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Associating Ecosystem Service Losses with Indicators of Toxicity in Habitat Equivalency Analysis

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ABSTRACT / Habitat equivalency analysis (HEA) was developed as a tool to scale mitigation or restoration when habitat is contaminated by hazardous substances or has been otherwise harmed by anthropogenic activities. Applying HEA involves balancing reductions in habitat quality against gains from restoration actions, and quantifying changes in habitat quality in terms of ecological services. We propose a framework for developing ecological service definitions and measures that incorporate knowledge about the impacts of chemical contaminants on biota. We describe a general model for integrating multiple lines of evidence about the toxicity of hazardous substances to allow mapping of toxicological inputs to ecological service losses. We provide an example of how this framework might be used in a HEA that quantifies ecological services provided by estuarine sediments contaminated with polycyclic aromatic hydrocarbons.

Habitat equivalency analysis (HEA) is a method used to quantify the effects of natural resource injuries resulting from releases of hazardous substances or other anthropogenic perturbations and to scale compensatory restoration (Dunford and others 2004; NOAA 1999b; Chapman and others 1998; Strange and others 2002; Penn and Thomasi 2002). Restoration scaling using HEA involves quantifying the expected effects of a restoration action so that the benefits of the restoration are equivalent to the losses associated with the habitat degradation. The practical definition of ecological services can be problematic when the habitat degradation in question includes contamination by hazardous substances, because, in that case, direct quantification of damages in easily measurable units (e.g., acres of a particular habitat type) might be an inadequate or inappropriate choice of metric. The methods for determining the equivalency of ecological services might play a key role in cases where multiple stakeholders with disparate and conflicting interests attempt to reach agreement about the adequacy and relative value of proposed restoration alternatives.

Interpreting reports about the toxic effects of hazardous substances and estimating the associated actual or potential environmental injuries and applying this knowledge in HEA is still an emerging aspect of the practice, and novel, case-specific algorithms for interpreting exposure data might be employed (e.g., Penn and Tomasi 2002). There are no standard methods for translating knowledge about toxicity into HEA formulations because the understanding of toxicity might be based on experimental and observational studies of toxic responses that include a wide variety of organisms and different types of toxic responses. Translating knowledge about toxicity into formulas useful for HEA typically requires some degree of subjective interpretation and justifications based on professional judgment.

We believe that HEA applications can be improved by minimizing HEA'S reliance on formulas that can be criticized as overly subjective. This article describes a philosophy and general framework for interpreting and integrating knowledge about various kinds of toxicity in a manner that we believe is useful in the context of HEA. Our framework does not completely eliminate subjectivity from HEA, but we believe that it constrains subjectivity in a manner that improves the overall reliability of a HEA by providing predetermined

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general schema. Elements of this framework have been applied previously HEA applications (e.g., Penn and Tomasi 2002). However, the practice as a whole has included a tendency to include interpretations of toxicological facts and the development of a loss function that occurs in isolation or with insufficient reference to established guidance about how to integrate multiple kinds of toxicological information in a manner that reflects a more general systematic approach to the process. Although we acknowledge that HEA will always rely to some extent on subjective considerations and case-specific concerns, our intention in describing our conceptual framework is to begin formalizing some of the rationale behind integrative consideration of toxicological concerns in HEA applications and to thereby enhance the reliability and acceptance of the process in general.

We begin with a brief review of the basic HEA model, with emphasis on the concept of service loss. Then we discuss the concept of a hierarchy of adverse biological responses to hazardous substances ranging from cellular to community level and we propose principles for interpreting those responses in terms amenable for HEA. Finally, we provide a practical example of how the framework could be used to interpret the effects of polycyclic aromatic hydrocarbons (PAHs) contamination on a benthic marine community.

The General HEA Model

To assess equivalency, both resource injuries and the benefits of restoration are quantified in terms of services (NOAA 1999b), but the nature of the replacement services are not necessarily identical to the lost services, and quantification on either side of the ledger might be problematic for various reasons. Equivalency can be considered for a single species or habitat function of concern or it can involve integrated services provided to many species.

The basic equivalency model used in a HEA to scale restoration actions, including consideration of discounting for time lag, is as follows:

$$\sum_{t=t_0}^{t_l} L_t(1+i)^{(P-t)} = \sum_{s=s_0}^{s_l} R_s(1+i)^{(P-s)} \qquad (1)$$

where L_t is lost services at time t, R_s is replacement services at time s, t_0 is the time when service loss begins, t_l is the time when service loss ends, s_0 is the time when replacement services are first provided, s_l is the time when replacement services are last provided, P is the present time when the natural resource damage claim is presented, and i is the periodic discount rate. The model quantifies service losses relative to a baseline condition, in terms both of the degree and duration of losses. More comprehensive descriptions of HEA are provided by Chapman and others (1998), NOAA (1999a, 1999b), and Strange and others (2002).

Ecological Services

The concept of ecological services is central to HEA, so we must clarify and define how service reductions are quantified. In the general HEA formulation (Equation 1), L_t and R_t must be expressed in a common unit, *services*. The concept of ecological service can embrace any number of different structural or functional attributes of an ecosystem (Daily 1997; Limburg and Folke 1999; Norberg 1999), but it typically is not a measurable quantity, *per se*. Habitats and ecosystems typically provide many types of service that are not necessarily obvious (Cairns and Niederlehner 1994), including ecological functions, physical or ecological structure, and welfare of lesser known (e.g., unglamorous or unexploited) species (Holmlund and Hammer 1999; Rowe and Freda 2000). Thus, a comprehensive treatment of equivalency between habitat injuries and the benefits of a restored habitat should rely on well-specified measures of ecological service. When the nature of ecological injuries and restoration actions are expressed in similar, holistic metrics [e.g., acres of severely injured habitat being replaced by acres of restored habitat of the same type (with appropriate scaling and discounting for time lags)], the useful measures of service are relatively obvious, but when habitat injuries involve releases of hazardous substances, there is a likelihood that service losses cannot be adequately expressed in such holistic terms. In such cases, toxicological considerations, including the possibility of nonlethal or other subtle kinds of biological injury should form the definition of service losses.

A Framework for Relating Toxic Effects to Service Loss

We propose a general framework for considering ecological service losses that integrates available information about multiple toxicity end points into a single measure of ecological services. This framework highlights several implicit principles that define applications of the ecological service concept with regard to adverse habitat impacts resulting from hazardous substances. The emphasis of the framework is on identifying and integrating information that can lead to

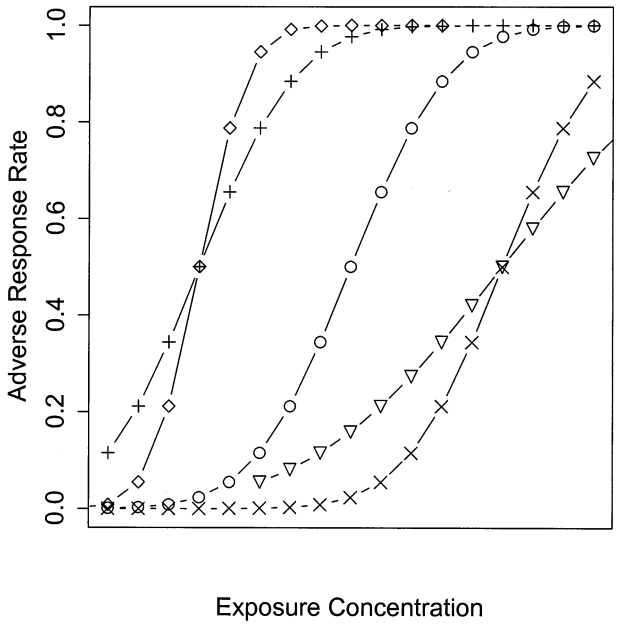

Figure 1. Examples of dose–response curves for different hypothetical responses, each modeled as sigmoidal functions with differing slope and location.

appropriate estimates of service losses (i.e., quantifications on the ''debit side'' of the HEA formulation). Although the framework does not explicitly address the ''credit side'' of the formulation, it is presumed that compensatory actions would either be cleanup actions to eliminate exposures to hazardous substances in the affected habitat or restoration of other uncontaminated habitats that are scaled by reference to replacement services that are deemed to be both relevant and sufficient.

In evaluating effects of contaminants on biota, HEA models should accommodate the possibility of multiple degrees of service loss, and the degree of loss should be linked to conditions (e.g., contaminant concentrations) associated with various types of physiological response in individual organisms. Responses patently detrimental to individuals, such as mortality or gross deformities, should be associated with high degrees of service loss, but sublethal physiological responses, if they do not have cascading effects on individuals or populations, should be associated with lesser degrees of service loss. For example, stimulation of enzyme pathways in response to contaminant exposure might represent a relatively minor service loss. Some analysts might argue that mere pathway stimulation is not a service loss at all, but we maintain that if an organism is induced to expend energy to engage in a detoxification process, the habitat is not providing full service; we need not assume that stimulation of an enzyme pathway is a precursor to death or other adverse effects. The response variable in this relationship is a physiological outcome that arises from adverse habitat conditions but might or might not have a simple correlation with a single stressor.

Many contaminants result in different toxic responses (e.g., mortality, growth, behavioral, biochemical effects) that typically manifest in a nonlinear, dose-dependent fashion. These responses are likely to vary among species. Conceptually, therefore, each response has a distinct dose–response relationship that associates the degree of that effect with an exposure concentration (Figure 1).

Organisms can suffer different types of toxicological effect at different exposure concentrations; therefore, a cumulative response profile for an organism should consider both the nature and the severity of responses for each exposure. Because organisms might be exposed to more than one contaminant, evaluating toxicity could require considering response profiles across multiple compounds where responses can be considered to be completely independent, additive, synergistic, or antagonistic.

In the HEA model, service reductions are expressed as percentages: The ecological service index of a unit of habitat is reduced by a certain fraction that reflects the difference in habitat quality relative to a baseline or reference condition. For ease of discussion, we use the term ''residual services'' to represent the amount of services provided by a habitat after suffering service losses, relative to a baseline or reference defined as 100% (Equation 2).

$$\text{Residual service}(\%) = 100\% - \text{Service losses}(\%) \quad (2)$$

For each distinct dose–response relationship under consideration, a corresponding concentration–service loss relationship can be constructed in which increasing exposure concentration is associated with decreasing residual service. This correspondence results from a mapping that translates empirical information about the action of a particular stressor, typically an exposure–response relationship for a relevant organism and hazardous substance, to individual and integrated indices of percent service loss (Figure 2). Because a variety of toxicity end points and ecosystem services could be evaluated and the nature and severity of adverse responses can differ across contaminants and ecological receptors, a variety of general relationships between physiological response and service losses is conceivable, including various forms of step function, smooth functions that are directly proportional to exposure–response relationships, and other variants

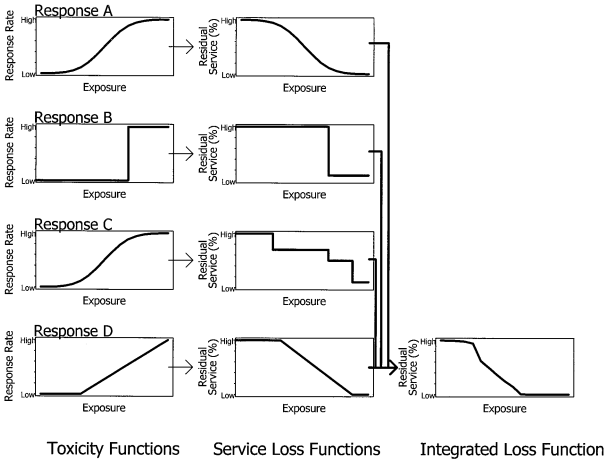

Figure 2. Schematic of hypothetical service loss functions associated with multiple independent adverse response end points, each with different functional forms, integrated into a single service loss function (multiplicative example; see text for details).

that might be selected to capture certain important, perhaps unique, features relevant to the particular organisms or habitats of concern.

We suggest that an appropriate definition of percent service loss from an adverse impact to an ecosystem should reflect several features of adverse toxicological responses, including the following:

- The *type* and *severity* of the effects
- The *degree* of the effects within individuals
- The *extent* of the effects within a population
- The *organizational level* at which the adverse effect occurs (subcellular to ecosystem)

Types of effect include lethality and various sublethal effects such as growth impairment or increased rates of histological abnormalities (Table 1). The severity of an effect is determined by the likelihood that the effect is an indicator of important physiological disruption such as acute mortality, narcosis, carcinogenesis, or reproductive failure. For example, certain types of tumor might be considered severe if they are known to be cancerous or precancerous or if they are known to impact the function of the host tissue. In contrast, an effect would be considered less severe if the likelihood of consequential physiological impairment were minimal. The degree of an effect relates to the extent of physiological impairment within an organism. For example, a fish with extensive gill deformation has suffered a greater degree of impact than a fish with limited gill deformation. The extent of the effect or impairment rate can be expressed as a fraction of an exposed population or as a spatial extent of impact. Certain effects can be considered less extensive if they occur rarely or in a limited part of a population, but they could be more serious (and therefore associated with greater service loss) if they are widespread in a population.

The level of biological organization that is affected should be used to scale service loss rates, where effects at the habitat or community level are associated with more service loss than effects at the cellular or organismal level. Negative effects on habitats or communities are not merely aggregates of effects on individuals because they include negative effects not solely due to direct toxicity. A community-level effect occurs if a species that is not directly exposed to toxicants (or that is exposed but relatively insensitive to a toxicant) experiences negative consequences because toxicity reduces the population of one or more different species and thereby alters food web structure or other competitive relationships. For example, increased mortality among zooplankton could impact higher levels of organization in systems, particularly in aquatic systems where zooplankton represents the major link between primary producers and higher trophic levels (Carpenter and Kitchell 1993). Effects that impair the function of higher levels of biological organization are typically more difficult to identify and are likely to be specific to the particular community or ecosystem in question (Clements and Kiffney 1994).

Some investigators have suggested that ecological services are not reduced unless there is a measurable reduction in population size (Martin and Richardson 1995; Attrill and Depledge 1997). We do not believe that this criterion should be used to determine whether contaminated habitats have suffered service losses. It is often very difficult to detect changes in the size of wild populations (see, e.g., Wedemeyer and others 1984; Adams and others 1993), and changes typically go unnoticed unless the population is monitored, because of its commercial importance or if the change is radical. Populations fluctuate for many reasons that are independent of hazardous substance concentrations or other qualities of the habitat. Determining a baseline population size against which negative impacts might be measured could be impossible either because the population size is not routinely monitored or because an assumption that the population was in an equilibrium condition before a hazardous substance release is unwarranted. Indeed, it might be as problematic to demonstrate that an ecosystem was in equilibrium condition as it is to demonstrate that deviations from equilibrium are a result of hazardous substances. Even

Table 1. General classes of adverse effects that inform associated ecological service loss evaluation

Class of adverse effect	Consideration	Examples of typical hierarchy
Type of effect	What physical or behavioral properties are associated with the stressor?	Lethality > skeletal deformities > enzyme pathway induction
Severity of effect	Are known effects a serious threat to survival of individuals?	Lethality > adduct formation > hepatic tumors
Extent of effect	Are effects widespread, either within individuals or in the population?	High frequency of effect > low frequency of effect
Organizational level affected	What are the ramifications of the effect?	Population size reductions > individual effects

where hazardous substances are known to reduce populations, it could be practically impossible to detect population changes on a meaningful time scale, especially in long-lived species or in species that have variable sensitivities in different portions of their life histories. These features also complicate efforts to assess the success of restoration efforts (Simenstad and Thom 1996; Miller and Simenstad 1997).

Although demonstrable population reductions might suggest higher degrees of service loss, we suggest that less drastic changes also be considered in developing mapping functions for relating toxicological impacts to service losses. Because organisms live on a finite energy budget, any stressor that occurs in addition to natural (i.e., nonanthropogenic) stressors might be detrimental to that organism because the organism must redirect energy to mitigate the effects of the stressor (e.g., Rowe and others 1998). Examples of stress-induced energy redirection include physiological detoxification (e.g., metallothionein induction) and additional caloric expenditures for motility (e.g., due to habitat avoidance behaviors or enlarged home range). Directing energy to these activities must come at the expense of other biological processes such as growth, reproduction, and avoidance of predation. If a particular contaminated habitat is more stressful than an uncontaminated reference site, it provides less service.

Using Toxicity Data to Map Service Loss Functions

Because mapping from dose–response curves to service loss functions is not inherently empirical, using a predetermined framework provides a rational and transparent way to synthesize available information and map from empirical data to a service index. Although mapping functions might not be wholly objective and reproducible, we believe that developing maps according to guidance from several key principles will lead to more consistent and, therefore, more useful mappings.

One such principle is that it is relatively simple to identify the extreme conditions on a percent service scale. If a region is rendered uninhabitable, it provides zero residual service (or perhaps another defined minimum value if the abiotic features or processes are considered to represent services), whereas an uncontaminated baseline condition or reference region provides 100% ecological service. In contrast, there is no obvious, intuitive way to quantify the spectrum of conditions that are intermediate to the ideal or the wasteland.

Our mapping procedure involves two phases. In the first phase, we associate knowledge about sensitivity of individual toxic response end points with a service loss function (Figure 2). For example, in the first phase, we could select a dose–response curve with a response rate that ranges from 0% to 100% and perform a direct translation of response rate to service loss: A 10% response rate is mapped to 10% service loss, an 85% response rate is mapped to 85% service loss, and so on (Figure 2, response A). In the second phase, we integrate service loss due to multiple, possibly independent, stressors into a single value indicating the residual service provided by a unit of habitat.

Several general classes of mapping are plausible, and different types can be used to describe different stressors within one HEA model. A mapping scheme appropriate for lethality responses might not be appropriate for a sublethal response such as induction of CYP1A (a protein biomarker of exposure to hazardous substances) (Stegeman and Hahn 1994). In addition to the simple dose–response inverse, some types of end points might be best described by a step function, perhaps because of the nature of the supporting evidence or the end point itself. A step function might be most appropriate when there is evidence of one or more clear toxicity thresholds and the response end point is relatively static otherwise

(Figure 2, responses B and C). A threshold model can be modified to reflect a graded response for exposures above the threshold (Figure 2, response D), e.g., where the dose–response relationship is well described by a hockey stick model. In some cases, such as the one described below for PAH, where individual relationships are interpreted in conjunction with knowledge of multiple relevant toxicological relationships, specific values of residual service loss might not be explicitly assigned to individual toxic responses.

The second phase enables considering type and severity of effect, degree of effect, extent of effects, and multiple levels of organizational responses. There are many plausible alternative algorithms for integration that could reflect analysts' understanding of the conditions and issues relevant to particular HEA applications, and the utility or validity of selecting a particular algorithm must be weighed through expert opinion within its particular context. Because of their simplicity and transparency, we consider two alternatives, which we term the ''minimum'' and ''multiplicative'' models, to be especially appealing. The minimum model simply assigns to a unit of habitat the smallest residual service value associated with any of the stressors and end points considered. The multiplicative model defines residual service as the product of the residual service values associated with any of the stressors and end points considered. The multiplicative model will typically yield lower values of residual service because each additional stressor considered implies a reduction in residual service, whereas under the minimum model, consideration of additional stressors does not necessarily change the resulting residual service values. Other approaches to mapping that integrate information about diverse types of stressors can be defined based on the available knowledge of various types of toxicological end point.

The end product of this mapping process is a single integrated relationship between contaminant exposure and residual service that is used to quantify residual services in the general HEA model (Equation 1). Figure 2 provides an example of the multiplicative method in which the integrated function is continuous. A drawback of the multiplicative method, or any algorithm that yields a continuous function, is the implication that very small differences in residual service can be associated with very small differences in contaminant exposure. A coarser granularity in the integrated loss function might be preferable in cases where the designs of the experiments that form the basis of the toxicity functions have coarse granularity, which is a common situation either because of the nature of the toxic end points considered, a limited number of experimental exposure concentrations, or both. For this reason, an integrated loss function with coarse resolution, such as a step function with restricted step magnitudes, might be more desirable. The above-described principles regarding the severity, degree, and extent of known effects should be used to help form decisions about the appropriate degree of vertical (service loss) granularity and for selecting the location of steps or other types of break points on the exposure scale.

PAH Example

To illustrate this proposed framework, we use an illustrative example of a HEA that considers the effects of hazardous substances on the ecological services in a shallow estuarine embayment. The example is inspired by an actual HEA analysis, but in this article we have greatly simplified and generalized the case for illustration, so the specific features and conclusions of the case are not presented. Sediments in the waterway are contaminated with varying concentrations of PAHs and a variety of estuarine species are exposed to them. In actuality, an embayment contaminated with PAHs might also be contaminated with other hazardous substances, but for the sake of this illustration, additional hazardous substances are not considered.

We begin by determining the community composition of the case-study waterway and focusing on species that are predominant, are considered to be either particularly at risk of PAH toxicity, or could serve as indicator species.

Flatfish are resident in the waterway and could be exposed to PAHs by the followng;

- Dietary ingestion of contaminated prey
- Incidental ingestion of contaminated sediments
- Transdermal exposure due to prolonged direct contact with contaminated sediments
- Gill exposure

The invertebrate community is also of particular concern because it is exposed to sediment contaminants, including PAHs, and because it functions as an important part of the estuarine food web. If invertebrates are impacted, the community as a whole might be at risk and the ecological services provided by the habitat reduced.

The objective in the assessment of service loss is to interpret the pertinent information about the kinds of toxic effects associated with various concentrations of PAHs in sediment and to use that information to

Table 2. Selected evidence of toxic effects of sediment PAHs on benthic marine organisms

Receptor	Effect	Sediment PAH concentration (ppm, dry weight)
Echinoderm	Adverse effects threshold	4.65
Oyster	Adverse effects threshold	29.8
Microtox	Adverse effects threshold	46.5
Neanthes	Adverse effects threshold	74.4
Amphipod	Adverse effects threshold	164.3
Benthic community	Adverse effects threshold	235.6
English sole	9% with 1 or more lesions	1
English sole	4% Infertile above baseline	1
English sole	18% with 1 or more lesions	2
English sole	10% Infertile above baseline	2
English sole	24% with 1 or more lesions	3
English sole	13% Infertile above baseline	3
English sole	31% with 1 or more lesions	5
English sole	17% Infertile above baseline	5
English sole	40% with 1 or more lesions	10
English sole	23% Infertile above baseline	10
English sole	71% with 1 or more lesions	100
English sole	42% Infertile above baseline	100

Note: For this example, we use Washington State sediment quality guidelines for AETs and data from Johnson (1999) for effects on English sole.

develop a reasonable basis for relating ambient PAH concentrations to reductions in ecological services.

Information about the toxicity of PAHs to estuarine species is available from various sources. Each source provides the results of experiments in which various species were exposed by various modalities, and the researchers considered a variety of lethal and sublethal end points. Casillas and others (1991) found evidence that elevated sediment PAHs (and/or PCBs) were associated with altered reproductive physiology in English sole, and other studies (e.g., Malins and others 1984, 1988) found associations between PAH exposure and hepatic lesions in English sole. Other studies considered PAH effects on several species of marine invertebrates (Table 2).

Here, we consider two particular sources of information about PAH toxicity. In principle, all relevant studies could be considered. We first look at experiments conducted by NOAA (Johnson 1999) that examined the relationship among sediment PAH exposure, the prevalence of various kinds of hepatic lesion, and indicators of reproductive success. The English sole studies (Johnson 1999) identified 1 ppm (dry weight) PAH in sediment as an important threshold above which the prevalence of hepatic lesions rises and above which the rates of various types of reproductive effect increase. The second source is a suite of apparent effects thresholds (AETs) in sediments associated with various adverse outcomes in marine invertebrates, including echinoderms, oysters, neanthes, and amphipods. AETs are determined by considering multiple lines of evidence about the likelihood of adverse toxic effects. Strictly speaking, they are not necessarily determined by controlled exposure studies. AETs are considered sediment quality guidelines and are particularly relevant to this example because they are specific to benthic invertebrates.

We assemble the various critical thresholds identified by these sources and rank them according to the severity, degree, and extent of impact that could be expected in regions of the waterway where PAH concentrations exceed the published thresholds (Table 2). In this case, our ranking of adverse outcomes consistently corresponds to increasing sediment PAH concentrations. We believe that it is appropriate in this case because (1) the responses in sole are clearly of increasing severity and (2) the various invertebrate AET thresholds suggest the possibility of food web disruptions, which indicates the possibility of wide extent.

We then use the data in Table 2 to map the toxicity data to a set of service loss functions (Figure 3). The service loss functions assigned to individual toxicity end points are described as functions similar to response types B and D of Figure 2. The forms of these loss functions are determined in part by subjective considerations. Furthermore, we describe the residual service in terms of ''high'' and ''low'' residual service rather than specific numeric values. In actual practice, specific scales would be determined by technical analysts and stakeholders through consid-

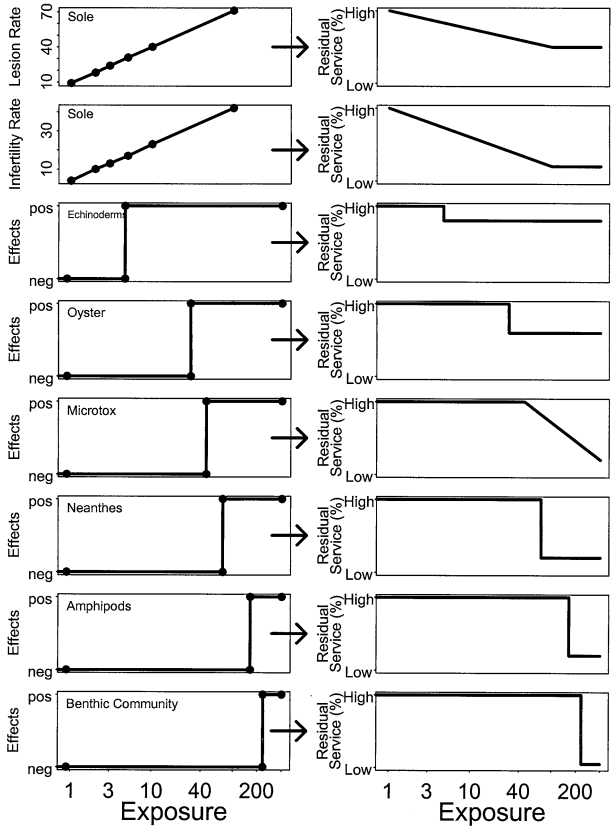

Figure 3. A mapping of eight toxicity relationships between sediment PAH concentration (mg/kg dry wt) and selected estuarine receptors to ecological service loss functions.

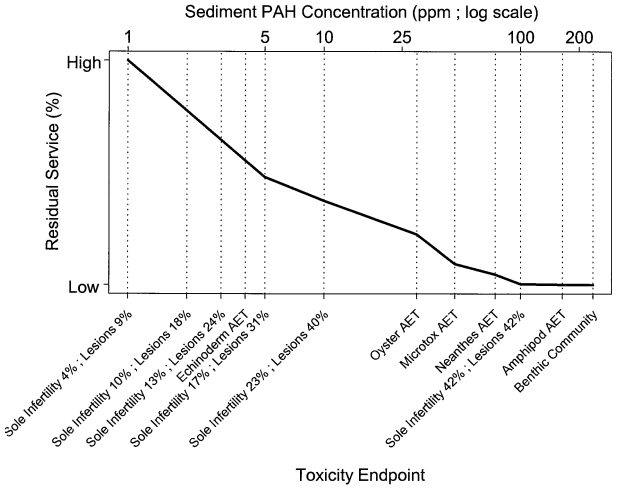

Figure 4. Integrated service loss function relating levels of toxic effects of sediment PAH (mg/kg dry wt) in various organisms to services provided by contaminated estuarine sediments derived from multiple service loss functions depicted in Figure 3.

eration of numerous factors: for example, the presence of additional contaminants, the presence of other ecological receptors with unknown toxic response profiles, and practical concerns relating to the particular social context of the overall analysis. The integration of the loss mapping in Figure 4 is derived through the multiplicative method. The resulting function is an irregular curve, with inflections corresponding to the features of the individual service loss functions. In practice, the integrated function would provide explicit values of residual service that would be associated with the sediment PAH concentrations in the habitat of concern, which could be determined for the region as a whole or for smaller subregions known to have different PAH concentrations. Although the integrated service loss function described here is a continuous curve, practitioners could elect to modify or simplify the function, for example, by describing the function as a step function that roughly corresponds to the continuous curve. This final step could be justified as a reflection of uncertainty about the precision of the numerous underlying analyses or, for practical reasons, related to ease of communication and other social considerations similar to those noted earlier.

Conclusions

The framework proposed here could be used when HEA methods are applied at sites where contamination by hazardous substances is believed to be toxic to biota and, therefore, injurious to the ecosystem. We recommend developing transparent relationships between service losses that incorporate information on the nature and the extent of toxicological end points. Although the general framework requires professional judgment on features relevant to a particular analysis, the principles described can serve as a useful guide for analysts who want to apply toxicological considerations in a HEA.

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