemicals’ ERAs.

Sensitivity. Until now, ecotoxicology has put greater emphasis on gathering data on the sensitivity of species than

on understanding the reasons why one species is apparently more sensitive to a chemical than another. While the obvious sensitive species groups in relation to the toxicological mode of action of chemicals such as pesticides are known (e.g., 13), sensitivity differences within these “target” groups remain unexplained and are therefore often described by statistical distributions using the species sensitivity distribution (SSD) concept (14).

Baird and Van den Brink (15) presented an alternative approach based on the hypothesis that the processes of toxicokinetics and toxicodynamics, which determine an organism’s sensitivity to stress, are (partly) driven by their biology, and can (partly) be predicted from species traits related to morphology, life history, physiology, and feeding

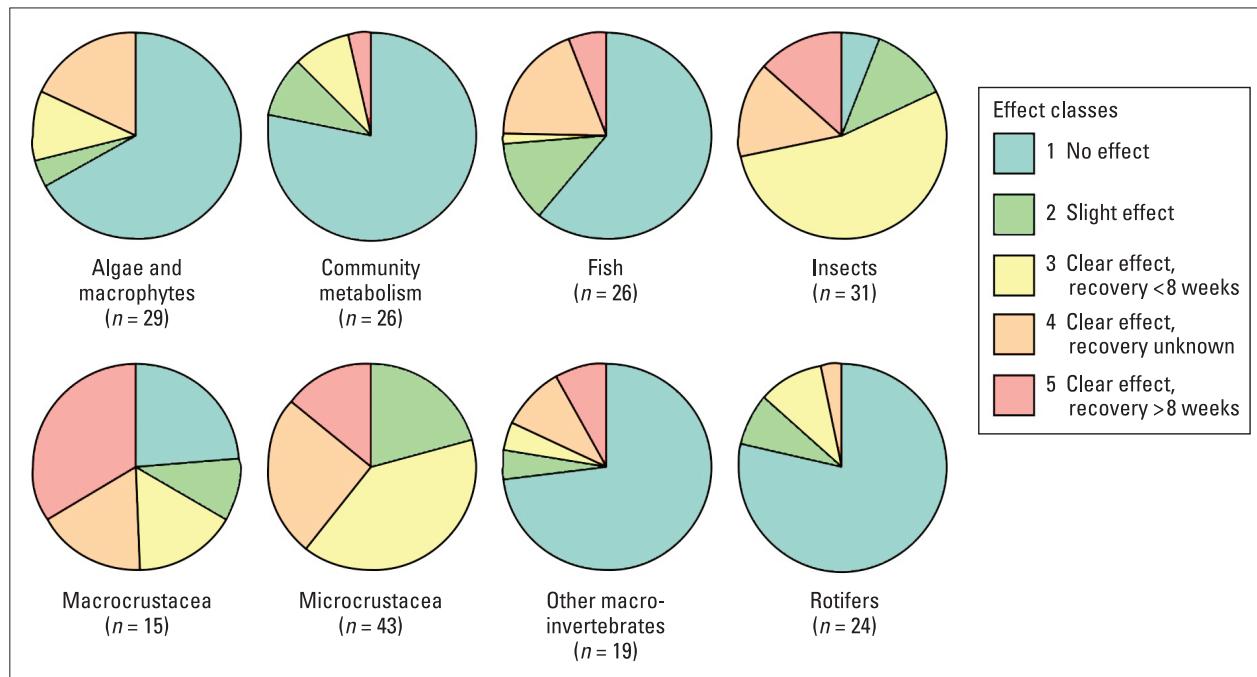


FIGURE 3. Effects (in probabilities of an effect class occurring) as predicted by PERPEST for 1 µg/L chlorpyrifos on eight grouped endpoints. In parentheses is the number of studies on which the prediction is based. As expected from chlorpyrifos' mode of action, the arthropods show the largest probabilities of a clear effect. The effects on non-arthropod invertebrates and primary producers are most likely due to indirect effects.

ecology. These authors presented a preliminary, limited empirical attempt to explain sensitivity differences between species from their traits and found that traits related to respiration, taxonomy, life span, and size explained significant proportions of these differences. Greater insight into the factors determining the response of species to chemical stressors will lead to a better estimation of the uncertainty associated with species sensitivity and thereby to better risk assessments, in which species sensitivity is not a random attribute. Future work in this field could consist of better defining sensitivity by describing the toxicokinetics and toxicodynamics of chemicals in different species and relate these with species traits such as lipid content, size, and mode of respiration (16).

Baird, Rubach, and Van den Brink (17) make a plea for the development of the field of trait-based ecological risk assessment (TERA), which would offer many advantages over the taxonomic approach. The conventional view that taxonomic species are the building blocks of ecosystems can be challenged by the fact that different life stages of the same species can have radically different ecological functions and roles within food webs. For example, functional traits like feeding type and/or dispersal ability can make the difference between a highly localized versus a widespread impact. On the other hand, different taxonomic species may have similar roles within the ecosystem and may be interchangeable from a functional point of view: functional redundancy. Therefore, expressing communities as combinations of functional trait characteristics rather than combinations of species would yield a more relevant description of ecosystem structure and function. Granted, implementation of TERA in biomonitoring faces some challenges, as problems with trait definitions, their intercorrelation, and intraspecific variation first have to be overcome (17).

Ecosystem interactions. Although mechanistic models describing the impact of chemicals on food webs do exist (e.g., 18–20), microcosms and mesocosms—collectively

referred to here as cosms—are currently the only ecosystem-level tool used routinely in the risk assessment of chemicals in the EU (e.g., 21). Cosm experiments allow threshold values of effects to be set at the population, community, and ecosystem levels, recovery patterns to be extracted for populations and communities (discussed below), and indirect effect patterns to be evaluated (5). Cosm experiments are often criticized for their lack of clear criteria for acceptability of effects. In Van den Brink (6) I argue that the lack of clear evaluation criteria is not a problem of semi-field experiments alone but of ERA in general, since risk managers have as yet not described or proposed protection goals in terms of quantifiable measurement endpoints. In fact, we should be glad that cosm experiments force scientists to further investigate the fundamental questions of cause, effect, and consequence.

The vast number of cosm experiments conducted with pesticides has enabled us to develop the empirical PERPEST model (22). PERPEST predicts the effects of a particular concentration of a pesticide on various (community) endpoints simultaneously (Figure 3). PERPEST shows that although we do not have a full understanding of the ecological processes involved in the translation of direct effects into indirect effects, we can still include them in our risk assessments via empirical approaches. In the future, this insight into indirect effects should be improved by the further development of ecosystem models using the wealth of information available from cosm experiments for hypothesis generation and validation (18, 23).

The onset of informatics is a major challenge to ERA, but surmounting this could unlock the full potential of the historical data generated by ecologists and ecotoxicologists. For instance, Mazzatorta et al. (24) used historical toxicity data and neural networks to predict the acute toxicity of 562 organic compounds to fish.

Recovery. The protection aims of various EU directives (e.g., 1) do accept some (short-term) effects at the population

level. Nevertheless, relatively little attention is being given to the recovery of affected populations. This is partly a result of the fact that recovery is context-specific, i.e., potentially variable in space and time, and therefore not a constant parameter that can be used unequivocally in ERA (7). This problem can be overcome by defining scenarios and developing computer models that integrate exposure to the chemical, toxicity, movement patterns of a species, and life-cycle characteristics, expressed at the landscape level. Population simulation models are able to simulate the dynamics of the abundance or distribution of a single species in a particular environment (8, 25, 26). As such, these models provide information on the protection of populations, biodiversity, and system function, rather than individuals.

In the future, models should be further developed that enable recovery times to be estimated as a function of species traits describing their life-cycle, movement pattern, reproduction, etc., as well as of the spatial and ecological infrastructure of the landscape and the spatial and temporal exposure dynamics of the chemical under consideration. Another important future focus will be that of measuring and modeling movement patterns of spatially structured populations and relating these to the ecology of the species. At present, the MASTEP model (7) is based on a situation where *Asellus* is assumed to be present in all parts of the water bodies, with movement represented as a random walk. In the future, individual life history and movement of a species should be defined independently of the assumed spatial habitat configuration, thereby integrating ecological (population) theory into chemical stress ecology. To increase the realism, population models like MASTEP could then be combined with toxicokinetic and toxicodynamic models, yielding a tool that describes how effects at the individual level propagate to the metapopulation level. The truly ecotoxicological information thus rendered could then feed directly into risk assessment schemes.

Extrapolation

One of the major challenges in chemical stress ecology is to develop methodologies, models, rules of thumb, etc., that can be used to extrapolate effects and recovery patterns observed or modeled for one specific situation to another situation (9). For instance, can a sensitivity value for a European species be used in a North American risk assessment? How protective is a threshold concentration derived from a plankton-dominated ecosystem for a macrophyte-dominated ecosystem? In this section, I will reflect on three types of extrapolation that I consider to be important for the ERA of chemicals, viz., extrapolation across exposure patterns, ecosystem complexity, and geography (Figure 1). For an all-embracing overview of types of extrapolations and extrapolation methods the reader is referred to Solomon et al. (9).

Exposure pattern. The risks of chemicals to aquatic ecosystems are often assessed by performing cosm experiments evaluating a particular exposure regime (e.g., one application), which does not necessarily correspond with the exposure for which the risk assessment is being conducted (e.g., multiple applications). To allow an appropriate linkage of the fate to the effects part of the risk assessment, the results of these cosm experiments therefore sometimes need extrapolation to a different exposure pattern than that evaluated in the cosm experiment itself (27). For this, experimental, empirical, and mechanistic approaches have to be established at different levels of biological organization.

At the individual level, models describing the toxicokinetics and toxicodynamics of chemicals within individuals are used for extrapolation across exposure regimes (16). Several modeling strategies are available at the population level (8). At the ecosystem level, however, mechanistic modeling is difficult, as discussed with respect to ecosystem

interaction above. An empirical approach like that of developing the PERPEST model would help to provide insight as to whether rules of thumb exist for extrapolation at the ecosystem level.

Ecosystem complexity. Recently, questions have been raised as to whether an ecosystem with decreasing numbers of species would be able to maintain functional properties and process rates that are comparable with those of species-rich ecosystems (diversity–stability hypothesis; 28). Microcosm experiments have demonstrated that communities with fewer species per functional group were less stable than those with more species (29). This is a result not only of functional redundancy but also of the sampling effect, i.e., a community with more species having a greater chance of hosting a species with a dominant negative or positive effect (30). Hence, biodiversity could be regarded as a factor contributing to the “reliability” of ecosystems (31). To maintain this diversity after a stress event, diversity in the response to different stressors (response diversity) within a (biological) functional group is also of great importance in order to preserve function. It has also been suggested that ecosystem functions are more robust in terms of resistance to change than diversity or other structural measures in ecosystems under stress (32).

To increase knowledge, more experiments should evaluate the relationship between levels of biological diversity and the importance of the processes of adaptation and functional redundancy for the response of structural and functional endpoints to chemical stressors. This will also enable the validity of the diversity–stability hypothesis for aquatic systems to be evaluated, providing a true example of the integration of ecology and ecotoxicology in the field of chemical stress ecology.

Geography. Natural populations and communities are spatially heterogeneous, which may result in a high degree of variability in their response to chemical stress. It is, however, neither financially nor practically feasible to test a large number of chemicals on a large number of species and communities in different locations. Therefore, the spatial extrapolation of ecotoxicological effect data is an important issue in ecological risk assessment. Two types of geographical extrapolations can be distinguished: between ecozones and between climate zones.

Various reviews have compared the outcomes of cosm experiments with the same pesticides in different ecozones, mainly the Nearctic (North America) and the Palaearctic (Europe) (e.g., 4). These comparisons comprised not only geographical variation but also variation in ecosystem structure, season, and exposure pattern. At the single-species level, no differences between Nearctic and Palaearctic species were found in their sensitivity to insecticides (13, 33). The Supporting Information shows that no differences in threshold value are found for the insecticide chlorpyrifos between experiments performed in the Palaearctic, Nearctic, and Australasian (Australia and Asia) ecozones.

For climate zones, growing concern about risks of chemical use in the tropics has made temperate-to-tropical ecosystem extrapolation a focus of research in recent years. Validation studies of the protective value of temperate toxicity threshold values for tropical freshwaters have focused mainly on a species-level (or species-assemblage-level) approach by comparing sensitivities of species between temperate and tropical freshwaters using SSDs. The most extensive sensitivity comparison between temperate and tropical species sensitivities was made in a recent study by Kwok et al. (34). In this study, SSDs of temperate and tropical species assemblages were constructed for 18 chemical substances (nutrients, metals, narcotics, and pesticides). For six of the chemicals, tropical organisms tended to be more sensitive

than their temperate counterparts. However, the opposite trend was noted for several other chemicals, especially metals (34). Future tropical effect assessment studies should include tropical model ecosystem studies evaluating pesticide concentration ranges. It is necessary to verify whether surrogate indigenous test species are representative of local tropical freshwater ecosystems and unravel which factors determine differences in exposure dynamics, indirect effects, and recovery patterns.

Synthesis

The views outlined above are not meant to provide a comprehensive assessment of chemical stress ecology. First, I have focused mostly on aquatic ecosystems and pesticides, for the simple reason that most of my research was, and is, in this field. Since modern pesticides tend to be less persistent and more toxic, this also means that the main focus of this view is on acute effects and less on chronic, sublethal effects, although this may be less true for herbicides and fungicides (13, 35).

Despite these limitations, I believe I have presented a coherent framework for addressing the effects at different levels of biological organization and the extrapolation of these effects. This framework is consistent in the sense that it starts by gathering empirical data. The historical focus of ecotoxicology on testing has yielded, as a blessing in disguise, a wealth of historical data available for empirical models and other eco-informatics tools. Theories, rules of thumb, and regularities extracted from these empirical analyses can guide the process of designing experiments and formulating hypotheses on *why* and *how* chemicals affect populations, communities, and ecosystems. These hypotheses can then be used to design experiments and formulate models to establish the state of knowledge and develop tools for prediction and extrapolation.

The limited available empirical evidence on extrapolation suggests that the threshold value for no effects can be extrapolated across ecosystem structure, season, and geography (see Supporting Information). When the threshold level is exceeded, however, indirect effects and recovery have proved to be context-dependent. This means that intrinsic sensitivity may be an inherent property of species and hence of communities and ecosystems, whereas recovery and ecosystem interactions are context-specific.

The overarching integrative concept linking the propagation of effects to higher levels of biological organization and the spatiotemporal extrapolation of direct and indirect effects and recovery patterns (Figures 1 and 2) is that of traits. Whereas taxonomy can be regarded as a higher-level expression of the genetic composition of organisms, traits can be seen as their functional consequence and thus have greater relevance for the field of ERA. It is essential for a science-based risk assessment of chemicals that the relations between chemical exposure and its ecological consequences are investigated at different levels of biological organization in a spatiotemporal context. Because testing chemicals for every ERA scenario is impossible, tools integrating toxicology, chemistry, and ecology *must* be developed to improve the ERA of chemicals.

Paul J. Van den Brink is a professor of chemical stress ecology and works at the research institute Alterra and the Aquatic Ecology and Water Quality Management Group of Wageningen University, both belonging to the Wageningen University and Research Centre (The Netherlands). Van den Brink is the current president of SETAC Europe (Society of Environmental Toxicology and Chemistry) and editor of the journal Environmental Toxicology and Chemistry. Address correspondence about this article to Van den Brink at Paul.vandenbrink@wur.nl.

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Supporting Information Available

Threshold levels determined using cosm experiments performed in different geographical regions for the insecticide chlorpyrifos. This information is available free of charge via the Internet at <http://pubs.acs.org>.

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