Does *Ecotoxicology* Inform ECOLOGICAL RISK Assessment?

Some argue that ecotoxicology is too simplistic to do the job effectively.

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What is ecotoxicology for? One important answer to this question is to inform ecological risk assessment, that is, to provide a basis for assessing whether industrial and agricultural chemicals are likely to have adverse effects on ecosystems and hence provide a foundation for managing them.

But ecotoxicology has evolved more from toxicology than ecology, and so there are those who argue that it is too simplistic to do the job effectively. This begs a number of questions that we shall address in this article: What kinds of adverse ecological effects are of concern in ecological risk assessment? How can we anticipate these effects to put into practice appropriate controls? How can we check the effectiveness of these approaches?

Extrapolating the results of ecotoxicological tests to likely ecosystem effects undoubtedly involves a number of uncertainties. We shall discuss sources of these uncertainties and suggest ways that they might be addressed. Recent research suggests that there may be some general rules, which should increase our confidence in extrapolating results from simple to more complex ecological systems. However, there remains substantial room for improvement.

What are the concerns for ecology?

The fundamental theoretical basis for toxicology is provided by the dose–response model, in which the number of individuals in a test population responding to different doses of a chemical is used as a measure of the chemical's toxicity (*1*). This is often expressed in terms of a fixed percentile—for example, the LD_{50}, or the dose at which 50% of the population suffers a lethal response (Figure 1). The toxicological concept of dose–response was adopted during the early days of ecotoxicology with the modification that dose—referring to an amount of chemical introduced into an organism by injection or ingestion—was replaced by the concentration of a chemical in the environment.

However, despite the continued focus of the most widely used ecotoxicological tests on survival or sublethal performance of individuals, it is generally recognized that such an approach is a considerable oversimplification of real ecological conditions. For one thing, ecology, unlike toxicology, is rarely concerned only with individuals. That is because populations, communities (mixed species groups), and ecosystems (communities in interaction with their abiotic surroundings) can persist, within limits, despite losses of individuals. What matters is persistence at a particular population size and structure, and preventing irreversible reductions that could lead to extinction. Communities are often very dynamic in composition, and there are complex inter-relationships between composition and ecosystem processes (*2*). Communities are therefore not as easily characterized as organisms, which is why the concept of "health" is not appropriate (*3*). With respect to ecosystems, whether concern should be focused on species composition or ecological processes has been widely debated, but as a matter of practicality, we usually focus on protecting species composition because that should ensure the protection of ecosystem processes (*4*).

Standard practice in regulatory ecotoxicology

Ecotoxicology and toxicology follow similar procedures for assessing dose/concentration–response relationships in laboratory systems. These relationships are typically based on measures of survival, individual growth, and reproduction in small groups of a few "wildlife species", usually including fish (representing "meat" eaters) and daphnids (representing plant-eating zooplankton). Population growth responses are measured in unicellular algae, which represent photosynthesizing plankton at the base of many food chains. The emphasis has been on freshwater systems because these are often the environments in which releases first take place.

For example, the European Centre of Ecotoxicology and Toxicology of Chemicals (ECETOC) Aquatic Toxicity (EAT) database, one of the most extensive ecotoxicological databases (*5*), contains a total of 2200 entries covering 368 chemicals and 137 aquatic species. Of the entries, 76% are freshwater species, and the remaining 24% cover marine species. Short-term tests at high concentrations (acute scenarios) are more common than long-term tests at lower concentrations (chronic scenarios). In the EAT database, 67% of the entries are acute tests, and the remainder are chronic or subchronic tests (*5*). In addition to the 50% response concentration (e.g., LC_{50}, for lethal responses or effective concentration for sublethal response in 50% of the

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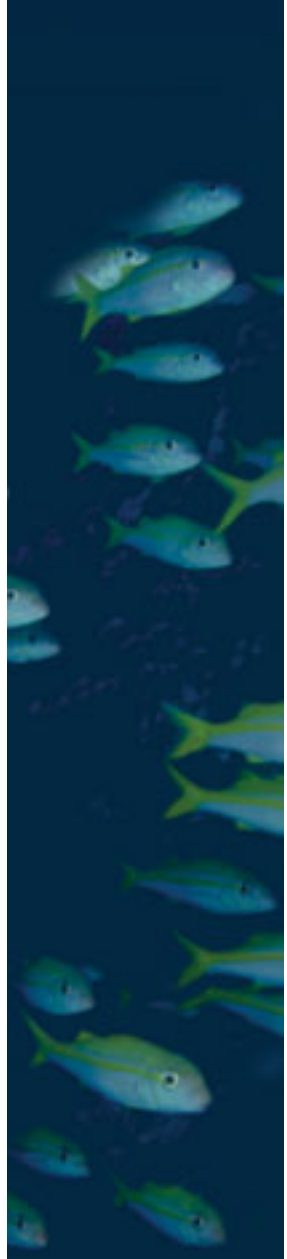
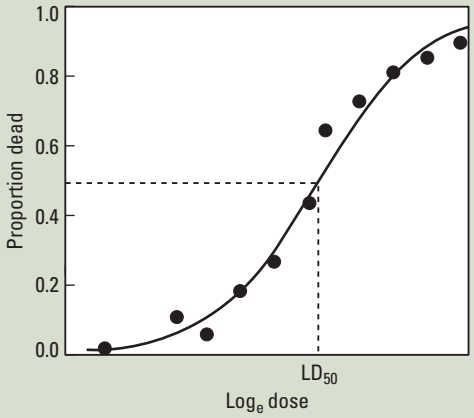


population [EC_{50}] for sublethal responses), two other important measures of response are the lowest-observed-effect concentration, which describes the lowest tested concentration that shows a statistically significant difference from an unexposed control group, and the no-observed-effect concentration (NOEC), which represents the highest tested concentration not showing a statistically significant difference from an unexposed control group. (For a detailed description of ecotoxicological terminology, see Reference (*6*).)

FIGURE 1

A classic dose–response curve in which the response is death

The *x* axis is typically log-transformed to make the curve symmetrical about its inflection point. LD_{50} is the concentration at which 50% of the test organisms die.



Although research in ecotoxicology has progressed substantially from simplified, single-species laboratory tests, the ecotoxicological data that provide the scientific input to risk assessments are based primarily on such simple tests. These tests are used because they are relatively quick, easy, and inexpensive to conduct; have internationally accepted protocols that facilitate validation and interpretation of test results; and are required by certain national and international chemicals legislation.

A tiered strategy of testing is commonly followed, in which initial risk assessments are generated on the basis of a few, very simple ecotoxicological tests and worst-case assumptions with regard to exposure. If low risk is indicated at the first step, no further testing may be necessary. However, if a chemical fails the initial risk assessment, additional, more sophisticated testing is often required. The aim of the tiered approach is to minimize testing of probable low-risk chemicals so that efforts can be focused on chemicals that are more likely to cause undesirable ecological effects. The critical next step is translating the test responses to effects of concern in complex ecological systems.

Translating responses to ecological effects

Ecological risk assessments may be performed before some planned activity or release (prospective risk assessments) or afterward (retrospective risk assessments). The aim of prospective risk assessment is generally to assess the likelihood that exposure to predicted levels of a chemical following a planned activity or release will cause adverse effects; in the retrospective assessments, the goal is often to identify the causes of adverse effects that have already occurred. Sometimes, we are concerned with particular systems, such as those under threat from a specific industrial site, but more often, we worry about protecting ecosystems in general from chemical releases. Extrapolating from observations on individuals in a few test species in simple laboratory systems to complex natural systems involving many individuals and species is therefore a central and challenging part of ecological risk assessment.

Figure 2 shows the two approaches that have been used for these extrapolations. One involves dividing endpoints representing realistic worst cases by fixed extrapolation factors, sometimes called application or safety factors, to derive a threshold value below which adverse ecological effects are defined to be unlikely (*4*). This threshold is referred to as a predicted no-effect concentration (PNEC).

The other method involves using available critical response data, usually an EC_{50} or NOEC, as a function of chemical concentration to construct a frequency distribution of the affected species. This is referred to as a species sensitivity distribution (SSD). Using SSD, it is possible to estimate the concentration of a chemical that exceeds the critical response for specified percentages of species (*7*, *8*). In such analyses, concentrations below the critical-effect concentration for some large fraction of the species in the distributions, usually 90–95%, are generally considered to be protective for intact ecosystems and are used instead of a PNEC for assessing risk.

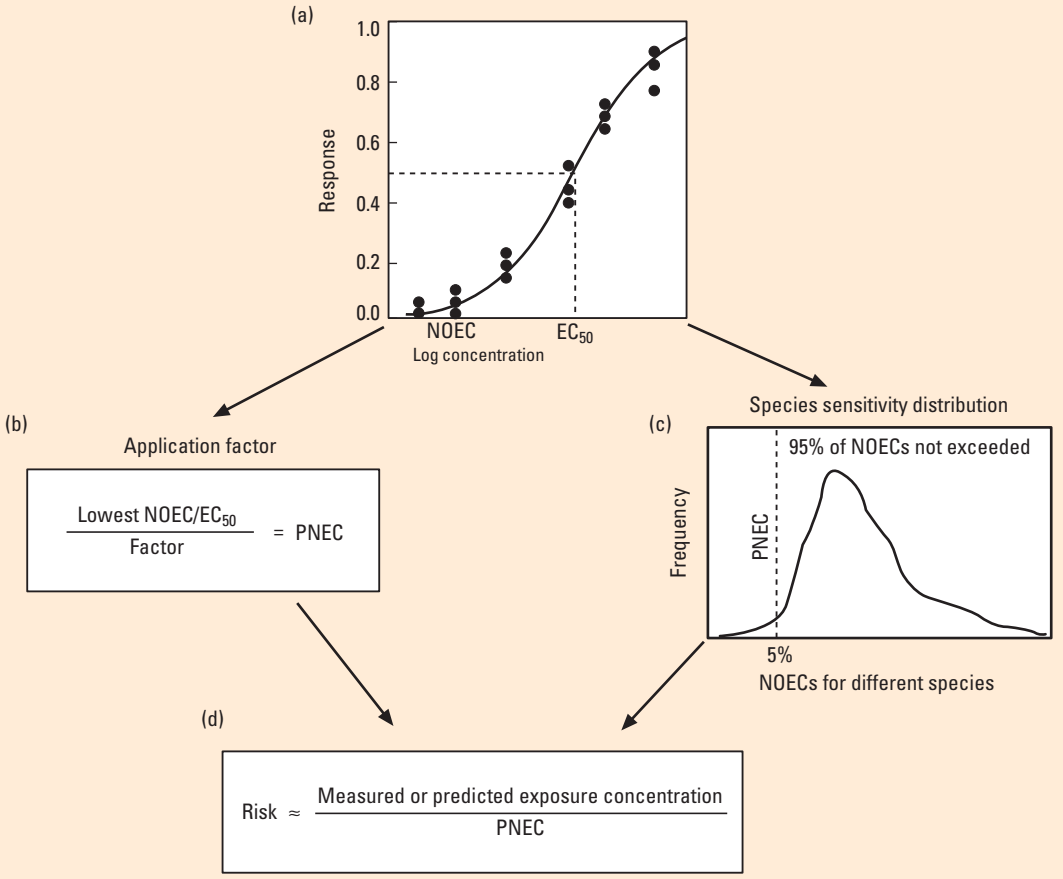
There is no doubt that if SSDs represent appropriate endpoints in relevant species from the community or communities under threat they provide a firmer basis for ecological risk assessment than those provided by a fixed-factor approach. However, the data usually are from such a small and biased set of species that the fundamental assumption of representativeness is violated (*9*). We rarely have enough information on the species sensitivities in particular communities to place much confidence in the precise risk probabilities generated by the SSD approach (*9*). Indeed, under most circumstances in which the two methods have been applied to date, the fixed-factor approach is arguably at least as useful as the SSD and is more easily used on a routine basis. In comparisons between fixed factors and SSDs with identical data sets (*10–12*), the differences go either way and are chemical-dependent. Whereas inconsistencies may be partly due to differences in the chemical mode of action, an important and largely overlooked contributor has to be that the data sets used for different chemicals are composed of different sets of species.

So, given the difficulties in generating the appropriate data sets that could make the SSD approach a more robust technique for assessing ecological impacts, it is likely that fixed extrapolation factors will

FIGURE 2

Ecotoxicological data extrapolated to estimate ecological effects for regulatory risk assessment

(a) Using ecotoxicological data with 50% effective concentration (EC_{50}) and no-observed-effect concentration (NOEC) determined, two approaches are used to (d) assess risk. (b) One involves dividing endpoints representing realistic worst cases with fixed extrapolation factors, such as 10, 100, or 1000, to derive a predicted no-effect concentration (PNEC) below which adverse ecological effects are defined to be unlikely. (c) The other approach uses data in (a) to construct a frequency distribution of affected species in terms of NOEC (or EC_{50}) as a function of chemical concentration to determine PNEC.



continue to play a prominent role in the risk assessment process for some time to come. Hence, it is critical to be sure that the factors adequately account for all of the uncertainties associated with the extrapolation, and most importantly that these include allowances for relationships between individual- and population-level changes, acute and chronic responses, and species sensitivities.

For the first of these uncertainties, significant insights have been provided by life-cycle and demographic models on how changes in survival, developmental rates, and reproduction influence population dynamics, which allow the sensitivity of population dynamics to changes in these vital rates to be quantified (*13, 14*). Figure 3 provides an example of how population growth rates are influenced by these vital rates and their sensitivities to toxicants. When we compare vital rate and population growth rate responses to chemicals, it appears that the population response is unlikely to be any more sensitive to chemical concentration, provided we measure the most sensitive vital rate (*15*). Unfortunately, it is rarely obvious which vital rate is likely to be the most sensitive to chemicals a priori.

With the other sources of uncertainty, it is unclear from first principles how short-term acute responses relate to long-term chronic responses (which are often more likely exposure scenarios in the field), and how sensitivity in one species relates to that of another, taking into account both physiological and life-cycle differences. Under these circumstances, we have had to rely more on correlation analyses than mechanistic models. The data sets available for these kinds of analyses are large but not customized for the questions being considered, and correlations never give the last word.

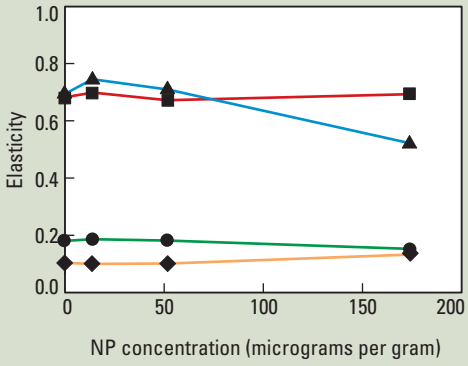
Figure 4 shows an example of the degree of variability between acute and chronic responses—ex-

pressed as the ratio of the concentration at which acute and chronic responses occur (ACR)—within and among chemicals having different modes of toxic action. These data show that the degree of variability in ACRs is substantial among chemical classes and even within groups of chemicals having similar modes of action. This variability can be up to about 3 orders of magnitude. This suggests that using a fixed factor to extrapolate from acute to chronic responses will often result in errors. Our recent analyses indicate that variability among species, particularly in chronic responses, may be greater than expected, based on the fixed factors typically applied to account for this source of variability (*4*).

FIGURE 3

Elasticity of population growth rate to life-history traits

The elasticity (e) of population growth rate (λ) is shown with respect to juvenile, S_j [red]; adult survivorship, S_a [orange]; time from birth to first reproduction, t_j [blue]; and fecundity, n [green], in a marine polychaete as a function of exposure to nonylphenol (NP). In this study, λ was most sensitive to changes in S_j followed by t_j, and relatively insensitive to changes in n and S_a. Neither S_j nor S_a were impaired by NP, whereas t_j was delayed by 17% and n was reduced by 61% at the highest NP exposure. Despite the smaller effect of NP on t_j relative to n, it made a larger contribution to the overall effect on λ as a result of its greater e. Elasticity of λ to a life-history trait (a) is defined by $e_a = (a/\lambda)\,(\delta\lambda/\delta a)$. Adapted with permission from Reference (*25*).



Another problem is that even if it were possible to determine factors that, on average, could account for the relevant uncertainties, an unavoidable disadvantage of applying fixed factors is that they will often be wrong. Ecological effects for some chemicals will be underestimated, whereas others will be overestimated. The fact that our mistakes may cancel each other out, on average, does not instill much confidence in the risk assessment process. An alternative strategy would be to choose large enough factors so that ecological effects are never underestimated. However, this would result in substantial overestimates of risks for most chemicals and might unnecessarily prevent society from enjoying their benefits.

Guarding against mistakes and surprises

Because there are uncertainties in the risk assessment process, we must monitor ecosystems for possible adverse effects. This is part of the rationale for focusing on the assessment and protection of ecological quality in the European Union's Water Framework legislation (*16*). The challenges here are the same as in human epidemiology, but with the addition of being able to recognize adverse changes in systems that often have ill-defined boundaries and are inherently noisy.

It is frequently argued that biomarker responses in cellular and molecular systems may be useful in ecological monitoring (*17*). However, there are some caveats: Those biomarkers that are specific for exposure scenarios may miss the key agents, and some biomarkers may signal effects that are hardly relevant for protecting populations and ecosystems. Nevertheless, used with care, they may be helpful as early warning systems.

Assuming that adverse changes have been recognized in ecological systems, which will depend on well-designed sampling programs (*18*), the problems for ecoepidemiology are analogous to those in epidemiology—ascribing cause to effect from imperfect historical data that are usually based on no more than correlations between variables. Here, it is important to come to a view by weighing the evidence in as systematic and transparent a way as possible. A developing methodology, based on epidemiological experience, now facilitates this (*19–21*).

Ecotoxicology plays an important part in assigning cause by providing experimental evidence that chemical x at concentration y may or may not have the effects observed in the ecological system concerned. In situ, direct toxicity assessment coupled with toxicity identification evaluation procedures can also be helpful in identifying causes of problems and tracking sources (*22*).

In addition to catching possible mistakes in our risk assessments arising from the uncertainties mentioned above, another important reason for prioritizing ecological monitoring programs is to guard against surprises. Observations of morphological deformities and population declines in wildlife populations in the field—and not formal risk assessment procedures—sounded the first alarm that endocrine-disrupting chemicals might be causing adverse effects. Few, if any, standard ecotoxicological test methods were then designed to detect endocrine disruptions. Today, efforts are under way to develop or adapt test protocols to detect these mechanisms of action. It would be naïve to assume that other such surprises are not likely, yet they are seemingly difficult to prepare for before they occur. Continued vigilance is thus essential.

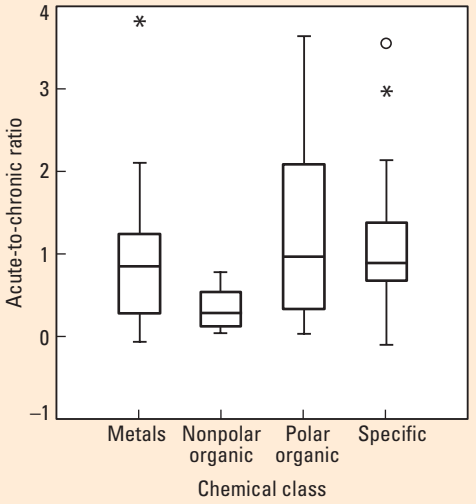
How ecotoxicology could be made more informative

Clearly, ecotoxicology does inform ecological risk assessment despite the uncertainties in the approach. These are different from the uncertainties between

FIGURE 4

Acute-to-chronic ratios for different chemical classes

Chemicals, such as metals, nonpolar organics, polar organics, and specific-acting chemicals (e.g., pesticides), have different effects. The *y* axis is on a log10 scale. The stars and circle represent outliers, and the horizontal lines in the data box represent the median. Data reanalyzed from Reference (*26*).



toxicological observations and human health risk assessment. In human health risk assessment, the concerns are in extrapolating from results on a few, supposedly surrogate, test species to a single target species. In ecological risk assessment, the concerns are in extrapolating from observations on a few individuals in a few species to groupings of many individuals and species.

To date, most of these issues in ecological risk assessment have been handled somewhat arbitrarily; yet a better understanding of the relative sensitivities of individual responses as measured in ecotoxicological tests and the responses of populations, communities, and ecosystems is beginning to provide a firmer basis for extrapolation.

Still, more work is required. In particular, exploring the importance of population density effects in modulating the action of toxicants needs more attention, because these effects are likely to be of general relevance in field populations. Unfortunately, they are rarely if ever considered in ecotoxicological tests or in risk assessments (*23*). In addition, the importance of life cycle variability and community species composition for constructing SSDs must be further evaluated (*9*, *24*). Finally, the relationship between community composition and ecosystem processes and services must be considered when assessing the ecological and societal consequences of pollution. However, in developing risk assessments that can be deployed routinely and generically, it will continue to be important to balance pragmatism with ecological realism.

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