Review of Short Elliott Hendrickson, Inc. and Dames & Moore Ecological Risk Assessments of Contaminated Offshore Sediments in Ashland, Wisconsin

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Summary

This report is an evaluation of two ecological risk assessments (ERA) of contaminated sediments in Lake Superior offshore from Ashland, WI, performed by Short Elliott Hendrickson (SEH) for the Wisconsin Department of Natural Resources, and Dames & Moore (D&M) for Northern States Power. Both ERAs used the same data, collected for the SEH assessment, but arrived at different conclusions. The TOSC program was asked to provide an independent review of the SEH and D&M ERAs, with particular focus on the findings and methodology. In this report, the sources of discrepancies between the two ERAs are outlined. The merit of the conclusions offered by each assessment is examined in the context of the scientific literature pertaining to the contaminants and organisms at the Ashland site. The probable impacts on biota, considering the available data regarding the level of contamination in the sediment and water, are discussed.

The major differences and sources of disagreement between the SEH and D&M risk assessments are outlined below.

1. ERA style – The assessment prepared by SEH more closely adhered to the structure suggested by the U.S. EPA's Guidelines for Ecological Risk Assessment. In the D&M assessment, important sections of the analysis and risk characterization phases were missing.

2. Measurement endpoints - Different models were used to compare sediment PAH concentrations with expected ecological impacts. SEH used Sediment Effects Concentrations derived from *Hyalella azteca* 28-day chronic exposures. D&M used an equilibrium-partitioning model to predict pore water PAH concentrations, and then a quantitative structure activity relationship (QSAR) to estimate an EC25, a concentration they claim is protective of populations.

3. Analysis – SEH characterized exposure by comparing individual sediment PAH concentrations to Sediment Effects Concentrations, then calculated a toxic unit from the summed values. D&M attempted to calculate a hazard quotient based on average sediment concentrations divided by the estimated EC25; however, D&M made several deviations from the model they used, as well as calculation errors. The D&M calculations, when performed as specified by the model from which they were derived, yield a result very similar to that of the SEH analysis.

4. Risk characterization – SEH used a triad sediment approach to evaluate sediment PAH concentrations, a benthic community survey, and laboratory sediment exposures. D&M considered the results of the benthic survey and laboratory exposures inconclusive, and used only sediment PAH concentrations in its risk characterization.

5. Phototoxicity – Because PAHs are known to exhibit enhanced toxicity in sunlight, phototoxicity experiments using contaminated sediment were conducted for the SEH assessment. D&M did not use these results in its assessment.

6. Conclusions – SEH concluded a high risk of detrimental ecological effects to the benthic communities in much of the sediment adjacent to the Ashland waterfront. D&M disputed the SEH conclusions, and predicted a smaller area of contamination requiring remediation.

Based on our evaluation of the two ERAs, it is the opinion of TOSC that the D&M assessment contained errors in calculations in the risk analysis that render its risk characterization invalid, and therefore, the D&M conclusions concerning PAH impacts on aquatic organisms cannot be considered accurate. No major flaws were found with the SEH analysis. Although there was variability between samples taken from the site, the benthic community survey and laboratory experiments in the SEH document demonstrated evidence of ecological impacts. The conclusions of the SEH risk assessment regarding the likely impacts on benthic communities are valid, and recommendations regarding clean up criteria necessary to ameliorate ecological effects are appropriate.

Background

In 1998, Short Elliott Hendrickson, Inc. (SEH), under contract from Wisconsin DNR, completed an ecological risk assessment (ERA) for sediments offshore from the Northern States Power (NSP) property in Ashland, Wisconsin, contaminated with polycyclic aromatic hydrocarbons (PAHs) and volatile organic compounds (VOCs). SEH found:

- The contaminants in sediment offshore from the Ashland site are present at very high concentrations, but are distributed in a heterogeneous (patchy) manner. Concentrations are similar to those found at other contaminated sites where toxicity to aquatic and benthic organisms has been demonstrated;
- There was degradation of benthic communities in contaminated areas, compared to less contaminated areas;
- Laboratory toxicity studies in which various aquatic organisms were exposed to sediment or overlying water indicated growth and survival of most organisms was impaired at sampling locations with high concentrations of PAHs.

Based on the chemical concentrations, the laboratory toxicity tests and the benthic survey, SEH concluded there is a high probability of adverse effects in aquatic organisms from contaminated sediments at the Ashland site. NSP has questioned the methods used in the SEH study and the study's conclusion that nearly 10 acres of sediments should be dredged. According to WI-DNR and NSP representatives, the ecological risk assessment is key to selecting a remedy for the sediment contamination related to the site, the most ecologically significant area of contamination, but the findings of the assessment remain in dispute.

The TOSC program was asked to provide an independent review of the SEH ecological risk assessment, with particular focus on the study's findings and methodology. In addition, project partners asked that we address the major areas of disagreement between the SEH and Dames and Moore reports. The objective of this review was to address questions of *basic science and engineering*—there may be questions over policy or economics (e.g., future use of the Ashland site) that our review does not address.

The scientific merits of the ecological risk assessments conducted by SEH and D&M were evaluated by comparing and assessing the two assessments with respect to:

- Adherence to the Environmental Protection Agency's guidelines for conducting ecological risk assessments
- Accuracy and appropriate use of data from the scientific literature, and their relevance to the Ashland site
- Likely source of contaminants at Ashland
- Assumptions regarding exposure of organisms to contaminants
- Appropriate choice of biological endpoints
- Appropriate use of toxicological models and statistics
- Likelihood of long-term effects on biota
- Validity of conclusions, given the scientific evidence presented

Adherence to the U.S. Environmental Protection Agency's Guidelines for Ecological Risk Assessment

The currently accepted procedure for conducting ecological risk assessments is detailed in the U.S. EPA's *Guidelines for Ecological Risk Assessment* (U.S. Environmental Protection Agency 1998). The EPA *Guidelines* provides a framework that promotes scientifically sound decisions regarding the appropriate choice of endpoints and models, as well as adherence to risk management goals. The *Guidelines* divides the process into three distinct phases: problem formulation, analysis, and risk characterization. Within each stage, there are recommended techniques for formulating conceptual models describing how organisms might be exposed to contaminants, the appropriate choice of endpoints to measure exposure and effects of contaminants, and statistical techniques for analyzing data gathered during the assessment.

In both the SEH and D&M ERAs, there is a statement that the assessment was conducted in accordance with EPA *Guidelines*. Indeed, both documents are similarly structured with respect to the major sections of the risk assessment process. Both documents include problem formulation, analysis, and risk characterization phases. However, the assessments differ in the detail provided. Because the D&M assessment relied on data gathered by SEH for its assessment, in the D&M document certain aspects normally considered essential to a comprehensive ERA were not specifically addressed. For example, the D&M document lacks an assessment of the study design and data quality. Additionally, the D&M ERA did not address alternate exposure endpoints except the analytical chemistry values used to estimate exposure to PAHs. The SEH ERA addressed these issues, and provided much more detail with regard to the choice of effects and exposure endpoints, the conceptual model, the work plan, and sources of uncertainty in the analysis.

Several of the major differences between the two assessments arose because the two assessments used different data sets as input to the analysis phase. SEH conducted a preliminary (or screening level) study in which contaminant concentrations in sediment collected in 1996 were compared to sediment quality guidelines. Subsequently, concentrations of contaminants in sediment collected in 1998, together with lab tests and the benthic diversity survey were used to conduct the baseline assessment. In contrast, D&M used both the 1996 chemistry data (for which there was no organic carbon content data) as well as the 1998 data in its risk assessment, and referred to the lab experiments and benthic survey conducted by SEH as "verification studies," essentially excluding them from the assessment, as those data were not used in the risk decision.

Some discrepancies in document structure between the ERAs exist simply because D&M used data acquired by SEH during its screening level assessment, rather than gather additional data. Many of the sections absent from the assessment correspond to activities not performed by D&M during its assessment (the study design and selection of measurement endpoints, for example). The analysis phase of the D&M document, in particular, does not include details regarding exposure and effects characterizations. Because the SEH assessment detailed these sections, reiteration in the D&M document was not strictly necessary. While a more comprehensive assessment decreases uncertainty during the risk characterization and decision phases, the EPA framework for risk assessment is flexible, in the sense that not all risk assessments require all activities identified by the *Guidelines*.

Problem Formulation

During the problem formulation phase of risk assessment, the nature of the stressor (contaminant) is described, assessment endpoints (exposure, ecosystem characteristics, and effect) are chosen, a conceptual model of exposure is formed, and an analysis plan is laid out. The two risk assessments disagreed on a number of points during this phase.

Source of contaminants

The source of the contaminants detected in sediments offshore from the Ashland Lakefront Property was a point of conflict between the SEH and D&M assessments. In 1996, SEH collected offshore sediments at 80 locations and from several depths and quantified the concentrations of PAHs, VOCs, metals and others. The SEH document stated that the source of the contaminants was not certain, but based on the high concentrations of PAHs and VOCs in the sediments, the most likely source was the former manufactured gas plant (MGP) on the NSP property adjacent to the lakefront. D&M contended the evidence points to other sources of contaminants.

There is convincing evidence supporting the hypothesis of contamination by waste from the former MGP. The high concentration of PAHs detected in sediments (including the non-aqueous phase liquid, or NAPL layer) is consistent with the coal tar waste byproducts from MGPs. The very high concentrations and substantial depth to which PAHs penetrate the sediment (several meters) suggest contamination of the sediments occurred over a substantial length of time, such as during the operation of an MGP over many years. SEH provided evidence of an open sewer terminating offshore, through which wastes may have been directly deposited to sediments. Alternatively, the hydrogeology of the area indicates possible groundwater migration of waste materials from the former MGP to the lakefront.

D&M pointed out the Ashland sediments contain many lower molecular weight compounds, and suggested the PAH profile is more indicative of "fuel oils" than MGP wastes. While a PAH profile may be used as a "fingerprint" to broadly classify the source of PAHs in relatively clean sediment, the PAH profile of weathered MGP waste is virtually indistinguishable from fuel oils or creosote, based on molecular weight (Su et al. 2000). In the absence of an alternate source of PAH contamination such as fuel oil, it is most likely the source of PAHs and VOCs in the offshore sediments at the Ashland site is waste from the former manufactured gas plant.

For the purposes of the risk assessment, both SEH and D&M documents assumed PAHs were the primary contaminants. However, it was suggested by D&M that other compounds not measured by SEH during its analysis may contaminate the sediments, and may have contributed to the observed toxicity in the laboratory studies. Phenols, dioxins, furans, PCBs and pesticides were specifically identified as possible contaminants. However, sediments historically contaminated with PAHs by MGPs and creosote operations do not typically contain appreciable quantities of dioxins, furans, or PCBs. Although these chemicals are widespread contaminants in aquatic environments, they do not usually co-occur with PAHs. Chlorinated hydrocarbons have been found in some sediments historically contaminated by wood preservative operations in which both PAHs and pentachlorophenol were used (McKee et al. 1990). However, these classes of chemicals are rarely found at concentrations capable of inducing acute toxicity (Malins et al. 1985; Ozretich et al. 2000). In the absence of any credible source for these other compounds, the primary contaminants inducing toxicity in Ashland sediments are most likely those associated with MGPs or creosote waste contamination, specifically PAHs, VOCs, and heterocyclics (Mueller et al. 1989; Padma et al. 1998).

Further evidence supporting PAHs as the primary contaminants comes from the toxicity of the sediment itself. The toxicity observed in the laboratory experiments with Ashland sediment is consistent with the known toxicity of PAHs. For example, exposure to sediment spiked with 1.24 mg $gTOC^{-1}$ fluoranthene caused 75% mortality in fathead minnows (Schlueter et al. 2000); a total PAH concentration of 1.0 mg $gTOC^{-1}$ caused approximately the same mortality in fathead minnows exposed to Ashland sediment elutriate (SEH, Figure 16). Other studies have found similar mortality associated with sediments contaminated with mixtures of PAHs from coal tars and creosote (Roberts et al. 1989; Sved et al. 1997; Tagatz et al. 1983). The demonstration of UV-enhanced phototoxicity of Ashland sediments is also strong evidence pointing to PAHs as the predominant toxicant in the sediments.

The presence of other contaminants and their contribution to toxicity, if any, in the Ashland sediments is merely speculative, unless the concentration of each purported chemical (and other environmental contaminants not specifically mentioned) is quantified by chemical analysis. However, this approach is neither practical nor appropriate for environmental risk assessments, in which the integration of all available information regarding the contaminants, including known past and present sources, is favored (U.S. Environmental Protection Agency 1998). The very high levels of PAHs in Ashland sediment, the absence (or low levels) of other contaminants, and the absence of any other credible source of contaminants, suggest PAHs are the predominant contaminant and the most likely source of observed toxicity in Ashland sediments.

The SEH and D&M ERAs disagreed as to the depth of PAH contamination in Ashland sediments, and by inference, the PAH concentrations to which organisms at the surface are exposed. SEH measured concentrations of PAHs (as well as other contaminants) at several discrete depths in sediment cores. There was substantial variability in concentrations from location to location in the bay likely due to the patchy distribution of wood chips. The D&M ERA contended PAH concentrations in the upper six inches in which organisms reside are lower than in deeper sediments, and therefore pose less of a threat to organisms near the surface. SEH interpreted its data to indicate PAH levels are greatest near the surface and decrease with depth. Examination of data from sampling locations for which PAHs were measured both in the upper four feet and in deeper sediments confirms SEH's assertion: concentrations in the 0-4 ft cores exceed those in deeper cores more often. Samples in which PAH concentrations were measured in the upper six inches alone as well as at greater depths in cores from the same sampling location (there were only three) gave less clear results. The heterogeneous distribution of PAHs in the bay makes it impossible to state unequivocally whether PAHs increase or decrease with depth in Ashland sediments. However, given the high concentrations of PAHs measured in the upper six inches alone (SEH Table 5), the probability of contact between organisms residing in surface sediment layers with PAHs is high.

Contaminant fate and transport

Both ERAs identified fate and transport mechanisms for PAHs in aquatic sediments, as well as numerous species likely at risk from exposure to PAHs. The two documents disagreed on a few points regarding exposure of benthic invertebrates to PAHs, uptake of PAHs by aquatic organisms, and natural degradation rates of PAHs.

The D&M ERA suggested some amphipods and chironomids do not come in contact with the sediment itself or the pore water expected to contain the highest concentrations of PAHs, but rather remain above the surface of the sediment in the overlying water where PAH levels are lower. SEH assumed in its ERA that organisms found in Ashland samples penetrate the sediment during feeding activities and burrowing actions, and are exposed to high concentrations of PAHs. The vast majority of the scientific literature supports the SEH position that benthic organisms are likely to be exposed to high PAH levels.

PAHs tend to partition rapidly to sediments (Bestari et al. 1998). However, some desorption from sediment to the aqueous phase can occur, especially from heavily contaminated sediments (Zhang et al. 2000). As pointed out in both ERAs, water circulation and bioturbation can enhance the rate of desorption. The overall contribution of simple dissolution of PAHs to the aqueous phase to exposure of organisms is likely small, as shown by the undetectable aqueous PAH concentrations in the water overlying the Ashland sediments.

The bulk of PAHs enter the water column associated with sediment solids. Chironomids have been shown in laboratory experiments to elevate levels of sediment-associated PAHs in overlying water by disturbing the sediment during burrowing activities (Ciarelli et al. 2000; Clements et al. 1994). D&M stated in its ERA that because PAHs in the water column remain bound to sediments, they are less bioavailable to organisms. However, some organisms such as mollusks filter indiscriminately on suspended particles, and can accumulate high levels of PAHs from suspended sediment (DeLeon et al. 1988; Gewurtz et al. 2000). In addition, some proportion of particles is phytoplankton, which can accumulate PAHs, and are selectively consumed by zooplankton.

PAHs may also be transferred to higher trophic levels by direct ingestion by benthic invertebrates. The literature suggests most benthic organisms accumulate PAHs to some degree when exposed to PAH-contaminated sediments. *Chironomus riparius* exposed in laboratory microcosms to spiked sediments accumulated benzo(a)pyrene or fluoranthene to very high concentrations (Clements et al. 1994). Numerous field studies have demonstrated uptake by isopods (van Hattum et al. 1998), amphipods (Gewurtz et al. 2000; Landrum et al. 1991), mussels (Metcalfe et al. 1997), and to varying degrees by fish (Burkhard and Lukasewycz 2000; Djomo et al. 1996). Organisms such as chironomids that are in close contact with sediment (e.g., during feeding) accumulate higher PAH concentrations than organisms with less contact (Gewurtz et al. 2000). Transfer of PAHs from sediment to benthos to fish through consumption has been demonstrated in laboratory studies (Clements et al. 1994). Therefore, the possibility for transfer of PAHs to higher trophic levels exists. Fish for which benthic invertebrates constitute a large fraction of their diet are at greater risk of exposure to PAHs.

Accumulation and transfer of PAHs to higher trophic levels is primarily a function of the ability of the organism to metabolize PAHs by the mixed function oxygenase (MFO) enzymes. Organisms with a well-developed MFO system, such as fish, rapidly metabolize PAHs; those with poor MFO systems, such as bivalve mollusks, accumulate PAHs (Albers 1995; Elder and Dresler 1988). As most aquatic organisms at higher trophic levels have well developed MFO systems, increases in accumulation through trophic levels do not occur.

Contrary to the suggestion in the D&M ERA, the resuspension of particles in the water column by the burrowing action of benthic invertebrates does not result in a significant degradation of PAHs. While the transfer of PAHs from sediment to biota may lower the sediment concentration somewhat, provided no new input occurs, it is incorrect to refer to this uptake by organisms as “bioremediation.” Metabolic degradation of parent PAHs (i.e., unsubstituted rings) by some bacteria occurs under certain conditions (Lantz et al. 1997; McNally et al. 1998). However, the anoxic state of sediments, the recalcitrant chemical nature of higher molecular weight PAHs, as well as toxicity to bacteria (McConkey et al. 1997) slows bacterial degradation and inhibits remediation of contaminated sediments (Mueller et al. 1989). Fate studies have demonstrated that natural (i.e., not manipulated) bacterial degradation diminishes PAH sediment concentrations very slowly (Bestari et al. 1998). Moreover, given the very high concentrations of PAHs in Ashland sediments after many years, it must be assumed that natural degradation rates are low, and will not result in any significant attenuation of PAHs in the Ashland sediments in the near future.

On the subject of PAH biotransformation, D&M tended to minimize the impacts of PAH metabolites. D&M stated in its ERA that metabolism of PAHs usually results in detoxification, and imply that the cytochrome P450 monooxygenase system in fish efficiently removes PAHs before they enter the blood stream. This statement is incorrect. In most fish, the P450 enzyme system is present in gills and gut tissues, but is concentrated primarily in the liver. D&M stated that fish are deficient in epoxide hydrolase, one of the enzymes involved in carcinogenesis of PAH metabolites, citing a reference to a study of rainbow trout. While apparently true for rainbow trout, this statement does not hold for many other types of fish. Observations of DNA adducts and tumors in fish (including rainbow trout) in the lab and collected from PAH-contaminated sites indicate a strong correlation between PAHs and carcinogenicity (Couch and Harshbarger 1985; Ericson et al. 1999; Metcalfe et al. 1988).

An important aspect of PAH chemistry ignored in both ERAs is photooxidation. There is evidence to suggest that the toxicity of many PAHs is associated with the oxygen-substituted PAHs produced upon exposure to UV light. PAHs taken up into the tissues of aquatic invertebrates and the gills of fish may absorb UVA wavelengths in sunlight and are transformed to oxyPAHs (Mallakin et al. 2000). Some of the oxyPAHs produced in sunlight are many times more toxic than the parent (unsubstituted) PAH (Lampi et al. 2001; Marwood et al. 1999; McConkey et al. 1997). The UVA wavelengths of sunlight responsible for photooxidation of PAHs penetrate to substantial depths in lakes (Williamson et al. 1996). Although UVA penetration in water varies greatly with season and location even within the same lake (Smith et al. 1999), it is possible UVA penetrates to the sediment at Ashland in sufficient quantity to induce photooxidation. This mechanism may play a part in exposure and toxicity of PAHs at Ashland, although the importance relative to other mechanisms is unknown without additional experiments.

A major discrepancy between the ERAs by SEH and D&M is the acknowledgement of the role of UV light in toxicity. SEH identified UV-photoinduced toxicity as a major factor in acute toxicity, and devoted several experiments to determining the relative importance of UV on toxicity of Ashland sediments. In this phase of the risk assessment, D&M did not identify UV as a factor in toxicity, but in the section referred to as "verification studies" D&M attempted to minimize the impacts of UV and discredit the UV studies performed for the SEH assessment. Multiple studies throughout the past decade have demonstrated greatly enhanced PAH toxicity in the presence of UV, both in laboratory studies such as those performed for SEH, and in the field under realistic exposure conditions (Ankley et al. 1995; Diamond et al. 2000; Oris and Giesy 1987). The implications of these studies cannot simply be dismissed, and should have been acknowledged by D&M at this phase of its ERA.

Conceptual model

A conceptual model integrates available information on the stressors, effects, and receptor characteristics into a written and visual representation of the predicted relationships between contaminants and susceptible organisms (U.S. Environmental Protection Agency 1998). The EPA *Guidelines* indicates the exposure profile should explicitly define the exposure, with respect to intensity, frequency and spatial extent. Numerous mathematical models exist for the estimation of uptake rates by organisms, given the potential daily doses, feeding behavior, frequency of exposure, etc. SEH formed a qualitative conceptual model describing susceptible species and exposure pathways, but not explicitly in mathematical terms. The spatial extent of exposure was defined with reasonable precision given the heterogeneity of the PAH distribution in Ashland sediments. The D&M assessment described a simple exposure pathway but did not identify a conceptual model.

The exposure pathway proposed by D&M included reference to the wood chip layer as a possible source of humic acids, which have been shown to ameliorate PAH toxicity by binding organic compounds in the water column (Gensemer et al. 1998; Oris et al. 1990). Humic material is a complex organic compound produced by the breakdown of plant material. It is unknown whether the wood chips could even be a source of humic acids, as degradation of the chips may be retarded by the presence of the coal tars in the sediments immediately below. The presence of wood chips, presumably persisting over many years from abandoned industrial activities adjacent to the bay, suggests this may be the case. Regardless, it is unlikely that degradation of the wood chips resulted in release of humic acids at a concentration required to diminish bioavailability of PAHs, given the high concentration of PAHs in sediments.

Assessment endpoints

At the end of the problem formulation phase, relevant assessment endpoints are chosen. The selection of assessment endpoints is crucial to the outcome of the ERA. However, the identification of suitable endpoints in complex ecosystems is often a contentious issue when multiple stakeholders disagree on the value of endpoints. For a scientifically defensible ERA, endpoints must be both ecologically relevant and susceptible to known stressors (U.S. Environmental Protection Agency 1998). The SEH document identified as its assessment endpoints the protection of several pertinent groups of organisms from PAH impacts. Assessment endpoints were not identified at this phase in the D&M risk assessment.

In the analysis section of the assessment, D&M pointed out that in ecological risk assessments, protection of populations rather than individual organisms is preferred, and identified “protection of populations of benthic invertebrates” as its only assessment endpoint. While the omission of the term “populations” from the SEH assessment endpoints seems to be the basis of the D&M argument for a re-examination of the data and justification for a second risk assessment, it is an argument based on semantics only. Protection of populations is implied and assumed in the SEH endpoints, since SEH did not identify specific organisms (i.e., key species) requiring protection. It is the opinion of TOSC that the SEH assessment endpoints are appropriate.

Analysis

During this phase of the ERA, stressor exposure and effects are quantified using measurement endpoints, which are quantifiable values related to the assessment endpoints. According to the EPA *Guidelines* these can include measures of exposure, effect and ecosystem characteristics. These endpoints are quantitatively compared to characterize both the exposure of organisms to the contaminant and the ecological effects.

SEH chose a number of measurement endpoints to assess exposure and effects on benthic organisms (benthic community survey, toxicity of sediment to benthos in laboratory experiments), zooplankton (laboratory sediment exposures) and fish (laboratory sediment exposures and PAH tissue concentrations). Because D&M chose to use the SEH sediment chemistry data, and took no additional measurements, its measurement endpoints were necessarily restricted to those identified by SEH. The striking discrepancies between the SEH and D&M documents in the analysis section of the assessments are due to the differential use of this data (D&M did not use results of laboratory experiments or benthic surveys in its ERA), and different techniques for comparing exposure and effects.

Exposure characterization

SEH characterized exposure by comparing sediment PAH concentrations to sediment quality benchmarks empirically derived from 28-day toxicity tests using *Hyalella azteca* (HA-28). The database was constructed using data from several contaminated sites in the Great Lakes, as well as other contaminated sites (Ingersoll et al. 1996). These benchmarks represent the concentrations below which toxicity is rarely observed (Effects Range Low, ERL) or above which toxicity is frequently observed (Effects Range Median, ERM). In many of the sediment samples taken during the spring and winter of 1998, PAH concentrations frequently exceeded the ERL, and often the ERM. SEH interpreted these data to indicate exposure of sediment-dwelling organisms to PAHs was likely at these sampling locations.

The approach used by SEH is commonly used in ERAs to characterize exposure. Biological effects-based quality criteria combine field data from multiple contaminated sites in an attempt to classify a site, as to the probable effects on the biota, based on the chemical concentrations in sediment or water. The incidence of biological effects associated with a range of chemical concentrations is determined to establish concentration categories in which, for example, no effect, low effect and severe effects are likely (Long et al. 1995). A variety of biological endpoints have been used as database input, including toxicity from contaminated sediments, toxicity in the laboratory to sediments or water (EC50s), equilibrium partitioning, and benthic community structure (Long et al. 1995). With a large enough database, the probability of certain category of effects (none/minor/severe) at a contaminated site can be estimated with some degree of confidence. During the initial screening level assessment, SEH selected the HA-28 benchmarks from a number of potential benchmarks used by various regulatory agencies, based on several criteria including size of the database, number of freshwater data points, etc. The choice of the HA-28 benchmark seems to have been carefully considered.

D&M criticized the sediment quality endpoints chosen by SEH, on the basis that the sediment quality criteria do not protect populations. D&M's argument that SEH based its conclusions on comparison to benchmark concentrations only is erroneous. SEH used the benchmarks only to assess exposure, and incorporated results of several other lines of evidence during its decision-making process. While D&M disagreed with SEH's choice of assessment and measurement endpoints, D&M chose not to take its own measurements. The absence of additional measurements undermines D&M's criticism of the endpoints selected by SEH.

A major difference between the SEH and D&M assessments was the use of different chemistry measurements – SEH used only the smaller set of measurements taken in 1998 for which organic carbon (OC) measurements were taken; D&M used both the 1998 data as well as the 1996 data, assuming an OC of 4%. However, the use of 1996 data versus 1998 data had no practical impact on the assessment, as D&M made numerous calculation errors in its exposure characterization.

In the D&M assessment, exposure was characterized using a model designed for sediments containing multiple PAHs (•PAH model) incorporating equilibrium partitioning, quantitative structure activity relationships (QSAR), with an additive toxic units approach (Swartz et al. 1995). In this model, organic carbon partitioning coefficients (K_{oc}) are used to estimate interstitial water concentrations from the field sediment concentrations, which are then used in a simple QSAR to generate toxic units (TU) for each PAH, where one TU is equivalent to the concentration of a PAH that kills 50% of the organisms. TUs are calculated as the estimated concentration in the pore water divided by the 10-day LC50, which were estimated using a simple QSAR equation based on empirically-derived LC50s for four aquatic test species exposed to three PAHs (fluoranthene, acenaphthene and phenanthrene). The fraction of organisms likely to experience acute toxicity is predicted from the sum of the TUs for each PAH (•TU), assuming additive toxicity. Models using QSARs and equilibrium partitioning to estimate pore water concentrations, such as the one favored by D&M, are useful for assessing sediment toxicity for chemicals such as PAHs with a narcotic mode of action (Di Toro et al. 2000). However, for a useful analysis the model must be applied properly. There were a number of errors in the application of the model.

Instead of calculating TUs for each PAH in the sediment and summing the TUs, D&M modified the model by calculating an "average" LC50, using a mean octanol-water partitioning coefficient (K_{ow}) for each sampling location. D&M pointed out that the model limits the contribution to toxicity from each PAH according to its water solubility. However, in using an average K_{ow}, D&M disregarded this stipulation of the model. As an example, anthracene from sampling location 2300-1500 was measured at 49,000 $\mu g\ kg^{-1}$ in the sediment, which, according to the Swartz equilibrium-partitioning model represents an interstitial water concentration of 84.35 $\mu g\ L^{-1}$. The model estimates an LC50 of 180 $\mu g\ L^{-1}$ for anthracene, and thus, the anthracene TU for this sampling location would be 84.35 / 180 = 0.469. However, the model places an upper limit of TU = 0.25 for anthracene, based on a maximum aqueous solubility of 44.6 $\mu g\ L^{-1}$. By using an average K_{ow} and ignoring limits on solubility built into the model, D&M overestimated the toxicity of sediments with high PAH concentrations.

D&M further deviated from the Swartz model. Swartz specifies a toxic units approach for comparing the PAH concentrations at each sampling location to the expected LC50, but D&M calculated a hazard quotient (HQ) from the total PAH concentration in each sample divided by an EC25. When dealing with mixtures of chemicals, an approach that acknowledges interactions between chemical species, such as the TU approach, is preferred over an HQ, which is used when considering single contaminants. D&M's rationale for using HQs and EC25 rather than TUs was based on the presumption that protection of fish populations can be achieved at PAH sediment concentrations less than the EC25. The choice of the denominator in the HQ seems somewhat arbitrary. The EC25 was derived from a general equation by Suter (1996; this reference was not identified in the ERA), in which population EC25s (for weight of juvenile fish per egg) were extrapolated from LC50s (96-h in fish) by linear regression for use as an index of population effects where no experimentally derived EC25s were available. Suter has stated this technique has the disadvantages of being both “unconventional” and “not conservative” (Suter 1996).

In addition to problems inherent in D&M’s approach, the HQ values reported by D&M seem to be incorrect. HQs based on the EC25 should be greater than the HQ based on the LC50, since the EC25 is by definition less than the LC50. It is assumed that this error, as well as the negative values sometimes reported for the HQ (which is not mathematically possible), is the result of modifying the Swartz model to use “average” LC50s for each sample. There were errors in the PAH sediment concentration data, octanol-water partition coefficients, molecular weights, as well as in the model calculations. Those calculation errors render invalid the results of the analysis phase of the D&M assessment. The overall consequence is that the conclusions of D&M regarding Ashland sediments toxicity to organisms are inaccurate.

A correct application of the Swartz •PAH model, using the TU terminology, yields •TU values that are similar to those derived by SEH using its HA-28 benchmarks. The similarity of the model with the ERL benchmark was predicted by the authors of the •PAH model (Swartz et al. 1995). For most Ashland sediment samples, •TU’s were greater than 1. At only one sampling location •TU < 0.186, a value below which the Swartz model predicts PAHs are unlikely to contribute to toxicity. At all sampling locations except five, •TU > 3.3, indicating acute toxicity will occur with virtual certainty. Based on those criteria, ***toxicity from PAHs can be expected in most of the sediments offshore from the Ashland Lakefront Property***. Only for those sediments on the outer edges of the contaminant distribution would toxicity to benthic invertebrates be expected to be low.

Ecological effects characterization

D&M excluded from its analysis results from both the benthic surveys and the laboratory toxicity tests conducted by SEH. In the analysis and risk characterization sections of the D&M assessment, sediment PAH concentrations (from 1996 versus 1998 in the SEH assessment) were compared to concentrations expected to cause adverse effects, but the benthic surveys and laboratory toxicity experiments were not included in the decision process. In an addendum referred to as “Verification Studies,” these experiments were re-examined, criticized, and dismissed as inconclusive. TOSC believes the exclusion of these data from the risk characterization compromises the conclusions of the D&M assessment.

SEH characterized ecological effects using three distinct analyses: sediment toxic units calculated from PAH concentrations and ERMs, benthic community indices, and laboratory sediment exposures. In addition to several locations sampled for PAH concentrations only, four locations were sampled for the benthic community survey and laboratory toxicity experiments. Two locations were sampled within the area of the bay indicated by previous analyses to be contaminated, and two locations outside this area were used as reference locations. Paired sampling locations, consisting of woody and non-woody substrate, were appropriately used in an attempt to minimize effects of co-variates such as the wood chips.

For each PAH identified in Ashland sediments, a toxic unit was calculated by dividing the sediment PAH concentration by the HA-28 ERM. The TUs for each PAH were then summed, to provide an overall estimated PAH toxicity of the sample. For sediment samples in which the summed TU was greater than one, adverse effects were expected. SEH calculated TUs based on PAH concentrations normalized to dry weight, and to organic carbon. The TU concept is commonly used with complex mixtures of contaminants with similar modes of action, and is similar to the approach used by D&M. The SEH TUs, in contrast with the D&M TUs, appear to be calculated correctly and applied appropriately. SEH found •TUs greater than one for both the PAH-contaminated sampling locations and the reference wood sampling location.

SEH conducted a survey to compare benthic organisms at each station. A cursory examination of the raw data (counts) suggests a strong correlation between PAH concentration and taxa abundance, with fewer organisms and less diversity in the contaminated samples. SEH calculated several indices related to abundance and species richness. Not surprisingly, there was a strong correlation between sediment PAH concentration and loss of abundance and richness. There was little effect of substrate; abundance and richness were predominantly related to dry weight PAH concentration. These data provide strong evidence supporting SEH's conclusion of detrimental effects of PAHs on benthic communities.

Although the benthic survey was not used in the D&M risk characterization, D&M provided its own interpretation of the results. It was suggested that overlap in the data distribution (due to high variability between the six subsamples at each sampling location) made the results inconclusive. It was also suggested that the presence of the wood chips at the sediment-water interface, rather than PAHs, contributed to differences between samples. None of those arguments to discard the benthic survey data are persuasive. High variability within subsamples is common in field studies and can make interpretation of results challenging. However, variability in the response does not justify discarding data. Field tests should be designed to have sufficient power to detect differences between contaminated and uncontaminated samples. SEH acknowledged this during the planning stage of its ERA, and ensured adequate samples were taken. SEH's plan for collection of samples from both sand and woody substrates was designed to allow the effects from the substrate to be removed. When results were compared for either the wood or sand substrate, there was clearly diminished abundance and richness in contaminated samples in all endpoints except one: total abundance was greater at the contaminated wood sampling location than at the reference wood sampling location. At this location, the abundance was due primarily to high numbers of sowbugs and immature tubificids in some of the subsamples from the contaminated sampling location. In this case, the diminished taxa richness in the contaminated wood samples more accurately reflects the data. Regardless, the variability in the Ashland benthic survey data is not as great as D&M implies, and certainly does not invalidate the results.

The third set of analyses used by SEH to characterize ecological effects were laboratory exposures to sediment using a variety of aquatic species: *Hyalella azteca* (amphipod), *Chironomus tentans* (midge), *Lumbriculus variegatus* (oligochaete), *Daphnia magna* (cladoceran), and *Pimephales promelas* (fathead minnow). Using sediment from the four sampling locations in which PAH concentrations were previously characterized (CW, RW, CS, RS), *H. azteca*, *C. tentans* and *L. variegatus* were exposed directly to sediment, and *D. magna* and *P. promelas* were exposed to sediment elutriate. Standard test methods for exposure to sediment or elutriate were followed. For all species examined, exposure to sediment from the two contaminated sampling locations reduced survival and growth endpoints. When expressed normalized to organic carbon, there was a decrease in survivorship and weight at higher PAH concentrations. Normalization to organic carbon is required when comparing toxicity from PAHs in sediments, to reduce variability from differences in bioavailability.

D&M had two major criticisms of the laboratory experiments reported in the SEH document. The first criticism, that PAH concentrations were not measured in the sediment samples used in the exposures, appears to be unfounded. Although sediment samples were collected on two dates separated by one week, samples for PAH analysis were taken from the same samples used for the laboratory exposures. The second criticism identified by D&M was that the four locations sampled for both the benthic survey and the laboratory experiments were too few to allow delineation of a no-effect concentration. D&M pointed out that PAH concentrations at the contaminated wood location were much greater than the concentrations at the other three sampling locations, and did not allow an adequate concentration-response curve to be constructed. D&M cited these as major flaws in the SEH study design. While these are valid points, it does not appear that the construction of a concentration response relationship from field-collected samples would be a feasible objective. The heterogeneous distribution of PAHs and woody substrate would result in an unmanageable number of samples and toxicity tests. Although a greater number of sampling locations would certainly increase confidence in the results, the approach used by SEH allowed an adequate description of the toxicity associated with contaminated versus reference sediments.

The SEH assessment also reported a series of UV co-exposure experiments. *L. variegatus*, *D. magna* and *P. promelas* were exposed to sediments under normal laboratory light without UV, and then transferred to clean water under light containing UV. Rapid mortality was observed in organisms previously exposed to the sediment from the contaminated wood sampling location. Two notable conclusions can be drawn from these results. The first is that PAHs were almost certainly the contaminant responsible for toxicity in the Ashland sediments; few other chemicals exhibit photoinduced toxicity. Secondly, the elevated toxicity in UV light compared to toxicity without UV suggests organisms exposed to sediments in the field (i.e., in sunlight) likely experience greater toxicity than indicated by the other laboratory exposures. D&M pointed out phototoxicity is not currently examined in standard assays of most regulatory agencies, and attempted to dismiss phototoxicity as having "tenuous applicability to the field." D&M's characterization of the relevance of phototoxicity contradicts numerous studies that have demonstrated convincingly the importance of evaluating phototoxicity of PAHs in the field (Barron et al. 2000; Marwood et al. 1999; Monson et al. 1995; Tagatz et al. 1983).

The absence of phototoxicity from regulatory standards for PAH toxicity assessment is simply a reflection of the recent recognition of impacts in the field.

D&M, in its ERA, suggested phototoxicity in the field may be minimized by the presence of dissolved organic matter (DOM), and attenuation of UV in the water column. Humic acids have been shown in laboratory studies to ameliorate phototoxicity of PAHs dissolved in water (Gensemer et al. 1998; Weinstein and Oris 1999). In bodies of water possessing high levels of DOM, such as small oligotrophic and mesotrophic lakes, DOM may provide some protection from PAHs in the water column. However, as the aqueous phase is only one (probably minor) route of exposure, the attenuation of toxicity of the Ashland sediments by DOM is likely minor. When discussing phototoxicity of PAHs, D&M referred frequently to UVB. It should be noted that PAH phototoxicity also involves UVA wavelengths. As UVA wavelengths constitute a greater fraction of sunlight than UVB, and also penetrate to greater depths in the water column, the induction of phototoxicity in the field from UVA wavelengths is greater than from UVB. Therefore, D&M's statements regarding the attenuation of UVB in the water column by suspended particles have little relevance to the question of phototoxicity. No underwater measurements of light attenuation were taken during sampling. Although water column characteristics vary greatly, typical attenuation coefficients measured at other locations in the Great Lakes (Smith et al. 1999; Williamson et al. 1996) indicate UV levels are likely similar to those used in the laboratory exposures. It is impossible to exactly duplicate all environmental conditions in a laboratory exposure. However, by asserting that the laboratory exposures have no relevance to the Ashland site, D&M has dismissed without justification evidence indicating the possibility of severe phototoxicity to organisms.

Risk Characterization

In the risk characterization phase, exposure and ecological effects are integrated to allow a statement regarding the risk and uncertainty associated with the stressor. Because D&M did not use the results of the benthic survey or the laboratory exposures, its treatment of the risk characterization phase was restricted to estimation of hazard quotients by dividing the sediment PAH concentrations by the EC25s from the Swartz model. D&M characterized the risk to benthic populations as "moderate" in some parts of the contaminated area. However, because of multiple errors in the analysis phase (calculation of EC25s), D&M's risk characterization must be considered flawed as well. Summed TUs correctly calculated as specified by the Swartz model result in an estimation of risk very similar to the SEH risk characterization using the HA-28 SECs.

D&M erroneously stated in its conclusions that SEH based its decision on a comparison of sediment PAH concentrations with benchmark concentrations. While the screening level assessment relied on this data alone to trigger a baseline risk assessment, SEH for its baseline assessment characterized risk using a "sediment triad" approach, in which three separate lines of evidence were examined: sediment benchmarks, benthic community surveys and laboratory toxicity tests. Combined, the corroboration of three different lines of evidence strongly supports SEH's conclusion of "high likelihood of risk" to benthic organisms and juvenile fish. This characterization was based on concentrations of PAHs at many sampling locations in the bay that exceeded the TUs developed from HA-28 ERMs (as well as numerous other sediment effects benchmarks), compared with toxicity observed in the laboratory experiments.

One criticism by D&M of SEH's risk characterization was that SEH's threshold effects concentrations triggering clean up were overly conservative. D&M claimed that on the basis of the toxic units derived from the HA-28 ERMs, ecological effects might be expected to be greater than those observed in the benthic survey and the laboratory exposures. SEH acknowledged in its ERA the HA-28 benchmarks are more conservative than other benchmarks. SEH calculated TUs of 7.1 for the reference sand sampling location, 14.4 for the reference wood sampling location, 119 for the contaminated sand sampling location and 3728 for the contaminated wood sampling location. According to SEH, one toxic unit calculated from HA-28 benchmark concentrations is expected to cause 50% mortality. The toxicity observed in laboratory exposures using other organisms does not support the HA-28 TUs, and therefore, conclusions regarding the toxicity of PAHs based on the *H. azteca* TU values alone would indeed be conservative. However, SEH compared its TUs with observed toxicity in a dilution study with *P. promelas* and determined toxicity occurred at organic carbon normalized TUs above 7, and a 20% impact could be expected between 7 and 15 TUs. The observed impacts on benthic populations and organisms in laboratory studies at each of the four sampling locations seemed to follow the TUs calculated by SEH, in that no effects were found with sediments from the sampling location with a TU of 7, minor impacts were observed at the sampling location with a TU of 14, and severe effects were found at the sampling locations with the highest TUs. Because threshold effect concentrations were calculated using results of the benthic survey and all the laboratory exposures, these values are probably not overly conservative, and likely reflect concentrations above which impacts to benthic populations can be expected.

D&M also pointed out that due to the great difference in sediment PAH concentrations between the highly contaminated wood sampling location and the less contaminated locations, an adequate dose response could not be constructed. Obviously, a greater number of sediment samples, spanning a wide range of PAH concentrations, would reduce the uncertainty associated with SEH's threshold concentrations. However, in combination with effects from the benthic survey and laboratory experiments, there is sufficient evidence that the values calculated by SEH represent reasonable sediment threshold concentrations.

Causality

Causality is established by linking the stressor with observed effects. In ERAs driven by observed fish kills, etc, establishing causality is paramount. However, in this ERA the stressor was identified with considerable confidence, and a feasible exposure route from sediment to biota was established. The EPA *Guidelines* identifies several criteria modified from Koch's Postulates that can be used to establish causality in an ERA:

1. the effect of the toxicant must be regularly associated with exposure to the toxicant;
2. effects must be observed when organisms are exposed to the toxicant under controlled conditions (i.e., laboratory conditions);

3. the same effects found in the laboratory must be observed in the field;
4. indicators of exposure to the toxicant must be found in the affected organisms.

Although each criterion enhances confidence in the conclusions, not all criteria must be satisfied to reasonably establish causality in an ERA. The results of biological surveys can sometimes be inconclusive because populations sampled may be resistant to the contaminant, resulting in different results from lab tests and field surveys. The presence in highly contaminated sediments of *high numbers* but *few species* of benthic organisms suggests this may be the case with the Ashland sediments. The SEH assessment satisfied all except the final criterion. SEH intended to assess PAH residues in benthic organisms collected during the benthic survey, but low numbers of organisms forced this part of the assessment to be abandoned. This is unfortunate, as the presence of PAHs in tissues of benthic organisms would positively verify the source of toxicity in Ashland sediments. Nevertheless, it can be concluded with reasonable confidence from other lines of evidence developed in the SEH assessment, especially the observation of phototoxicity, that PAHs are the source of toxicity.

D&M criticized the "weight of evidence" approach used by SEH to evaluate the likely effects on biota at the Ashland site, suggesting the lack of correspondence between levels of PAHs in sediment and toxicity in some of the laboratory bioassays using this sediment provided evidence contrary to SEH's hypothesis, and required abandonment of SEH's hypothesis that PAHs were causing toxicity. While this approach may be valid for simple testable hypotheses, a more encompassing approach is required for risk assessments. In contrast to laboratory studies, simple statistical tests of null hypotheses (i.e., toxicity or no toxicity) cannot be tested in an ERA, because risk hypotheses are not the same as statistical hypotheses. Because uncertainty is an inherent aspect of risk assessment, an ERA cannot be expected to unequivocally state that adverse effects will, or will not occur in a given ecosystem. Risk assessments in which uncertainty is acknowledged and described are more scientifically defensible and provide risk managers with the information required to make appropriate decisions.

The weight of evidence approach used by SEH, in which causality is supported by various lines of evidence, is more appropriate for field studies (Lowell et al. 2000), and is in fact recommended by the EPA *Guidelines* during the risk characterization phase (U.S. Environmental Protection Agency 1998). (In the *Guidelines*, "lines of evidence" is preferred to "weight of evidence," to favor a more inclusive approach, including qualitative evidence). The weight-of-evidence approach is not designed to provide experimental "proof." Rather, it is a process which can be used to assess the *likelihood* of toxicity. The use of several lines of evidence (positive as well as negative) can increase confidence in the risk decision.

The *Guidelines* suggests several criteria that should be satisfied when using the lines-of-evidence approach, including the abundance and quality of data, the degree of uncertainty, and the pertinence of the evidence to the original risk assessment questions. In our opinion, the SEH assessment adequately addressed uncertainty and relevance in its risk assessment. D&M considered the benthic survey and laboratory assays invalid due to high variability and lack of conclusive evidence of effects between contaminated and reference sampling locations. While the SEH risk characterization and conclusions might have been reinforced by additional data, specifically with respect to the benthic survey, we consider the D&M characterization of the results as overly critical. Taking into consideration the high variability inherent in field studies, SEH ensured there was sufficient statistical power in its sampling design to evaluate impacts on benthic organisms. While variability was high, there were meaningful differences between the contaminated sampling locations and the reference sampling locations. As part of a triad, lines-of-evidence approach, the benthic survey reinforces other data (lab tests, benchmarks) and enhances confidence in the overall conclusions. To reiterate, we consider D&M's decision to exclude results from the benthic survey and the laboratory exposures as seriously compromising its assessment.

In predicting effects on populations not examined in its ERA, SEH made an extrapolation from the laboratory to the field, and from a small scale to a large one. Extrapolations necessarily increase uncertainty in ERAs, but are unavoidable since direct measurements of impacts are not normally feasible. There are many factors that may alter the response in the field, such as resistance to the contaminant, natural fluxes in environmental parameters, and other physical stressors not present in the laboratory. A major source of uncertainty is interspecies variability in response to the toxicant. SEH chose measures of effect that were closely related to the assessment endpoints, so extrapolation to other trophic levels was minimized. Integrated approaches that use multiple lines of evidence, such as the sediment triad used by SEH, provide the least uncertainty.

Probable Impacts on Organisms

Some inferences can be drawn with respect to the probable current and future effects on biota residing in the Ashland sediments, or otherwise contacting the PAH contaminants.

Nature and intensity of effects

The impact of PAHs in Ashland sediments on populations of benthic organisms may be severe. The concentrations of PAHs measured in the sediments are similar to other contaminated sites in which severe impacts have been observed. Because PAHs tend to remain bound to organic sediments, sediment-dwelling or burrowing organisms are at the greatest risk. The limited benthic population survey conducted for the SEH assessment suggests that benthic communities within the highly contaminated sediments have been impacted by PAHs. Aquatic organisms that reside on the sediment surface, or are restricted to the water column only, are likely to receive less exposure to the sediment-bound PAHs, and are therefore at less risk. However, elutriate exposures conducted in the laboratory suggest aqueous concentrations of PAHs liberated from the Ashland sediments are toxic to aquatic organisms. The demonstration of toxicity to all five species examined reflects the broad range of aquatic organisms susceptible to PAH toxicity.

The SEH risk assessment concluded substantial impacts to populations (a 20% reduction in abundance) are likely to occur when sediment PAH concentrations exceed 7 summed toxic units (normalized to organic carbon). The SEH calculations indicate this level is exceeded in most samples from Ashland. It is reasonable to assume that populations of benthic organisms in a large portion of the offshore area have been negatively affected. In many samples, the summed TUs exceed those calculated for the highly contaminated woody location sampled for the benthic survey and laboratory experiments. Based on the results of the benthic survey, it is likely that the benthic community in most of the affected area is severely diminished. It is reasonable to assume that other aquatic organisms not residing in the sediment have been negatively affected as well, although it is more difficult to measure impacts on these populations because they possess the ability to avoid the contaminated areas.

One possible impact of PAHs identified but not examined in these ERAs is the induction of tumors in fish. Most PAHs have been shown to be mutagenic in laboratory studies. In other PAH-contaminated sites that have been examined in more detail, DNA adducts and elevated incidence of (mainly hepatic) neoplasms has been observed in fish (Ericson et al. 1999; Myers et al. 1987; Vogelbein et al. 1990). Although the contaminated area is small compared to other contaminated sites and the frequency of contact with contaminated sediments is relatively low, mutagenicity and carcinogenicity should not be ruled out as potential impacts of PAHs on fish populations.

Spatial scale

The patchy distribution of PAHs makes it difficult to delineate a precise boundary to the contamination. However, the tendency of PAHs to tightly bind to sediment and remain near to their point of introduction rather than migrating great distances suggests the majority of PAH impacts are likely restricted to the area adjacent to the Ashland waterfront and do not extend beyond the enclosed area identified in the ERAs. The incidence of toxicity and effects on benthic populations are associated with areas in which PAH concentrations are greatest. It is unlikely that severe effects of PAHs extend beyond the immediate area of contamination; however, exposure of PAHs to non-resident organisms should not be discounted. Community-level interactions could occur, through consumption of contaminated benthic organisms by transient fish. The result would be greater exposure to PAHs than estimated assuming the resident benthic and aquatic species are the only receivers of PAH exposure.

Potential for natural recovery

The potential for natural recovery at the Ashland site without intervention is minimal. PAHs are metabolized very slowly in sediments (Su et al. 2000). The most compelling evidence that this is the case at Ashland is the fact that even after many years, PAH concentrations remain high in the sediments. Therefore, ecological effects are not likely to be mitigated by time alone. While some species may have achieved a tolerance to PAHs (some benthic species were apparently abundant in highly contaminated areas) the populations of most benthic organisms at Ashland have been reduced by exposure to the PAHs.

Without remediation, future development of the adjacent shoreline may result in greater exposure of PAHs to organisms. Any process that disturbs sediments, such as dredging or boat traffic, may result in the introduction of sediment bound PAHs from deeper sediments to the water column, where they may be transported to other locations, and undergo photochemical reactions that will result in greater toxicity to aquatic organisms (Bonnet et al. 2000).

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