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Coastal and estuarine ecological risk assessment: the need for a more formal approach to stressor identification

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Abstract Application of ecological risk assessment to coastal and estuarine systems is accelerating although it initially lagged behind applications to land and freshwaters. Broader spatial and temporal scales, and multiple stressor integration are appropriately being considered more frequently in all risk assessment activities. This expansion and integration is essential for coastal risk assessment. Because coastal assessments must deal with co-occurrence of several candidate stressors manifesting within broad spatial and temporal scales, wider use of formal methods for assessing causal linkages is needed. Simple Bayesian inference techniques are discussed here to demonstrate their utility in quantifying the belief warranted by available information. The applicability of Bayesian techniques is illustrated with two examples, possible causes of fish kills on the Mid-Atlantic US coast and possible causes of hepatic lesions in fish of Puget Sound (Washington, US).

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Lagoons and Coastal Wetlands in the Global Change Context: Impacts and Management Issues

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Introduction

Initially, application of ecological risk assessment to U.S. marine habitats lagged behind applications to freshwater and land. The reason was not that coastal resource assessment was less important. More than three quarters of all commercial and recreational fish and shellfish species depend on estuaries (Lewis et al., 2001) yet these valuable coastal habitats remain in serious trouble (U.S. Commission on Ocean Policy, 2004). A lack of legal mandates was not the reason: ample U.S. federal legislation existed (see Rand & Carriger, 2001). The reasons for delay seem to arise from cultural biases (Newman & Evans, 2002) and political boundaries. There was an historical delay in implementation as North Americans slowly began to question the assumption that the oceans were too vast to be impacted by humans. As an important example, coastal eutrophication research lagged two decades behind that addressing freshwater eutrophication (Arhonditsis et al., 2003). Historical use patterns of coastal resources tended to be more laissez-faire than use patterns established for land ownership and obligations associated with

terrestrial landscapes. Relative to political boundaries, marine pollution issues are more likely to require interstate or international action for which issues of sovereignty and coordination delay decisions (Deacon et al., 1998).

Coastal habitats now receive an appropriate level of attention, and ecological risk assessment concepts framed initially with terrestrial or freshwater systems in mind are rapidly being adapted to marine systems. Ecological risk assessment, as currently practiced, requires some shift in emphasis to be most effective for marine systems. Crucial changes include expansion of the ecosystem context to encompass central ecotonal and landscape themes, e.g., Brown et al. (2002) and Kiddon et al. (2003). Some assessments of coastal systems can be compromised by not considering the appropriate spatial and temporal scale (Yanagi & Ducrotoy, 2003). Conservation action associated with tributyltin use is a good example of spatial shortcomings in coastal management. Terlizzi et al. (2004) concluded that marine protected areas (MPAs) do not protect *Hexaplex trunculus* populations from tributyltin-induced imposex, stating, ''The most important reason for the limited biological effectiveness of MPAs is that the scale of processes in marine systems is often much larger than scales the reserve can encompass.'' Hawkins et al. (2002) also argue that the temporal scale applied to assessments of tributyltin is too short. Lastly, the co-occurrence of significant multiple stressors is more likely in coastal systems than in terrestrial or freshwater systems.

Consideration of broader scales and possible multiple stressors necessitates more integrated and formal identification of stressors (e.g., Brown et al., 2002; Munns et al., 2002). Qualitative approaches to identifying the most likely stressors from a suite of candidates in conventional assessments are being developed based on qualitative methods such as those of Hill (1965) or Fox (1991). The sufficiency of these different approaches remains untested for coastal assessments requiring vigilant determination of the likelihood that a candidate stressor is, in fact, the stressor needing attention. Being qualitative and often dependent on unstructured expert opinion, these approaches can be susceptible to common errors in human cognition and problem solving (Newman & Evans, 2002). Bayesian techniques provide more formal mathematics for causal assessments, and consequently, for reducing the likelihood of making such errors. Applying Bayesian methods can enhance decision-making.

Two examples illustrate this last point about formally identifying the most plausible stressor(s) in complex coastal systems. Recent controversy about fish kills notionally due to the dinoflagellate, *Pfiesteria piscicida*, demonstrates the high costs of causal model development without adequate consideration of alternate causes. Cancer prevalence in Puget Sound fish demonstrates Bayesian methods for isolating the most plausible cause from many possible causes.

Fish kills caused by *P. piscicida*

From Bacon's 1620 *Novum Organum* to recent cognitive theory (Piattelli-Palmarini, 1994), one thing is clear: the unaided human mind has limited abilities to accurately determine causality and likelihood. The minds of coastal risk assessors are no exception as can be illustrated with recent investigations of fish kills along the Mid-Atlantic U.S coast. In 1992, Burkholder et al. (1992) proposed that *P. piscicida* caused large fish kills in coastal North Carolina. Uncertainty about the cause of these and subsequent fish kills generated much debate among regional scientists and resource managers with early expert opinion favoring the hypothesis that excess nutrient-induced blooms of the toxin-producing *P. piscicida* caused the kills. Suggestions were made that *P. piscicida* blooms also pose a health hazard to humans contacting infested waters. When subsequent large fish kills occurred in Mid-Atlantic coastal waters, substantial revenue was lost in the associated States as seafood sales dropped and tourists avoided the region. Regional decision-making was compromised because the informal expert opinion process became mired in accusations of ethical misconduct, risk exaggeration, and legislative stonewalling (Newman & Evans, 2002; Whitehead et al., 2003; Belousek, 2004). How important *Pfiesteria* or low dissolved oxygen conditions were relative to causing fish kills remains in active area of debate and research.

Table 1 Hill's nine aspects of noninfectious disease association applied to causality assessment for fish kills notionally related to *P. piscicida*

Strength of Association	The probability of fish kill when *P. piscicida* is present is 20.5%, as opposed to 4.9% when it is not present (calculated from Burkholder et al., 1995; Newman & Evans, 2002). Numerous laboratory bioassays have shown that, when *P. piscicida* is present at a level high enough, the fish percent mortality is as high as 100%, as opposed to 0% in the control.
Consistency of association	North Carolina data indicate that there were 9 (Burkholder et al., 1992: Table 1) and 10 (North Carolina Division of Water Quality, http://h2o.enr.state.nc.us/esb/Fishkill/fishkillmain.htm) fish kills, in 1991–1992 and 1997–2003, respectively, associated with *Pfiesteria*.
Specificity of association	North Carolina fish kill data from 1997 to 2003 indicate that among 371 fish kills, only 10 were suspected to be related to toxic effect of *Pfiesteria*. About 93 were associated with low DO conditions. Other causes such as accidental toxic spills or field runoff may also have had significant roles. The specificity of association is low.
Temporal sequence	Among the fish kills linked to *Pfiesteria*, it is difficult to tell the order of occurrences of fish kills and high densities of *Pfiesteria*.
Biological gradient	In the reported field fish kill data, there's no apparent gradient between the number of fish killed and the exposure density and duration to *P. piscicida*.
Plausible biological Mechanism	There is no consensus about the mechanism of *P. piscicida* killing fish yet. One possible mechanism is toxin release. The structural information of the toxin has been only partially defined (Moeller et al., 2001) and it has been demonstrated to be present in extracts from *P. piscicida* strain (Burkholder & Glasgow, 2002). The other possible mechanism is micropredatory feeding (Vogelbein et al., 2002).
Coherence with general Knowledge	The relationship between *P. piscicida* and fish kills is coherent with the generally accepted knowledge that extensive algal blooms can cause fish death, though the mechanisms vary.
Experimental evidence	Considerable experimental evidence exists in the literature, indicating that *P. piscicida* can cause fish death.
Analogy	Most of the toxic dinoflagellates produce polyketide toxins (Miller & Belas, 2003). The toxin-generating mechanism described for *P. piscicida* is similar to this although its structure is still unclear. Its biological activity is often lost within a short period of time, while the typical toxins from other dinoflagellates are stable or can be easily stabilized (Moeller et al., 2001). On the other hand, the experiments with *Pfiesteria shumwayae* cultures of Vogelbein et al. (2002) suggested that the organisms caused fish mortality by micropredatory feeding, not exotoxin production.

In the presence of significant uncertainty about causal relationships, qualitative rules-of-thumb, such as Hill's nine aspects of disease association (Hill, 1965) and Fox's rules of practical causal inference (Fox, 1991), can guide judgments about plausibility of candidate causes of adverse effects. Table 1 illustrates the application of Hill's rules to fish kills notionally caused by *P. piscicida.* These rules are intended to foster a qualitative sense of plausibility for a candidate cause: they are not designed to rigorously compare candidate causes. More quantitative abductive methods are afforded by Bayesian statistics and are presented here as a means of improving causal inferences in marine systems.

Stow (Stow 1999, Stow & Borsuk 2003) used straightforward Bayesian methods to identify an influential, but misleading, inference about coastal North Carolina fish kills. *Pfiesteria piscicida* was found at sites of fish kills 17 of 33 times during three consecutive years of sampling, leading Burkholder et al. (1995) to conclude that "*P. piscicida* was implicated as the causative agent of 52±7% of the major fish kills ... on an annual basis in North Carolina estuaries and coastal waters." The flaw in this conclusion can be illustrated with Bayes's Theorem,

$$p(\text{Fish Kill}|\textit{Pfiesteria}) = \frac{p(\text{Fish Kill})p(\textit{Pfiesteria}|\text{Fish Kill})}{p(\textit{Pfiesteria})}$$

In words, the probability of a fish kill occurring given *P. piscicida* was present is equal to the product of the probability of a fish kill occurring times the probability of finding *P. piscicida* if a fish kill did occur divided by the probability of finding *P. piscicida*. The data collected during the three years estimated

$p(Pfiesteria|\text{Fish Kill})$ but the conclusion was incorrectly made about $p(\text{Fish Kill}|Pfiesteria)$. Estimates of $p(\text{Fish Kill})$ and $p(Pfiesteria)$ are needed to calculate the level of belief warranted about *P. piscicida* causing a fish kill. When Newman & Evans (2002) did this, the odds dropped from the stated 1:2 to 1:5 of a fish kill occurring when *P. piscicida* was present. The causal evidence was not as strong as originally suggested.

This basic approach can be extended to analyze two or more competing causal explanations in coastal systems, e.g., Borsuk et al. (2004). Here, it is extended to calculate the relative likelihood of two competing causes for fish kills being low dissolved oxygen (Low DO) versus *P. piscicida* at the time that Burkholder et al. (1995) made the causal influence about *P. piscicida*. The equation for calculating the probability of a fish kill given the presence of *P. piscicida* is provided above. That for low dissolved oxygen causing a fish kill is the following:

$$p(\text{Fish Kill}|\text{Low DO}) = \frac{p(\text{Fish Kill})p(\text{Low DO}|\text{Fish Kill})}{p(\text{Low DO})}$$

The two competing explanations can be expressed as the quotient of the two probabilities:

$$\frac{p(\text{Fish Kill}|Pfiesteria)}{p(\text{Fish Kill}|\text{Low DO})} = \frac{\frac{p(\text{Fish Kill})p(Pfiesteria|\text{Fish Kill})}{p(Pfiesteria)}}{\frac{p(\text{Fish Kill})p(\text{Low DO}|\text{Fish Kill})}{p(\text{Low DO})}}$$

$$= \frac{p(Pfiesteria|\text{Fish Kill})p(\text{Low DO})}{p(\text{Low DO}|\text{Fish Kill})p(Pfiesteria)}$$

The four probabilities needed to calculate this quotient can be estimated with existing data. As estimated by Burkholder et al., 1995, $p(Pfiesteria|\text{Fish Kill})$ is 0.52 based on the major fish kills in North Carolina coastal waters from 1991 to 1993. Estimation of $p(Pfiesteria)$ can be expressed in two ways because field surveys report either densities of *P. piscicida* or *Pfiesteria*-like organisms (PLO). $P(Pfiesteria)$ was 0.345 for PLO or 0.205 for *P. piscicida* (Newman & Evans, 2002).

The entire coastal North Carolina dissolved oxygen (DO) data set for the same period as the Burkholder et al. (1995) study (1/1/1991 to 12/31/1993) was retrieved from the EPA STORET database. The designation of low oxygen conditions used here was somewhat arbitrary but consistent with regulatory definitions. Solely for purposes of illustration, all DO values lower than 4.0 mg/l were considered as indicative of low DO conditions. There were 674 cases of low DO out of 7100 DO measurements; therefore, $p(\text{Low DO})$ was estimated to be 0.095.

Fish kill data from 1991 to 1993 in North Carolina Coastal waters and estuaries were obtained from Dr. Mark Hale, Division of Water Quality, North Carolina Department of Environmental and Natural Resources (personal communication). The fish kill event was categorized as co-occurring with low DO condition if there was a specific comment that low DO (generally less than 4.0 mg/l) was observed during the period of fish kill. This occurred for nine out of 41 cases so 0.220 is the estimated probability of the presence of low DO when fish kills happened ($p(\text{Low DO}|\text{Fish kill})$).

In this illustration, the likelihood ratio of fish kills due to *P. piscicida* versus fish kills due to low DO concentration can be calculated based on the above estimates for PLO and *P. piscicida* data, respectively:

$$\frac{p(Pfiesteria|\text{Fish Kill})p(\text{Low DO})}{p(\text{Low DO}|\text{Fish Kill})p(Pfiesteria)} = \frac{(0.52)(0.095)}{(0.220)(0.345)} = 0.651$$

$$\frac{p(Pfiesteria|\text{Fish Kill})p(\text{Low DO})}{p(\text{Low DO}|\text{Fish Kill})p(Pfiesteria)} = \frac{(0.52)(0.095)}{(0.220)(0.205)} = 1.095$$

The results show that, when PLO is used in the calculations, the likelihood that *P. piscicida* was

the cause of a fish kill was lower than that of low DO. When *P. piscicida* only was used in the calculations, the likelihood of *P. piscicida* being the cause was approximately the same as that of low DO. Again, after the application of simple Bayesian methods, the level of belief warranted by the data changed from that originally inferred in 1995.

Two points can be made from this example. The decision based on available fish kill data to increase resources and funding to address the *P. piscicida* issue, but not dissolved oxygen issues, was not optimally informed. Also, these analyses suggest that putting more funding into producing better assessments of $p(Pfiesteria|\text{Fish Kill})$, $p(Low\ \text{DO}|\text{Fish Kill})$, $p(\text{Low DO})$, and $p(Pfiesteria)$, and gathering information relative to Hill's rules-of-thumb would result in much more informed decision-making.

Liver cancer in a sentinel fish species

Bayesian tools are also applicable for assessing the most plausible cause of an observed effect in situations where there are many candidate causes. The causal assessment of liver cancer in a sentinel fish species of Puget Sound (Washington, USA) will be used here to illustrate this point.

Hill's (1965) rules-of-thumb can be used to qualitatively judge plausibility of a particular stressor causing an effect. Newman (2001) applied Hill's nine aspects of disease association to hepatic cancer prevalence in English sole from Puget Sound, generating the following conclusions. (1) *Strength of association* between sediment PAH concentrations and cancer prevalence generally enhanced belief that PAH contamination caused cancerous lesions. (2) The *consistency of the association* was high between cancer prevalence and PAH concentration. (3) Logistic regression incorporating many candidate contaminants suggested that the *specificity of the association* between cancer and PAH concentration was moderate to high. (4) Unfortunately, the long latency period between exposure and cancer manifestation did not allow the fourth aspect (*consistent temporal sequence* of exposure then effect manifestation) to be directly assessed; however, experiments did demonstrate the appearance of precancerous lesions after juvenile exposure to PAH. (5) There was a *biological gradient* with cancer prevalence increasing with increasing PAH concentration. (6) A *plausible mechanism* existed, i.e., P450-mediated production of free radicals that form DNA adducts. (7) The proposed causal link of PAH to liver cancer was *coherent with existing, general knowledge* of carcinogenicity. (8) *Laboratory evidence* was produced to support this causal link. (9) Many other *analogous situations* existed in the literature. This application of Hill's rules suggested that sediment PAH contamination was a likely cause of the liver cancers in English sole. However, many candidate causes were present and quantitative discrimination among candidate causes was not done rigorously. Hill's nine rules-of-thumb can be adapted to such purposes but are not designed specifically to discriminate among candidate causes.

Formal Bayesian techniques allow one to be more explicit in large-scale assessments with several potential causes of adverse effect (e.g., Jones, 2001) but Bayesian techniques are infrequently applied to coastal assessment. The recent work of Borsuk (Borsuk, 2004; Borsuk et al., 2003; 2004) is a notable exception. More studies such as those of Borsuk et al. are warranted for complex coastal assessments in which several possible causes exist or the likelihood is high that effects result from multiple causes.

A Bayesian network can be constructed at the beginning of such a study. Bayesian networks show probabilistic connections (i.e., lines or arcs) between variables (nodes). Figure 1 is an example of a network that could be developed for the work of Myers et al. (1998). In Fig. 1, each connection represents a probabilistic dependency between a parent and a child node, and is represented by a function that mathematically defines its dependence on the parent variables.

Uncertainty about probabilistic dependencies (i.e., arcs) can be represented and quantified in Bayesian networks. Bayes's theorem allows both model parameters and observations to be probabilistically distributed and random. Using a

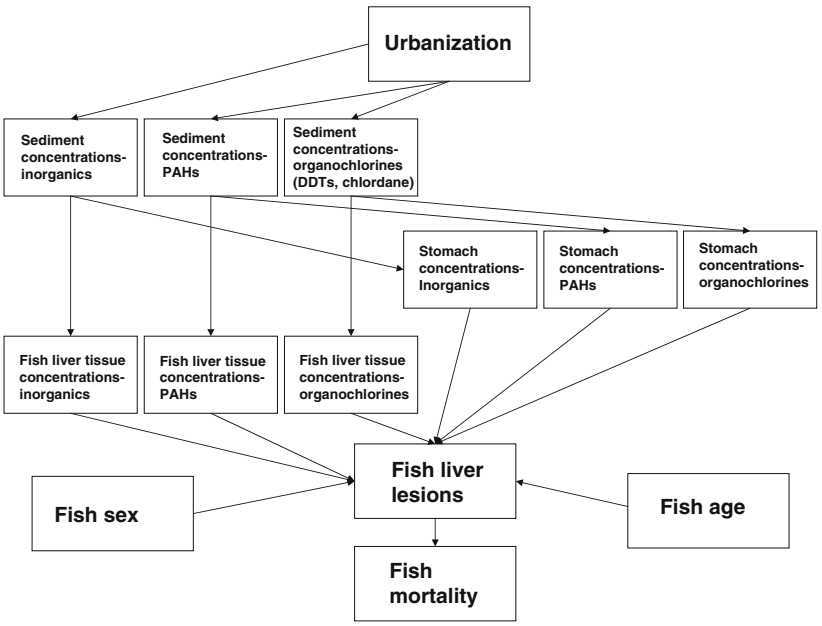

Fig 1 Bayesian belief network structured from the results of Myers et al. (1998). Arrows represent probabilistic dependencies and boxes represent variables

process called inversion, insight can be gained about causes using effects information (e.g., liver cancer prevalence and PAH): experimental observations (effects) may be used to infer the parameters (causes) of a probabilistic model (Robert, 1994). Conditioned on model parameters (causes), the future course a system takes may be described. The ability of Bayesian statistics to account for model uncertainty, by describing parameter uncertainty, makes them a powerful tool for coastal risk assessors. They have some drawbacks in developing uncertain model parameters for risk assessment as discussed by Aven & Kvaløy (2002).

The simple depiction of the nodes in Fig. 1 could be further detailed to reflect understanding of mechanisms and to reduce model uncertainty. For instance, nodes representing the various contaminant concentrations in different environmental compartments could be expanded into more specific chemical categories (e.g., organochlorines could have separate nodes for DDT compounds, dieldrin, chlordanes, PCB congeners) and hepatic lesions could be separated into lesion types (Myers et al., 1994).

Besides these expansions, additional data may cause shifts in the model functionality, making the network development an iterative process. For instance, Bayesian networks could be developed to reflect the statistical relationships for specific fish species because the appearance of lesions and relevance of different chemical classes in biotic or sediment compartments were found to vary among three studied fish species (Myers et al., 1994). These networks could take into account the relative risk for fish lesion prevalence from the output of the stepwise logistic regressions generated by Myers et al. (1990; 1994; 1998). Because Myers et al. (1994) conducted field studies to measure the strength of association of hepatic lesion prevalence in fish with different classes of contaminants, laboratory bioassays might be essential for updating and reinforcing the relationships in the network established from their field surveys. This was acknowledged in Horness et al. (1998), where a National Oceanic and Atmospheric (NOAA) database of sediment contaminant concentrations and hepatic lesion prevalence in English sole was used to set hypothetical

sediment quality criteria. Although clear evidence correlates hepatic neoplasms and sediment contaminant concentrations, the association between tumor prevalence in fish and mortality was variable, and dose-dependence was not clear in some cases (Moore & Myers, 1994; Horness et al., 1998).

For management and predictive purposes, the interconnected nodes of a Bayesian model place multi-layered studies with many candidate causes (e.g., Myers et al. (1994)) under a single modeled framework. In creating a Bayesian network like the one in Fig. 1 for stressor assessment, it is important to include variables that may be changed through future interventions by management as was done in Borsuk et al. (2004). Of course, some variables such as fish age and sex (Fig. 1) are not amenable to manipulation by risk managers, but nonetheless, are pertinent as the state of such variables influence model outcomes. From the results of Myers et al. (1994), the age variable was an important determinant for the presence of some lesions. Sex did not have a significant relationship and a weaker probabilistic dependency would reflect this. Additional nodes with greater relevancy to remedial action by management could include loadings of specific contaminants from point and non-point sources. Resource managers can use such a model to set goals for criteria to protect fish species and can manipulate nodes to determine the implications from management activities or the sensitivity of various components in the model. In Fig. 1, the nodes representing PAH and organochlorine concentrations in sediment would have a stronger correlation with fish hepatic cancer. Variables regulating these concentrations would be the focus of future interventions. Easily implemented dependency analysis can help determine Bayesian network structure and foster implementation with available data sets (e.g., Cheng et al., 2002).

Bayesian networks are also conducive to linking data from separate studies into a cohesive model. In the studies of fish cancer and PAH from Myers et al. (1990; 1994; 1998) and Horness et al. (1998), where several years of data were gathered to identify potential causal agents, the incorporation of prior information is especially suited to this task. The frequentist methods used in Myers et al. (1990; 1994; 1998), and Horness et al. (1998) generally focus on the results from a single relevant study and attempt to objectively analyze those data. In the construction of prior probabilities, Bayesian methods can quantify results from previous experiments and combine them with those from a current experiment, even if they were conducted under dissimilar conditions (Spiegelhalter et al., 1999). For instance, previous studies cited in Myers et al. (1994) found lesions associated with exposure to contaminants including laboratory bioassays with field collected English sole and extracts of PAH from site-specific contaminants. Such information can be useful in establishing a prior distribution that does not rely on subjective information. Subjective probabilities can be used if there is a lack of such knowledge. In situations such as these, Borsuk et al. (2001) and Berry et al. (2003) implemented Bayesian hierarchical methods for cross-system meta-analyses to generate parameter estimates to specific systems that were information poor.

In addition to aiding causal assessment, Bayesian networks can contribute to risk management, remediation, and other aspects of risk assessment. Within a Bayesian network, nodes can reflect processes likely to influence an outcome, or how a management decision might change processes or outcomes. Three types of nodes can be used for these purposes: chance, decision, and utility nodes (Bacon et al., 2002). Chance nodes define processes related to the system; perhaps representing processes or states that affect or are affected by restoration activities. For instance, an assessment-derived conceptual model might be built within a Bayesian network framework that included probabilities associated with pathways of contaminant fate and transport amenable to restoration. Decision nodes are nodes representing potential decisions by the risk manager. Belief networks can contain nodes representing decision variables as well as decision constraints or criteria (Varis, 1997). The iterative procedure used in risk assessments allows for interim goals and changes in the Bayesian network structure can reflect the need for interim goals and decisions as they arise. Also, in the risk

assessment process, assessment endpoints and measurement endpoints can serve as one of many foundations for criteria useful to decision nodes in a network.

Utility nodes are nodes that represent the satisfaction or gain that might accrue from decisions. To normalize cost/benefit variables across aesthetic and economic values, scales and weights can be assigned as was done in Bacon et al. (2002) for land management options and by Fenton and Neil (2001) to increase or decrease the importance of certain utilities. Different remediation techniques have various turn-around times, effectiveness, risks, and costs that can be included in utility nodes for a network. Along with listing the possible remediation alternatives, the strength of each alternative can be factored into the utility nodes of the network.

Concluding remarks

Optimal coastal and estuarine ecological risk assessment requires that the ecological risk assessment process be expanded to allow more integration of potential stressors and to include wider spatial and temporal scales. Fortunately, the means and impetus now exist for this to occur. The informal expert opinion approach, even when guided by sound rules-of-thumb, can be insufficient, as more candidate causes require consideration and wider scales are assessed. Simple Bayesian concepts and tools will be essential to effective risk assessment in the immediate future. The number of publications applying Bayesian statistics has increased in epidemiological and environmental journals, providing more examples to adapt in coastal risk assessments. Increased computation power and the availability of software such as Analytica, Netica (Norsys), or WinBUGS makes implementation of these methods easier. The explicit form and ability to express probabilities for plausible causes will accelerate decision making and remediation because, when probabilities are clear, judgments are more accurate and individuals are more willing to act (Keynes, 1921; Ellsberg, 1961; Chow & Sarin, 2001). Hopefully, application of methods such as those of Borsuk, Stow and Reckhow will become more routine and allow more effective coastal resource decision-making and action.

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