Comments on the Significance and Use of Tissue Residues in Sediment Toxicology and Risk Assessment

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Introduction

The explicit use of body residues in ecotoxicology and risk assessment is relatively new and is currently hampered by the limited availability of residue-effect data. However, the body-residue-based approach is theoretically sound and ultimately lies at the heart of existing environmental-media-based methodologies. It is expected that residue-effect methodologies will soon supplant toxicity-only and bioaccumulation-only based procedures. In the interim, methods to combine and exploit existing toxicity and bioaccumulation knowledge are being developed and refined. However, there is still an opportunity to examine and direct how residue-based approaches may be best employed. Five topics are reviewed and recommendations offered.

What are the assumptions, applications, and limitations for each bioaccumulation or risk assessment methodology being described?

All bioaccumulation-related approaches make the assumption that bottom-up extrapolation is valid and has been validated. That is to say that results and information obtained in laboratory or controlled testing can be readily generalized and extrapolated to population/community/ecosystem levels of organization. This is essentially a matter of faith or at best a policy decision. Such approaches may be useful and/or effective in protecting the environment, but they are not firmly based in science since most such ecological organization theories have not been validated.

For example, the conventional levels of a biological organization model are presented by Odum (1971). However, the practical ability to exploit this model for environmental protection and environmental risk assessment is not universally accepted. Consider Fry's Paradigm, which addresses it: "You take the properties of a level of organization and use those observations to analyze the next level of organization below it. If you take the properties too many steps down, you're being stupid; and you cannot go the other direction." (Kerr, 1976)

Fry's Paradigm clearly indicates that the very essence of much of the current risk assessment process—extrapolation from laboratory testing at the level of the individual organism (and below) to effects in the field at the population, community, and ecosystem levels of organization—is unwise if not impossible. Furthermore, it should be pointed out that the model presented by Odum is not the only model for looking at how ecology is organized. In fact, there is no single, generally accepted ecosystem paradigm (Botkin, 1990 as cited by Suter, 1993). Less than half of about two dozen ecosystem concepts proposed by ecologists have been employed by risk managers/assessors and only about one-third are used with any regularity (Vigerstad, manuscript).

Munkittrick and McCarty (1995) examined the uneasy relationship between ecology and toxicology known as ecotoxicology. They point out that such conceptual models of environmental impacts, although considering direct and indirect factors, do not usually consider what they term nondirect factors, or induced factors as they are called in other disciplines. Nondirect factors are those which do not originate with a response to a chemical stressor and cannot be expressed in terms of a toxicological dose. For example, what is the dose metric of loss of half of the habitat of a species and how is it quantitatively combined with the direct and indirect effects of toxicant stress?

Basing a comprehensive and extensive regulatory framework on using a risk assessment process that depends heavily on the discipline of ecology, which itself is in turmoil and undecided on a generally accepted paradigm, and toxicology, which is struggling to expand beyond the boundaries of the laboratory, is clearly policy rather than science. The biblical warning about building an edifice on a foundation of sand appears appropriate. It may be good policy, but scientists

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and risk assessors should be wary of confusing science-policy with science.

How can bioaccumulation assessment be effectively applied to human health and ecological risk assessments?

Bioaccumulation in and of itself is not an adverse environmental effect. Only accumulation that is associated with an adverse effect in organisms which have accumulated the material, either directly or via the food chain, is of importance. Furthermore, many materials produce adverse effects without any significant accumulation. For environmental protection and associated regulations bioaccumulation is not the issue, adverse effects are!

More work has been done on examining dose-response relationships for changes and adverse effects in the lab, effects that are possible, than for changes and adverse effects in the field, effects that are probable. As too little is known about ecosystems and effects at higher levels of organization (Calow, 1994), it is not surprising that relationships between lab effects at the individual/population levels and field effects at the community/ecosystem levels are poorly understood.

Furthermore, science policy must provide technical definitions of significant adverse effects in the field that are quantifiable by ecologists and risk assessors and consistent with current ecological theories. It is safe to say, with the multiplicity of changes and putative adverse effects being studied and reported in the literature, that currently there is no general agreement on what constitutes significant adverse environmental effects. Risk assessment is useful only in a comprehensive risk management framework where risk management goals are specified in the technical terms that scientists practicing risk assessment can quantitatively address (McCarty and Power, in press).

What are the requirements for selecting species for bioaccumulation testing? Are indigenous species necessary?

Using residue information and appropriate bioassay interpretation, real differences in species sensitivity can be separated from differences associated with bioavailability and toxicokinetics. Indigenous species may be useful and may provide important information but only when the nature of "sensitivity" differences are clear and sensitivity itself is clearly defined. For example, the influence of bioavailability, exposure medium, and various modifying factors (e.g., body size) is often not considered in toxicity test interpretation. There is an almost mythical belief that a most sensitive species can be determined and that this determination will be useful. However, most sensitivity examinations use exposure-medium-based LC50s or similar estimates and do not fully interpret toxicity test results to remove the effects of modifying factors. Such efforts are largely futile (Power and McCarty, manuscript).

Some headway can be made with a residue-based approach. Lanno and McCarty (in press) discuss a case comparing pentachlorophenol toxicity to a freshwater fish, a freshwater benthic invertebrate, and the common earthworm. Based on exposure-based LC50 bioassay results, it appears that the fish is more sensitive (threshold LC50 of 0.00039 mmol/L) than the benthic invertebrate (threshold LC50 of 0.0019 mmol/L). The threshold LC50 of 0.14 mmol/kg dry soil for the earthworm is not comparable due to the differences in exposure media. However, when the lethal body residues (LR) are examined, a sensitivity comparison is possible between the three organisms. The LR50 range is 0.08-0.17, 0.33-0.79, and 0.3-101 mmol/kg wet weight for the fish, earthworm, and benthic invertebrate, respectively. The fish appears to be slightly more sensitive, but the effect of differing body lipid contents has yet to be determined and may alter the relationship.

How can tissue-specific residue levels be coupled with chronic toxicity response data to develop dose-response relationships for bioaccumulative contaminants?

This issue is examined in detail in McCarty and Mackay (1993) and in Rand et al. (1995). The basic approaches are estimation using existing toxicity data and bioaccumulation relationships (i.e., exposure-based toxicity estimate* bioconcentration factor = whole-body residue-based toxicity estimate) and generation of residue-effect data from new experimental testing.

Work by Mayer (Mayer et al., 1986, 1992) on the relationship between acute and chronic toxicity endpoints is particularly valuable in both approaches. He has established that, in many cases, the lower tail (specifically, LC0.01) of the distribution of acutely lethal toxicity is equivalent to the maximum acceptable toxicant concentration (MATC)/lowest observed effect concentration (LOEC) obtained in chronic toxicity testing for growth/survival but not reproduction endpoints. Figure 1 (modified from Figure 5, McCarty and Mackay, 1993) illustrates the point. When the toxicity data from a test are transformed to a linear relationship using the log-probit transformation, any proportional response can be interpolated or extrapolated. When measured exposure-based (LCx) or residue-based (LRx) toxicity data are available, values can be directly estimated. Similarly, LRx data can be estimated from LCx data where bioconcentration factor information is available.

This approach provides many additional insights due to a more complete exploitation of toxicity test information. It was the basis for the suggestion that the current separate acute toxicity, chronic toxicity, and bioconcentration tests be combined into a single aquatic toxicity test protocol (McCarty, 1991). Such a combined approach would require an alteration in direction from

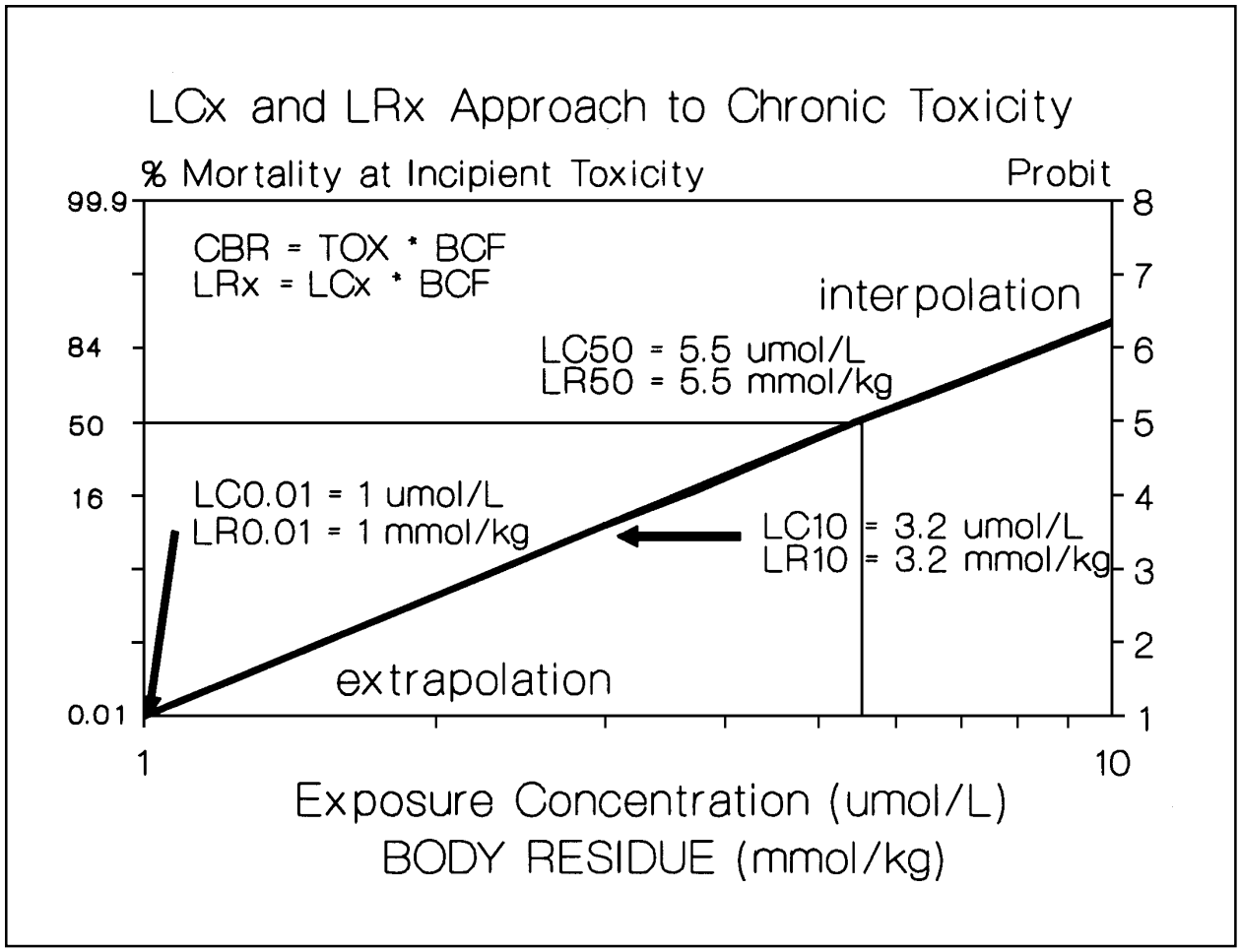

Figure 1. LCx and LRx approach to chronic toxicity.

the current minimalistic trend in testing protocols. Despite the additional observations and sampling that would be required, it is still likely to be less costly than the current trio of tests. As well, when conducted as scientific research experiments, such an approach will ultimately be more informative than abbreviated "regulatory" testing.

Although there is an increasing demand for more chronic toxicity data, both exposure-based and residue-based chronic information have a number of limitations. These include the following: chronic data are more expensive to collect, chronic results usually have greater uncertainty, some chronic endpoints are not readily interpretable, relationships between whole body levels versus those found in selected tissues have not been worked out, and differences in body lipid content confound precise residue-based interpretation.

It should be possible to develop protocols and procedures to address these limitations. The current limited availability of chronic residue-effect data can be overcome by estimation and new experimentation. However, the desirability of shifting the bulk of ecotoxicological testing to chronic effects is based on the largely mythical belief that chronic data are somehow "better" as the basis of environmental regulations. This appears to be largely based on the assumption that chronic effects in the laboratory are equivalent to and readily comparable to chronic effects in actual field situations. This issue is addressed in the next section.

How can bioaccumulation assessment methods, including testing and models, be used to address population-level effects?

At the present time only acutely toxic effects and major growth or reproductive effects can be effectively modeled by population modelers. The major difficulty is density-dependent responses, which are poorly known. The objective of environmental protection is protection of communities and local ecosystems. However, density-dependent and other interspecies interactions, which are largely unknown, represent a poorly quantified level of complexity that effectively renders extrapolation from bioassay to field largely an exercise in professional judgment, not quantitative analysis and modeling (Power and McCarty, manuscript).

Laboratory toxicity testing is focused primarily on addressing bioavailability, kinetics, and the resistance/

tolerance of organisms exposed under highly controlled conditions. The density-dependent factors that influence toxic effects in the field are rarely studied in the lab. An exception is work by Arthur and Dixon (1994). Juvenile fathead minnows were placed in 1-L screen cages at a density of 1, 5, and 10 individuals per cage and then placed in a flow-through system exposure apparatus. The chronic growth effects of pentachlorophenol and 2,4,5-trichlorophenol were examined for 28 days. Up to about twofold differences in chronic toxicity were found, e.g., low-density LOEC = 71 µg/L PCP; high-density LOEC = 121 µg/L PCP.

There is a significant effect of density on the outcome of a toxicity test in tightly controlled experimentation in the lab with a single species over a relatively short time period. It is clear that, with the myriad of opportunities for confounding influences in field situations of multiple populations under varying conditions, density-dependent factors remain a serious obstacle to extrapolating laboratory testing data to the field. The severity of the problem increases as consideration moves from short-term acute effects to long-term chronic effects as there is more opportunity for density-dependent factors to operate the longer organisms are stressed but still living. Thus, more chronic residue-based toxicity information alone will not improve the success of extrapolating such information to the field. A much better understanding of the influence of density-dependent factors is also required before any substantial improvement can be expected.

Conclusions

1. Basic ecological theories need further development and clear separation from science policy.
2. Bioaccumulation is not intrinsically an adverse effect endpoint.
3. Body residues can help identify true differences in species sensitivity by improving the understanding of modifying factors.
4. Chronic body residue data can assist in interpretation of toxicity testing results if improved methods and analyses are adopted.
5. Improvement in lab-to-field extrapolation requires both greater residue-based toxicity knowledge and a better understanding of the density-dependent modifying factors acting within and between species in the field.

Recommendations

Policy. Separate science from science-based policy by use of a clear risk management framework.

Toxicity 1. Develop a single generic bioassay protocol that integrates acute and chronic toxicity as well as bioconcentration.

Toxicity 2. Do not use a local species unless at least one standard species, selected from a very restricted list, is also tested. Clarify that an indigenous species is actually more sensitive than the standard species using tissue residue-effect relationships to determine the influence of modifying factors such as body size, temperature, behavior, and nutritional characteristics.

Ecology. Further develop a basic ecological paradigm and enhance population/community knowledge, especially for density-dependent interactions.

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