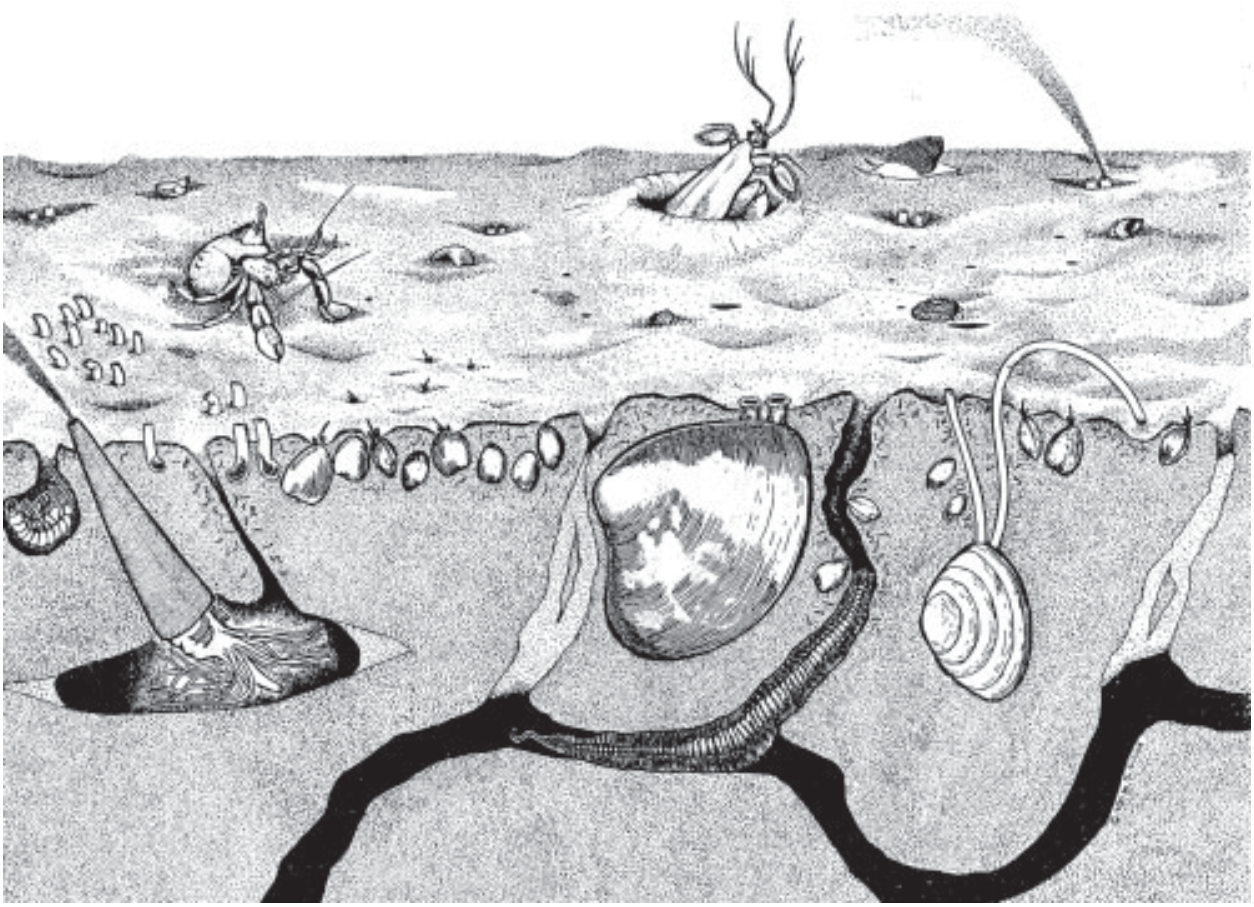
United States Environmental Protection Agency
Office of Research and Development Washington, DC 20460
EPA-600-R-02-013
www.epa.gov

Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures



EPA/600/R-02/013
November 2003

Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures

David J. Hansen
(formerly with U.S. EPA)

Dominic M. DiToro
Univ. Delaware, Newark, DE; HydroQual, Inc.,
Mahwah, NJ

Joy A. McGrath
HydroQual, Inc., Mahwah, NJ

Richard C. Swartz
(formerly with U.S. EPA)

David R. Mount
Robert L. Spehar
National Health and Environmental Effects Research Laboratory
Mid-Continent Ecology Division
Duluth, MN

Robert M. Burgess
National Health and Environmental Effects Research Laboratory
Atlantic Ecology Division
Narragansett, RI

Robert J. Ozretich
National Health and Environmental Effects Research Laboratory
Western Ecology Division
Newport, OR

Heidi E. Bell
Mary C. Reiley
Office of Water, Washington, DC

Tyler K. Linton
Great Lakes Environmental Center
Columbus, OH

U.S. Environmental Protection Agency
Office of Research and Development
National Health and Environmental Effects Research Laboratory
Atlantic Ecology Division, Narragansett, RI
Mid-Continent Ecology Division, Duluth, MN
Western Ecology Division, Newport, OR

Notice

The Office of Research and Development (ORD) has produced this document to provide procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for mixtures of polycyclic aromatic hydrocarbons (PAHs). ESBs may be useful as a complement to existing sediment assessment tools. This document should be cited as:

U.S. EPA. 2003. Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures. EPA-600-R-02-013. Office of Research and Development. Washington, DC 20460

The information in this document has been funded wholly by the U.S. Environmental Protection Agency. It has been subject to the Agency's peer and administrative review, and it has been approved for publication as an EPA document.

Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

Abstract

This equilibrium partitioning sediment benchmark (ESB) document describes procedures to derive concentrations of PAH mixtures in sediment which are protective of the presence of benthic organisms. The equilibrium partitioning (EqP) approach was chosen because it accounts for the varying biological availability of chemicals in different sediments and allows for the incorporation of the appropriate biological effects concentration. This provides for the derivation of benchmarks that are causally linked to the specific chemical, applicable across sediments, and appropriately protective of benthic organisms.

EqP can be used to calculate ESBs for any toxicity endpoint for which there are water-only toxicity data; it is not limited to any specific effect endpoint. In this document, the Final Chronic Value (FCV) for PAHs derived using the National Water Quality Criteria (WQC) Guidelines was used as the toxicity endpoint for this ESB. This value is intended to be the concentration of a chemical in water that is protective of the presence of aquatic life. For this PAH mixtures ESB, narcosis theory was used to (1) demonstrate that the slope of the acute toxicity-octanol water partition coefficient (K_{OW}) relationship was similar across species; (2) normalize the acute toxicity of all PAHs in water to an aquatic species using a reference K_{OW} of 1.0 (where the concentration in water and lipid of the organism would be essentially the same); (3) establish an acute sensitivity ranking for individual species at the K_{OW} of 1.0 and to use the rankings to calculate a Final Acute Value (FAV) following the WQC Guidelines; (4) calculate the final acute-chronic ratio (ACR) from water-only acute and chronic toxicity tests; (5) calculate the Final Chronic Value (FCV) at the reference K_{OW} of 1.0 from the quotient of the FAV and ACR; and (6) to calculate the PAH-specific FCV in µg/L using the FCV at the reference K_{OW} of 1.0, the PAH-specific K_{OW}, the slope of the K_{OW}-K_{OC} relationship and the universal narcotic slope of the K_{OW}-acute toxicity relationship. The EqP approach and the slope of the K_{OW}-K_{OC} relationship was then used to calculate, from the product of the PAH-specific FCV and $K_{OC,}$ the FCV concentration for each specific PAH in sediment ($C_{OC,PAHi,FCVi}$, µg/g organic carbon). Based on this approach, the recommended ESB for total PAH should be the sum of the quotients of a minimum of each of the suggested 34 individual PAHs in a specific sediment divided by the $C_{OC,PAHi,FCVi}$ of that particular PAH. This sum is termed the Equilibrium Partitioning Sediment Benchmark Toxic Unit ($\Sigma ESBTU_{FCV}$). Freshwater or saltwater sediments containing ≤ 1.0 $\Sigma ESBTU_{FCV}$ of the mixture of the 34 PAHs or more PAHs are acceptable for the protection of benthic organisms, and if the $\Sigma ESBTU_{FCV}$ is greater than 1.0, sensitive benthic organisms may be unacceptably affected.

The ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with PAH mixtures or the potential for bioaccumulation and trophic transfer of PAH mixtures to aquatic life, wildlife or humans.

Foreword

Under the Clean Water Act (CWA), the U.S. Environmental Protection Agency (EPA) and the States develop programs for protecting the chemical, physical, and biological integrity of the nation's waters. To support the scientific and technical foundations of the programs, EPA's Office of Research and Development has conducted efforts to develop and publish equilibrium partitioning sediment benchmarks (ESBs) for some of the 65 toxic pollutants or toxic pollutant categories. Toxic contaminants in bottom sediments of the nation's lakes, rivers, wetlands, and coastal waters create the potential for continued environmental degradation even where water column contaminant levels meet applicable water quality standards. In addition, contaminated sediments can lead to water quality impacts, even when direct discharges to the receiving water have ceased.

The ESBs and associated methodology presented in this document provide a means to estimate the concentrations of a substance that may be present in sediment while still protecting benthic organisms from the effects of that substance. These benchmarks are applicable to a variety of freshwater and marine sediments because they are based on the biologically available concentration of the substance in the sediments. These ESBs are intended to provide protection to benthic organisms from direct toxicity due to this substance. In some cases, the additive toxicity for specific classes of toxicants (e.g., metal mixtures or polycyclic aromatic hydrocarbon mixtures) is addressed. The ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with PAH mixtues or the potential for bioaccumulation and trophic transfer of PAH mixtures to aquatic life, wildlife or humans.

ESBs may be useful as a complement to existing sediment assessment tools, to help assess the extent of sediment contamination, to help identify chemicals causing toxicity, and to serve as targets for pollutant loading control measures.

This document provides technical information to EPA Regions, States, the regulated community, and the public. It does not substitute for the CWA or EPA's regulations, nor is it a regulation itself. Thus, it cannot impose legally binding requirements on EPA, States, or the regulated community. EPA and State decisionmakers retain the discretion to adopt approaches on a case-by-case basis that differ from this technical information where appropriate. EPA may change this technical information in the future. This document has been reviewed by EPA's Office of Research and Development (Mid-Continent Ecology Division, Duluth, MN; Atlantic Ecology Division, Narragansett, RI), and approved for publication.

This is contribution AED-02-050 of the Office of Research and Development National Health and Environmental Effects Research Laboratory's Atlantic Ecology Division.

Front cover image provided by Wayne R. Davis and Virginia Lee.

This page is left blank intentionally.

Contents

Notice iii

Abstract iii

Forward iv

Acknowledgments xii

Executive Summary xiv

Glossary xvi

Section 1
Introduction 1-1

1.1 General Information 1-1
1.2 General Information 1-3
1.2.1 PAH Chemistry 1-3
1.2.2 PAH Mixtures 1-5
1.3 Applications of Sediment Benchmarks 1-5
1.4 Data Quality Assurance 1-6
1.5 Overview 1-6

Section 2
Narcosis Theory: Model Development
and Application for PAH Mixtures 2-1

2.1 Section Overview 2-1
2.2 Narcosis Model Background 2-1
2.3 Body Burden Model 2-2
2.4 Target Lipid Model 2-2
2.5 Acute Lethality Database Compilation 2-4
2.5.1 Aqueous Solubility 2-5
2.5.2 Exposure Duration 2-6
2.6 Data Analysis 2-6
2.6.1 Regression Model 2-7
2.6.2 Testing Model Assumptions 2-9
2.6.3 Volume Fraction Hypothesis 2-9
2.6.4 Chemical Classes 2-9
2.6.4.1 Statistical Analysis of K_{OW}-Toxicity Relationships 2-10
2.6.4.2 Standard Errors and Residuals 2-11
2.6.4.3 Chemical Class Corrections 2-11
2.7 Universal Narcosis Slope 2-12

2.8 Comparison to Observed Body Burdens 2-15
2.9 Mixtures and Additivity 2-17
2.10 Aqueous Solubility Constraint 2-17

Section 3
Toxicity of PAHs in Water Exposures
and Derivation of PAH-Specific FCVs 3-1

3.1 Narcosis Theory, EqP Theory and Guidelines:
Derivation of PAH-specific FCVs for Individual PAHs 3-1
3.2 Acute Toxicity of Individual PAHs: Water Exposures 3-8
3.2.1 Acute Toxicity of PAHs 3-8
3.2.2 Acute Values at a K_{OW} of 1.0 3-8
3.3 Applicability of the WQC as the Effects
Concentration for Benthic Organisms 3-9
3.4 Derivation of the FAV at a K_{OW} of 1.0 3-11
3.5 Chronic Toxicity of Individual PAHs: Water Exposures 3-13
3.5.1 Acenaphthene 3-13
3.5.2 Anthracene 3-14
3.5.3 Fluoranthene 3-14
3.5.4 Phenanthrene 3-14
3.5.5 Pyrene 3-14
3.5.6 Naphthalene 3-15
3.5.7 Derivation of the Final Acute Chronic Ratio 3-15
3.6 Derivation of FCVs 3-15
3.6.1 Derivation of the FCV at a K_{OW} of 1.0 3-15
3.6.2 Derivation of the PAH-Specific FCVs 3-15

Section 4
Derivation of the PAH $\Sigma ESBTU_{FCV}$ 4-1

4.1 Derivation of Potencies for Individual PAHs in Sediments ($C_{OC,PAHi,FCVi}$) 4-1
4.2 Derivation of the ESB_{FCV} for PAH Mixtures 4-1
4.3 Aqueous Solubility Constraint 4-2
4.4 Comparison of the $\Sigma ESBTU_{FCV}$ for Mixtures of PAHs in Estuarine Sediments 4-2

Section 5
Actual and Predicted Toxicity of PAH Mixtures
in Sediment Exposures 5-1

5.1 Introduction 5-1
5.2 Spiked Sediment Toxicity Tests 5-1
5.2.1 Interstitial Water Concentrations and Sediment Toxicity:
Relevance to Water-Only Toxicity Tests and WQC FCVs 5-1
5.2.2 Sediment Toxicity: Prediction Using
Water-Only Toxicity and K_{OC} 5-2
5.2.3 Toxicity of Individual PAHs 5-2

5.2.4 Comparison of Sediment Toxicity to $C_{OC,PAHi,FCVi}$ 5-7
5.2.5 PAH Mixtures 5-8
5.2.6 Additivity of PAH Mixtures 5-8
5.2.7 PAH Additivity Demonstrated Using the Universal Narcosis Slope 5-9
5.2.8 Additivity of Mixtures of High K_{OW} PAHs 5-11
5.3 Field Sediments versus ESB_{FCV} for PAH Mixtures 5-17
5.3.1 Toxicity to *R. abronius* of Field Sediments Containing PAH Mixtures vs. ΣPSTUs Derived from Narcosis Theory 5-18
5.3.2 Organism Abundance vs. ESB_{FCV} for PAH Mixtures 5-19

Section 6
Implementation 6-1

6.1 Introduction 6-1
6.2 Defining Total PAH Concentration in Field Collected Sediments 6-1
6.2.1 Introduction 6-2
6.2.2 Data Collection 6-2
6.2.3 Methodology 6-4
6.2.4 Uncertainty in Predicting $\Sigma ESBTU_{FCV,TOT}$ 6-4
6.3 Example Calculation of ESB_{FCV} for PAHs and EqP-based Interpretation 6-7
6.4 Interpreting ESBs in Combination with Toxicity Tests 6-12
6.5 Photo-activation 6-13
6.5.1 Overview 6-13
6.5.2 Implications to Derivation of ESB 6-14
6.6 Teratogenicity and Carcinogenicity 6-14
6.6.1 Calculations 6-16
6.6.2 Critical Sediment Concentrations for Teratogenic and Carcinogenic Effects versus ESBs for PAH Mixtures 6-17
6.7 Equilibrium and ESBs 6-19
6.8 Other Partitioning Phases 6-19
6.8.1 Overview 6-19
6.8.2 Implications to Derivation of ESB 6-20
6.9 Aqueous Solubility Under Non-standard Conditions 6-21

Section 7
Sediment Benchmark Values: Application and Interpretation 7-1

7.1 Benchmark Value 7-1
7.2 Special Considerations 7-1
7.2.1 Fewer than 34 PAHs have been measured 7-1
7.2.2 Interaction of PAHs with UV light 7-2
7.2.3. Influence of soot carbon and coal on PAH partitioning 7-2
7.2.4. Unusual composition of organic carbon 7-2
7.2.5 Presence of additional narcotic compounds 7-2
7.2.6 Site-specific temperature and salinity corrections. 7-3
7.3 Summary 7-3

Section 8
References 8-1

Appendix A A-1

Appendix B B-1

Appendix C C-1

Appendix D D-1

Appendix E E-1

Appendix F F-1

Appendix G G-1

Appendix H H-1

Tables

Table 2-1. Regression results: y-intercepts and chemical class corrections.

Table 2-2. Comparison of body burdens observed in aquatic organisms acutely exposed to narcotic chemicals and body burdens predicted from target lipid narcosis theory.

Table 3-1. Summary of the chronic sensitivity of freshwater and saltwater organisms to PAHs; test-specific data.

Table 3-2. Summary of acute and chronic values, acute-chronic ratios and derivation of the final acute values, final acute-chronic ratios, and final chronic values.

Table 3-3. Results of the approximate randomization (AR) tests for the equality of freshwater and saltwater FAV distributions at a K_{ow} of 1.0 and AR tests for the equality of benthic and combined benthic and water column FAVs for freshwater and saltwater distributions.

Table 3-4. $C_{OC,PAHi,FCVi}$ concentrations and properties required for their derivation.

Table 5-1. Water-only and spiked-sediment LC50 values used to test the applicability of narcosis and EqP theories to the derivation of ESB for PAHs.

Table 5-2. Percent mortality of benthic invertebrates in relation to the sum of the equilibrium partitioning sediment benchmark toxic units (ΣESBTUs) of mixtures of PAHs spiked into sediment.

Table 5-3. Chemicals included in the high K_{OW} PAH mixture experiment conducted by Spehar et al., (*in preparation*).

Table 6-1. Relative distribution of $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,13}$ and $\Sigma ESBTU_{FCV,23}$ for the combined EMAP dataset.

Table 6-2. PAHs measured in various sediment monitoring programs.

Table 6-3. Example calculations of ESBs for PAH mixtures: three sediments.

Table 6-4. Teratogenic and carcinogenic effects of benzo(a)pyrene (BaP) and anthracene on freshwater and saltwater fishes.

Figures

Figure 1-1. Ring structures of representative polycyclic aromatic hydrocarbons. The numbering and lettering system for several PAHs is also given. A, naphthalene; B, 2-methylnaphthalene; C, phenanthrene; D, anthracene; E, benz[*a*]anthracene; F, pyrene; G, benzo[*a*]pyrene; H, benzo[*e*]pyrene; I, fluorene; J, fluoranthene; K, benz[*j*]aceanthrylene = cholanthrene; L, 3-methylcholanthrene; M, chrysene; N, 5-methylchrysene; O, dibenzo[*cd,jk*]pyrene = anthranthrene; P, perylene; Q, benzo[*ghi*]perylene; R, coronene; S, indeno[1,2,3-*cd*]pyrene (from Neff 1979).

Figure 2-1. Schematic diagram of the $\log_{10}$LC50 versus $\log_{10}K_{OW}$ relationship. At $\log_{10}K_{OW} = 0$ ($K_{OW} = 1$), the concentration in water equals the concentration in octanol.

Figure 2-2. Comparisons of (A) $\log_{10}K_{OW}$ predicted by SPARC versus measured $\log_{10}K_{OW}$ using slow stir method and (B) reported $\log_{10}$LC50 values versus the aqueous solubility estimated by SPARC. The diagonal line represents equality.

Figure 2-3. Ratios of (A) 48- to 96-hour LC50 values and (B) 24- to 96-hour LC50 values versus $\log_{10}K_{OW}$. The line in (B) is the regression used to correct the 24-hour LC50 to 96-hour LC50.

Figure 2-4. Log_{10}LC50 versus $\log_{10}K_{OW}$ for the indicated species. The line has a constant slope of −0.945. The y-intercepts vary for each species. Outliers are denoted by a plus symbol (+).

Figure 2-5. Statistical comparison of slopes fitted to individual species to the universal slope of −0.945 showing (A) the probability that the difference occurred by chance (filled bars) and number of data points in the comparison (hatched bars) for each species in the database, and (B) the deviations of the individual estimates from the universal slope.

Figure 2-6. Chemical class comparisons of residuals from the regression grouped by class with (A) mean ± 2 standard errors and (B) chemical class corrections included in the regression.

Figure 2-7. The coefficient of variation of the estimated species-specific body burdens versus (A) the number of data points for that species, (B) the log probability plot of the residuals, and (C) the residuals versus $\log_{10}K_{OW}$.

Figure 2-8. Log_{10}LC50 versus $\log_{10}K_{OW}$ for (A) *Lepomis macrochirus*, (B) *Daphnia pulex*, and (C) *Gambusia affinis*. The line connects the individual estimates of the $\log_{10}$LC50 values, including the chemical class correction.

Figure 2-9. Comparison of target lipid model, line-of-fit and observed LC50 data for individual PAHs, by species.

Figure 2-10. Predicted and observed body burdens for four species.

Figure 2-11. Additivity of type I narcosis toxicity. Comparison of the observed TU concentrations calculated from four studies to the predicted TU of 1.0.

Figure 3-1. Probability distributions of FAV difference statistics to compare water-only toxicity data from (A) freshwater versus saltwater genera and (B) benthic versus WQC.

Figure 3-2. GMAVs at a $\log_{10}K_{OW}$ of 1.0 from water-only acute toxicity tests using freshwater and saltwater genera versus percentage rank of their sensitivity.

Figure 4-1. Probability distribution of the ΣESBTU_{FCV} for PAH mixtures in sediments from individual coastal and estuarine locations in the United States.

Figure 4-2. Probability distribution of the ΣESBTU_{FCV} for PAH mixtures in sediments from all of the coastal and estuarine locations in the United States from Figure 4-1.

Figure 5-1. Percent mortality versus predicted interstitial water toxic units for six chemicals and three sediments per chemical.

Figure 5-2. Percent mortality versus predicted sediment toxic units for seven chemicals and three sediments per chemical.

Figure 5-3. Percent mortality of *Rhepoxynius abronius* in sediments spiked with acenaphthene, phenanthrene, fluoranthene, or pyrene concentrations in sediment normalized to $ESBTU_{FCVi}$.

Figure 5-4. Percentage rank, based on $ESBTU_{FCVi}$, of the sensitivities of genera of benthic organisms from spiked sediment toxicity tests.

Figure 5-5. Mortality of the amphipod, *Rhepoxynius abronius*, from 10-day spiked sediment toxicity tests with four parent PAHs separately (open symbols) and in combination (closed circles) (A) and in tests with sediments from the field (B) versus predicted sediment toxic units (PSTUs). PSTUs are the quotients of the concentration of each PAH measured in sediments from the individual spiked sediment treatments, or individual sediments from the field, divided by the predicted PAH-specific 10-day sediment LC50 values for *R. abronius*. The predicted PAH-specific 10-day sediment LC50 values for *R. abronius*is were calculated using the critical body burden of 15.8 μmol/g octanol and Equation 5-2. PSTUs were summed to obtain the total toxic unit contribution of the mixture of PAHs in spiked or field sediments.

Figure 5-6. Response of *Hyalella azteca* exposed for 10 days under flow-through conditions to sediment spiked with a mixture of high K_{OW} PAH.

Figure 5-7. Response of *Hyalella azteca* exposed for 28 days under flow-through conditions to sediment spiked with a mixture of high K_{OW} PAH.

Figure 5-8. Survival (after 28 days) and growth (after 10 days) of *Hyalella azteca* expressed on the basis of measured PAH concentrations in tissues (lipid normalized).

Figure 5-9. Response of *Hyalella azteca* exposed for 10 days (3 renewals) to sediment spiked with a mixture of high K_{OW} PAH.

Figure 5-10. Response of *Leptocheirus plumulosus* exposed for 10 days under static conditions to sediment spiked with a mixture of high K_{OW} PAH.

Figure 5-11. Amphipod (*Ampelisca abdita*) abundance versus $\Sigma ESBTU_{FCV}$.

Figure 6-1. Comparison of observed $\Sigma ESBTU_{FCV,TOT}$ to observed $\Sigma ESBTU_{FCV,13}$ from (A) 13 PAHs and $\Sigma ESBTU_{FCV,23}$ from (B) 23 PAHs for the combined dataset including U.S. EPA EMAP Louisianian and Carolinian Provinces.

Figure 6-2. Probability distribution of the (A) $\Sigma ESBTU_{FCV,13}$ and (B) $\Sigma ESBTU_{FCV,23}$ values for each sediment from the entire database.

Figure 6-3. BaP concentration of 539 sediment samples from the EMAP and Elliott Bay datasets versus (A) the $\Sigma ESBTU_{FCV}$ values of 34 PAHs and (B) a probability plot of these BaP concentrations at an $\Sigma ESBTU_{FCV}$ =1.0.

Figure 6-4. Anthracene concentration of 539 sediment samples from the EMAP and Elliott Bay datasets versus (A) the $\Sigma ESBTU_{FCV}$ values of 34 PAHs and (B) a probability plot of these anthracene concentrations at an $\Sigma ESBTU_{FCV}$=1.0.

Figure 6-5. Computed solubilities of nine PAHs relative to their 25°C solubilities as a function of temperature.

Acknowledgments

Coauthors

David J. Hansen	Environmental consultant (formerly with U.S. EPA)
Dominic M. Di Toro	Univ. of Delaware, Newark, DE; HydroQual, Inc., Mahwah, NJ
Joy A. McGrath	HydroQual, Inc., Mahwah, NJ
Richard C. Swartz	Environmental consultant (formerly with U.S. EPA)
David R. Mount*	U.S. EPA, NHEERL, Mid-continent Ecology Division, Duluth, MN
Robert L. Spehar	U.S. EPA, NHEERL, Mid-continent Ecology Division, Duluth, MN
Robert M. Burgess*,**	U.S. EPA, NHEERL, Atlantic Ecology Division, Narragansett, RI
Robert J. Ozretich	U.S. EPA, NHEERL, Western Ecology Division, Newport, OR
Heidi E. Bell*	U.S. EPA, Office of Water, Washington, DC
Mary C. Reiley	U.S. EPA, Office of Water, Washington, DC
Tyler K. Linton	Great Lakes Environmental Center, Columbus, OH

Significant Contributors to the Development of the Approach and Supporting Science

David J. Hansen	Environmental consultant (formerly with U.S. EPA)
Dominic M. Di Toro	Univ. of Delaware, Newark, DE; HydroQual, Inc., Mahwah, NJ
Joy A. McGrath	HydroQual, Inc., Mahwah, NJ
Richard C. Swartz	Environmental consultant (formerly with U.S. EPA)
David R. Mount	U.S. EPA, NHEERL, Mid-continent Ecology Division, Duluth, MN
Robert M. Burgess	U.S. EPA, NHEERL, Atlantic Ecology Division, Narragansett, RI
Robert J. Ozretich	U.S. EPA, NHEERL, Pacific Ecology Division, Newport, OR
Robert L. Spehar	U.S. EPA, NHEERL, Mid-continent Ecology Division, Duluth, MN

Technical Support and Document Review

Walter J. Berry	U.S. EPA, NHEERL, Atlantic Ecology Division, Naragansett, RI
Lawrence P. Burkhard	U.S. EPA, NHEERL, Mid-continent Ecology Division, Duluth, MN
Patricia DeCastro	Computer Sciences Corporation, Narragansett, RI
Edward Dettmann	U.S. EPA, NHEERL, Atlantic Ecology Division, Naragansett, RI

Jim Dwyer	U.S. F&WS Columbia, MO
Phillip M.Gschwend	Massachusetts Institute of Technology, Cambridge, MA
Susan B. Kane-Driscoll	Menzie-Cura & Associates, Inc., Chelmsford, MA
Peter F. Landrum	NOAA, Great Lakes Research Laboratory, Ann Arbor, MI
Richard J. Pruell	U.S. EPA, NHEERL, Atlantic Ecology Division, Narragansett, RI
Marc Tuchman	U.S. EPA, Great Lakes National Program Office, Chicago, IL

*Principal U.S. EPA contacts

** Series editor

Executive Summary

This equilibrium partitioning sediment benchmark (ESB) document recommends an approach for summing the toxicological contributions of mixtures of 34 polynuclear aromatic hydrocarbons (PAHs) in sediments to determine if their concentrations in any specific sediment would be protective of benthic organisms from their direct toxicity. The combination of the equilibrium partitioning (EqP), narcosis theory, and additivity provide the technical foundation for this benchmark. These approaches were required because PAHs occur in sediments in a variety of proportions as mixtures and can be expected to act jointly under a common mode of action. Therefore, their combined toxicological contributions must be predicted on a sediment-specific basis. This overall approach provides for the derivation of this Tier 1 ESB that is causally linked to the specific mixtures of PAHs in a sediment, yet is applicable across sediments and appropriately protective of benthic organisms.

EqP theory holds that a nonionic chemical in sediment partitions between sediment organic carbon, interstitial (pore) water and benthic organisms. At equilibrium, if the concentration in any one phase is known, then the concentrations in the others can be predicted. The ratio of the concentration in water to the concentration in sediment organic carbon is termed the organic carbon partition coefficient (K_{OC}), which is a constant for each chemical. The ESB Technical Basis Document (U.S. EPA, 2003a) demonstrates that biological responses of benthic organisms to nonionic organic chemicals in sediments are different across sediments when the sediment concentrations are expressed on a dry weight basis, but similar when expressed on a μg chemical/g organic carbon basis ($\mu g/g_{OC}$). Similar responses were also observed across sediments when interstitial water concentrations were used to normalize biological availability. The Technical Basis Document (U.S. EPA, 2003a) further demonstrates that if the effect concentration in water is known, the effect concentration in sediments on a $\mu g/g_{OC}$ basis can be accurately predicted by multiplying the effect concentration in water by the chemical's K_{OC}.

EqP can be used to calculate ESBs for any toxicity endpoint for which there are water-only toxicity data; it is not limited to any specific effect endpoint. In this document, the Final Chronic Value (FCV) for PAHs derived using the National Water Quality Criteria (WQC) Guidelines (Stephan et al., 1985) was used as the toxicity endpoint for this ESB. This value is intended to be the concentration of a chemical in water that is protective of the presence of aquatic life. For this PAH mixtures ESB, narcosis theory was used to (1) demonstrate that the slope of the acute toxicity-octanol water partition coefficient (K_{OW}) relationship was similar across species; (2) normalize the acute toxicity of all PAHs in water to an aquatic species using a reference K_{OW} of 1.0 (where the concentration in water and lipid of the organism would be essentially the same); (3) establish an acute sensitivity ranking for individual species at the K_{OW} of 1.0 and to use the rankings to calculate a Final Acute Value (FAV) following the WQC Guidelines (Stephan et al., 1985); (4) calculate the final acute-chronic ratio (ACR) from water-only acute and chronic toxicity tests; (5) calculate the Final Chronic Value (FCV) at the reference K_{OW} of 1.0 from the quotient of the FAV and ACR; and (6) to calculate the PAH-specific FCV in μg/L using the FCV at the reference K_{OW} of 1.0, the PAH-specific K_{OW} and the universal narcotic slope of the acute-K_{OW} toxicity relationship. The EqP approach and the slope of the K_{OW}-K_{OC} relationship was then used to calculate, from the product of the PAH-specific FCV and $K_{OC,}$ the FCV concentration for each specific PAH in sediment ($C_{OC,PAHi,FCVi}$, μg/g organic carbon).

Importantly, because PAHs occur in sediments as mixtures and their toxicities in water, tissues, or sediments are additive or nearly additive, their combined toxicities must be considered so that the benchmark is appropriately protective. For this reason, the combined toxicological contributions of the PAH mixture must be used. In this document, the 34 PAHs monitored in the EMAP program are used to derive a concentration of "total PAH." Many monitoring and assessment efforts measure a smaller group of PAHs, such as 13 or 23 PAHs. While adjustment factors have been calculated to relate these smaller subsets to the expected concentration of the 34 PAHs, their imprecision precludes their use in critical sediment assessments. Therefore, this document recommends that the ESB for total PAH should be the sum of the quotients of the concentrations of each of the 34 individual PAHs in a specific sediment divided by the $C_{OC,PAHi,FCVi}$ of that particular PAH. This sum is termed the Equilibrium Partitioning Sediment Benchmark Toxic Unit ($\Sigma ESBTU_{FCV}$), which is based on the FCV. Freshwater or saltwater sediments containing ≤ 1.0 $\Sigma ESBTU_{FCV}$ of the mixture of the 34 PAHs or more PAHs are acceptable for the protection of benthic organisms, and if the $\Sigma ESBTU_{FCV}$ is greater than 1.0, sensitive benthic organisms may be unacceptably affected. This provides for the derivation of a benchmark that is causally linked to the specific mixtures of PAHs in a sediment, applicable across sediment types, and appropriately protective of benthic organisms. A sediment-specific site assessment would provide further information on PAH bioavailability and the expectation of toxicity relative to the $\Sigma ESBTU_{FCV}$ and associated uncertainty.

These ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with PAHs or the potential for bioaccumulation and trophic transfer of PAHs to aquatic life, wildlife or humans. Consistent with the recommendations of EPA's Science Advisory Board, publication of these documents does not imply the use of ESBs as stand-alone, pass-fail criteria for all applications; rather, ESB exceedances could be used to trigger the collection of additional assessment data. ESBs apply only to sediments having $\geq 0.2\%$ organic carbon by dry weight.

Tier 1 and Tier 2 ESB values were developed to reflect differing degrees of data availability and uncertainty. Tier 1 ESBs have been derived for polycyclic aromatic hydrocarbon (PAH) mixtures in this document, and for the nonionic organic insecticides endrin and dieldrin, and metal mixtures in U.S. EPA (2003c,d,e). Tier 2 ESBs are reported in U.S. EPA (2003f).

Glossary of Abbreviations

ACR	Acute-Chronic Ratio
AR	Approximate Randomization
ASTM	American Society for Testing and Materials
BaP	Benzo[a]pyrene
BCF	Bioconcentration factor
C_{d}	Freely-dissolved interstitial water concentration of contaminant
C_{L}	Chemical concentration in target lipid
C_{*L}	Critical body burden in the target lipid fraction of the organism
C_{OC}	Chemical concentration in sediments on an organic carbon basis
$C_{OC,PAHi}$	PAH-specific chemical concentration in sediment on an organic carbon basis
$C_{octanol}$	Chemical concentration in octanol
C_{Org}	Chemical concentration in the organism
C_{*Org}	Critical body burden in the organism
C_{IW}	Total interstitial water concentration of contaminant
$C_{OC,PAHi,FCVi}$	Effect concentration of a PAH in sediment on an organic carbon basis calculated from the product of its FCV and K_{OC}
$C_{OC,PAHi,Rhepox,LC50}$	Sediment LC50 concentration on an organic carbon basis for a specific PAH for *Rhepoxinus* calculated from the product of its LC50 value at a K_{OW} of 1.0 and K_{OC}
$C_{OC,PAHi,Maxi}$	Maximum solubility limited PAH concentration in sediment on an organic carbon basis
CV	Coefficient of Variation
CWA	Clean Water Act
DOC	Dissolved Organic Carbon
EC50	Concentration affecting 50% of the test organisms
EMAP	Environmental Monitoring and Assessment Program
EPA	United States Environmental Protection Agency
EqP	Equilibrium partitioning
ESB	Equilibrium Partitioning Sediment Benchmark(s)

$ESBTU_{FCVi}$	Equilibrium Partitioning Sediment Benchmark Toxic Unit for PAH_i based on the FCV
$ESBTU_{Rhepox}$	Equilibrium Partitioning Sediment Benchmark Toxic Unit for PAH_i based on the LC50 of *Rhepoxynius abronius*.
$\Sigma ESBTU_{FCV}$	Sum of Equilibrium Partitioning Sediment Benchmark Toxic Units, where the units are based on FCV values
f_{Lipid}	Fraction of lipid in the organism
f_{OC}	Fraction of organic carbon in sediment
f_{SC}	Fraction of soot carbon in sediment
FACR	Final Acute-Chronic Ratio
FAV	Final Acute Value
FCV	Final Chronic Value
GMAV	Genus Mean Acute Value
IWTU	Interstitial Water Toxic Unit
$IWTU_{FCV}$	Interstitial water toxic unit calculated by dividing the dissolved interstitial water concentration by the FCV
K_{DOC}	Dissolved organic carbon: water partition coefficient
K_{LW}	Lipid: water partition coefficient
K_{OC}	Organic carbon: water partition coefficient
K_{OW}	Octanol: water partition coefficient
K_P	Sediment: water partition coefficient
K_S	Setschenow constant
K_{SC}	Soot carbon: water partition coefficient
LC50	Concentration estimated to be lethal to 50 % of the test organisms within a specified time period
LFER	Linear free energy relationship
MV	Molar Volume
NA	Not Applicable, Not Available
NAPL	Non-aqueous Phase Liquid
ND	Not Determined, Not Detected
NOAA	National Oceanographic and Atmospheric Administration

NOEC	No Observed Effect Concentration
NTU	Narcotic Toxic Units
OEC	Observable Effect Concentration
PAH	Polycyclic aromatic hydrocarbon
PAH_{OC}	Organic carbon-normalized PAH concentration in sediment
PCB	Polychlorinated Biphenyl
POC	Particulate Organic Carbon
PSTU	Predicted Sediment Toxic Units
QSAR	Quantitative Structure Activity Relationship
REMAP	Regional Environmental Monitoring and Assessment Program
S	Aqueous Solubility
SAB	U.S. EPA Science Advisory Board
SCV	Secondary Chronic Value
SE	Standard Error
SMAV	Species Mean Acute Value
SPARC	SPARC Performs Automated Reasoning in Chemistry
TOC	Total Organic Carbon
TU	Toxic Unit
WQC	Water Quality Criteria
$WQCTU_{FCVi}$	Water Quality Criteria Toxic Unit based on the FCV

Section 1

Introduction

1.1 General Information

Toxic pollutants in bottom sediments of the Nation's lakes, rivers, wetlands, estuaries, and marine coastal waters create the potential for continued environmental degradation even where water column concentrations comply with established WQC. In addition, contaminated sediments can be a significant pollutant source that may cause water quality degradation to persist, even when other pollutant sources are stopped (Larsson, 1985; Salomons et al., 1987; Burgess and Scott, 1992). The absence of defensible equilibrium partitioning sediment benchmarks (ESBs) make it difficult to accurately assess the extent of the ecological risks of contaminated sediments and to identify, prioritize, and implement appropriate cleanup activities and source controls (U.S. EPA 1997a, b, c).

As a result of the need for a procedure to assist regulatory agencies in making decisions concerning contaminated sediment problems, the U.S. Environmental Protection Agency (EPA) Office of Science and Technology, Health and Ecological Criteria Division (OST/HECD) and Office of Research and Development National Health and Environmental Effects Research Laboratory (ORD/NHEERL) established a research team to review alternative approaches (Chapman, 1987). All of the approaches reviewed had both strengths and weaknesses, and no single approach was found to be applicable for the derivation of benchmarks in all situations (U.S. EPA, 1989, 1992). The equilibrium partitioning (EqP) approach was selected for nonionic organic chemicals because it presented the greatest promise for generating defensible, national, numeric chemical-specific benchmarks applicable across a broad range of sediment types. The three principal observations that underlie the EqP approach to establishing sediment benchmarks are as follows:

1. The concentrations of nonionic organic chemicals in sediments, expressed on an organic carbon basis, and in interstitial waters correlate to observed biological effects on sediment-dwelling organisms across a range of sediments.

2. Partitioning models can relate sediment concentrations for nonionic organic chemicals on an organic carbon basis to freely-dissolved concentrations in interstitial water.

3. The distribution of sensitivities of benthic organisms to chemicals is similar to that of water column organisms; thus, the currently established water quality criteria (WQC) final chronic values (FCV) or secondary chronic values (SCV) can be used to define the acceptable effects concentration of a chemical freely-dissolved in interstitial water.

The EqP approach, therefore, assumes that (1) the partitioning of the chemical between sediment organic carbon and interstitial water is at or near equilibrium; (2) the concentration in either phase can be predicted using appropriate partition coefficients and the measured concentration in the other phase (assuming the freely-dissolved interstitial water concentration can be accurately measured); (3) organisms receive equivalent exposure from water-only exposures or from any equilibrated phase: either from interstitial water via respiration, from sediment via ingestion or other sediment-integument exchange, or from a mixture of exposure routes; (4) for nonionic chemicals, effect concentrations in sediments on an organic carbon basis can be predicted using the organic carbon partition coefficient (K_{OC}) and effects concentrations in water; (5) the FCV or SCV concentration is an appropriate effects concentration for freely-dissolved chemical in interstitial water; and (6) ESBs derived as the product of the K_{OC} and FCV are protective of benthic organisms. ESB concentrations presented in this document are expressed as µg chemical/g sediment organic carbon ($\mu g/g_{OC}$) and not on an interstitial water basis because (1) interstitial water

is difficult to sample and (2) significant amounts of the dissolved chemical may be associated with dissolved organic carbon; thus, total concentrations in interstitial water may overestimate exposure.

Sediment benchmarks generated using the EqP approach are suitable for use in providing technical information to regulatory agencies because they are:

1. Numeric values
2. Chemical specific
3. Applicable to most sediments
4. Predictive of biological effects
5. Protective of benthic organisms

ESBs are derived using the available scientific data to assess the likelihood of significant environmental effects to benthic organisms from chemicals in sediments in the same way that the WQC are derived using the available scientific data to assess the likelihood of significant environmental effects to organisms in the water column. As such, ESBs are intended to protect benthic organisms from the effects of chemicals associated with sediments and, therefore, only apply to sediments permanently inundated with water, to intertidal sediment, and to sediments inundated periodically for durations sufficient to permit development of benthic assemblages. ESBs should not be applied to occasionally inundated soils containing terrestrial organisms, nor should they be used to address the question of possible contamination of upper trophic level organisms or the synergistic, additive, or antagonistic effects of multiple chemicals. The application of ESBs under these conditions may result in values lower or higher than those presented in this document.

ESB values presented herein are the concentrations of PAH mixtures in sediment that will not adversely affect most benthic organisms. It is recognized that these ESB values may need to be adjusted to account for future data. They may also need to be adjusted because of site-specific considerations. For example, in spill situations, where chemical equilibrium between water and sediments has not yet been reached, sediment chemical concentrations less than an ESB may pose risks to benthic organisms. This is because for spills, disequilibrium concentrations in interstitial and overlying water may be proportionally higher relative to sediment concentrations. In systems where biogenic organic carbon dominates, research has shown that the source or "quality" of total organic carbon (TOC) in natural sediments does not affect chemical binding when sediment toxicity was measured as a function of TOC concentration (DeWitt et al., 1992). K_{OC}s have also been demonstrated to not vary in gradients of chemicals across estuarine sediments (Burgess et al., 2000a). However, in systems where other forms of carbon are present at elevated levels, the source or 'quality' of TOC may affect chemical binding despite expressing toxicity as a function of TOC concentration. At some sites, concentrations in excess of an ESB may not pose risks to benthic organisms because the compounds are partitioned to or a component of a particulate phase such as soot carbon or coal or exceed solubility such as in the case of undissolved oil or chemical (e.g. conditions at a manufactured gas plant site). In these situations, an ESB would be overly protective of benthic organisms and should not be used unless modified using the procedures outlined in "Procedures for the Derivation of Site-Specific Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms" (U.S. EPA, 2003b). If the organic carbon has a low capacity (e.g., hair, sawdust, hide), an ESB would be underprotective. An ESB may also be underprotective where the toxicity of other chemicals are additive with an ESB chemical or where species of unusual sensitivity occur at the site.

This document presents the theoretical basis and the supporting data relevant to the derivation of ESBs for PAH mixtures. The data that support the EqP approach for deriving ESBs for nonionic organic chemicals are reviewed by Di Toro et al. (1991) and EPA (U.S. EPA, 2003a). Before proceeding through the following text, tables, and calculations, the reader should also consider reviewing Stephan et al. (1985).

1.2 General Information: PAH Mixtures

The EPA developed ESBs for metal mixtures (Cd, Cu, Pb, Ni, Ag, Zn) (U.S. EPA 2003c) and the insecticides endrin and dieldrin (U.S. EPA 2003d,e) and proposed ESBs for the individual polycyclic aromatic hydrocarbons (PAHs) acenaphthene, fluoranthene and phenanthrene (U.S. EPA 1993a,b,c). Because PAHs occur in the environment as mixtures, rather than single chemicals, ESBs for individual PAHs have the potential to be substantially under-protective because they do not account for other co-occurring PAHs. This ESB for PAH mixtures replaces the earlier draft individual PAH documents.

Numerous efforts have previously sought to address and estimate the toxicity of PAH mixtures in sediments (Barrick et al., 1988; Long and Morgan, 1991; PTI Environmental Services, 1991; Long et al., 1995; Swartz et al., 1995; Ingersoll et al., 1996; MacDonald et al., 1996, 2000; Cubbage et al., 1997; Di Toro and McGrath, 2000; Di Toro et al., 2000; Ozretich et al., 1997, 2000). The resultant sediment benchmarks have engendered considerable controversy over such issues as the correlative versus causal relations between dry weight sediment chemistry and biological effects, the bioavailability of sediment contaminants, the effects of covarying chemicals and mixtures, and ecological relevance. Overviews of the various approaches are useful (Mount et al., 2003; Swartz et al., 1999). The use of sediment benchmarks derived in a variety of ways must be linked to the derivation procedure and specific intent of the methodology. The U. S. EPA research team has concluded, based upon additional investigation, that recommendation of sediment benchmarks for PAHs based on EqP, narcosis theory and additivity was necessary to resolve outstanding issues related to causality. Sediment benchmarks for mixtures of PAHs that are derived using these approaches are adequately protective of benthic organisms, as well as ecologically relevant.

The ΣPAH model developed by Swartz et al. (1995) and based upon a combination of the EqP approach, quantitative structure activity relationships (QSAR), narcosis theory, and additivity models provided initial insight into a technical approach for resolving these complexities. This EqP-based ΣPAH model provides a method to address causality, account for bioavailability, consider mixtures, and predict toxicity and ecological effects. The most significant contribution to the development of the scientific basis for deriving ESBs for PAH mixtures is described by Di Toro et al. (2000) and Di Toro and McGrath (2000). This pioneering research in developing a methodology for deriving ESBs for mixtures of narcotic chemicals and PAHs forms major portions of this document.

1.2.1 PAH Chemistry

Portions of the following overview of PAH chemistry are directly, or in part from, Neff's1979 classic book "Polycyclic Aromatic Hydrocarbons in the Aquatic Environment" and to a lesser extent Schwarzenbach et al. (1993). PAHs are composed of two or more fused aromatic or benzene rings. Two aromatic rings are fused when a pair of carbon atoms is shared. The resulting structure is a molecule with all carbon and hydrogen atoms lying in a single plane. Naphthalene ($C_{10}H_8$), which consists of two fused aromatic rings, is the lowest molecular weight PAH. The ultimate fused-ring aromatic system is graphite, an allotropic form of elemental carbon. Of primary environmental concern are mobile compounds ranging in molecular weight from naphthalene ($C_{10}H_8$, molecular weight 128.17) to coronene ($C_{24}H_{12}$, molecular weight 300.36). Within this range is an extremely large number of PAHs differing in the number and positions of aromatic rings and in the number, chemistry, and position of substituents on the ring system. Figure 1-1 presents a selection of PAH structures.

Physical and chemical characteristics of PAHs vary in a more or less regular fashion with molecular weight. Resistance to oxidation and reduction tends to decrease with increasing molecular weight. Vapor pressure and aqueous solubility decrease almost logarithmically with increasing molecular weight. As a consequence of these differences, PAHs of different molecular weights vary substantially in their behavior and

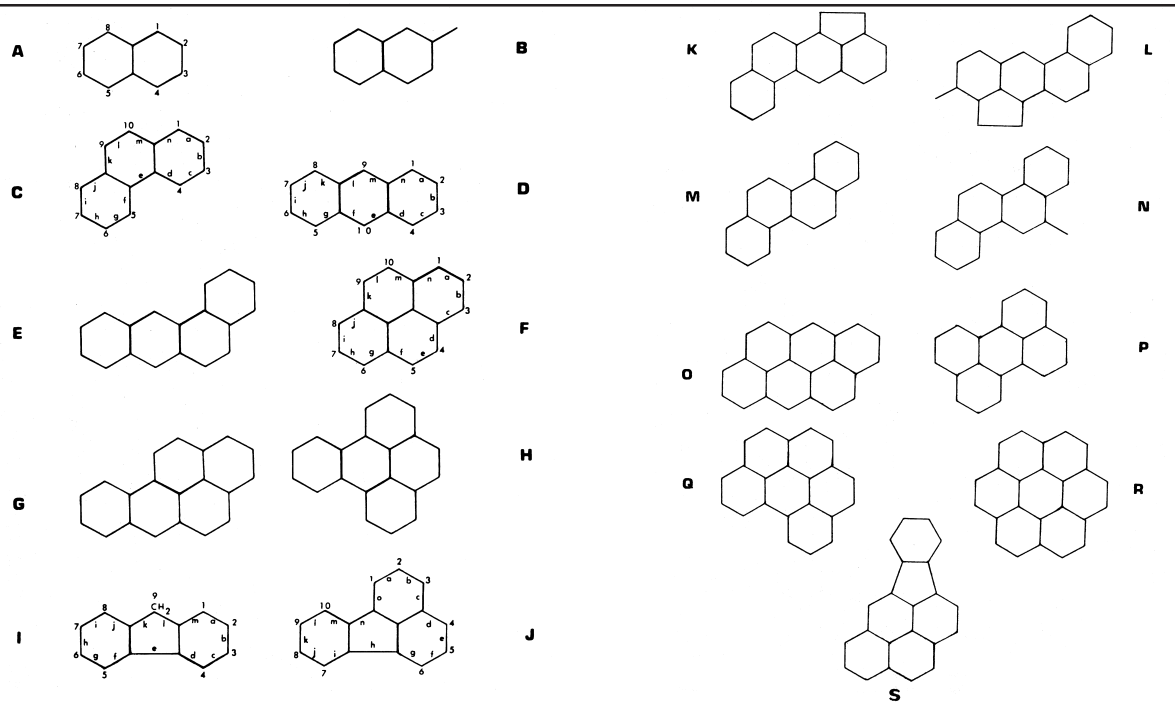


Figure 1-1. Ring structures of representative polycyclic aromatic hydrocarbons. The numbering and lettering system for several PAHs is also given. A, naphthalene; B, 2-methylnaphthalene; C, phenanthrene; D, anthracene; E, benz[*a*]anthracene; F, pyrene; G, benzo[*a*]pyrene; H, benzo[*e*]pyrene; I, fluorene; J, fluoranthene; K, benz[*j*]aceanthrylene = cholanthrene; L, 3-methylcholanthrene; M, chrysene; N, 5-methylchrysene; O, dibenzo[*cd,jk*]pyrene = anthranthrene; P, perylene; Q, benzo[*ghi*]perylene; R, coronene; S, indeno[1,2,3-*cd*]pyrene (from Neff 1979).

distribution in the environment and their toxic effects. PAHs undergo three types of chemical reactions characteristic of aromatic hydrocarbons - electrophilic substitution, oxidation, and reduction. Oxidation and reduction reactions destroy the aromatic character of the affected benzene ring but electrophilic substitution does not.

Several systems of nomenclature have been used to describe PAH ring structures. Nomenclature used in this document is that adopted by the International Union of Pure and applied Chemistry (IUPAC) and described in detail in *The Ring Index* (Patterson et al., 1960).

As noted above, there is an extremely large number of possible PAH structures (>10,000). Later in this document, 34 PAH structures (specific non-alkylated compounds and generic alkylated forms) are identified as representing a minimum for 'total PAHs'. It is recognized that this subset of all possible PAHs is not complete; however, the 34 PAHs identified are the ones that are generally most abundant and commonly measured as part of environmental monitoring programs. As analytical techniques improve, the number of PAHs composing 'total PAHs' will most certainly increase and users of this document are encouraged to include newly quantified PAHs in the derivation of benchmark values assuming good supporting data are available (e.g., K_{OW}s, solubilities).

PAHs found in aquatic environments originate from three possible sources: pyrogenic, petrogenic and diagenic. Pyrogenic PAHs result from the incomplete but high temperature, short-duration combustion of organic matter including fossil fuels and biomass (Neff 1979; Meyers and Ishiwatari 1993). These pyrogenic PAHs are believed to form from the breakdown or 'cracking' of organic matter to lower molecular weight radicals during pyrolysis, followed by rapid reassembly into non-alkylated PAH structures (Neff 1979). Petrogenic PAHs are created by diagenic processes at relatively low temperatures over geologic time scales, leading to the formation of petroleum and other fossil fuels containing PAHs (Meyers and

Ishiwatari 1993; Boehm et al., 2001). PAHs formed at relatively low temperatures (~150 °C) over long periods of time will be primarily alkylated molecules. The alkylated structure of petrogenic PAHs reflects the ancient plant material from which the compounds formed (Neff 1979). Diagenic PAHs refer to PAHs from biogenic precursors, like plant terpenes, leading to the formation of compounds such as retene and derivatives of phenanthrene and chrysene (Hites et al., 1980; Meyers and Ishiwatari 1993; Silliman et al., 1998). Perylene is another common diagenic PAH. Although its exact formation process remains unclear, an anaerobic process appears to be involved (Gschwend et al., 1983; Venkatesan 1988; Silliman et al., 1998). While diagenic PAHs are frequently found at background levels in recent sediments (i.e., deposited over the last 150 years), they often dominate the assemblage of PAHs present in older sediments deposited before human industrial activity (Gschwend et al., 1983). A potential fourth source of PAHs is biogenic; that is, purely from bacteria, fungi, plants or animals in sedimentary environments without any contributions from diagenic processes. However, attempts to produce biogenic PAHs have arguably failed, indicating this source is not significant (Hase and Hites 1976; Neff 1979).

The majority of PAHs found in aquatic environments originate from pyrogenic sources (Blumer 1976; Suess 1976; Hites et al., 1977; LaFlamme and Hites, 1978; NRC 1985; Wu et al., 2001). However, petrogenic PAHs do also occur alone or in combination with pyrogenic PAHs (Lake et al., 1979; Wakeham et al., 1980; NRC 1985; Gschwend and Hites 1981; Readman et al., 1992). In general, petrogenic PAHs appear to be associated with local or point sources, such as refineries and other petroleum industries, and adjacent to roads and navigational routes. This contrasts with the distribution of pyrogenic PAHs, which occur on a broader geographic scale. These distribution are also affected by the relative persistence of pyrogenic and petrogenic PAHs in the environment. As compared to petrogenic PAHs, pyrogenic PAHs are found more extensively in the sediment core record and appear to be less vulnerable to biotic and abiotic degradation (Burgess et al., 2003). Finally, diagenic PAHs occur at background levels although anthropogenic sources (e.g., perylene) can contribute to these types of PAHs.

1.2.2 PAH Mixtures

Unlike most other organic chemicals in the environment, PAHs are not released in a 'pure' or well-characterized form. Rather, because PAHs consist of thousands of structures originating from at least three sources, they always occur in the environment as complex mixtures (Burgess et al., 2003). As discussed above, pyrogenic PAHs, although not generally alkylated, are produced as mixtures of parent PAHs based on the conditions of their combustive formation (e.g., temperature, presence of oxygen, original organic matter). Similarly, the composition of petrogenic PAHs is a function of the diagenic conditions under which the original organic matter was exposed for thousands of years (e.g., pressure, temperature). Of course, human industrial practices convert some crude petrogenic PAH mixtures into more purified forms (e.g., fuel oils, creosote). These purified forms also contain complex mixtures of PAH molecules. As a consequence of these factors, when PAHs are released into the aquatic environment from the burning of fossil fuels and biomass, discharge of industrial chemicals, and transport of petroleum products they eventually accumulate in the sediments as complex mixtures (Neff 1979).

1.3 Application of Sediment Benchmarks

ESBs as presented in this document are meant to be used with direct toxicity testing of sediments as a method of sediment evaluation, assuming the toxicity testing species is sensitive to the chemical(s) of interest. They provide a chemical-by-chemical specification of sediment concentrations protective of benthic aquatic life. The EqP method should be applicable to nonionic organic chemicals with a K_{OW} above 3.0.

For the toxic chemicals addressed by the ESB documents Tier 1 (U.S. EPA, 2003c, d, e, and this document) and Tier 2 (U.S. EPA, 2003f) values

were developed to reflect the differing degrees of data availability and uncertainty. Tier 1 ESBs are more scientifically rigorous and data intensive than Tier 2 ESBs. The minimum requirements to derive a Tier 1 ESB include: (1) Each chemical's organic carbon-water partition coefficient (K_{OC}) is derived from the octanol-water partition coefficient (K_{OW}) obtained using the SPARC (SPARC Performs Automated Reasoning in Chemistry) model (Karickhoff et al., 1991) and the K_{OW}-K_{OC} relationship from Di Toro et al. (1991). This K_{OC} has been demonstrated to predict the toxic sediment concentration from the toxic water concentration with less uncertainty than K_{OC} values derived using other methods. (2) The FCV is updated using the most recent toxicological information and is based on the National WQC Guidelines (Stephan et al., 1985). (3) EqP-confirmation tests are conducted to demonstrate the accuracy of the EqP prediction that the K_{OC} multiplied by the effect concentration from a water-only toxicity test predicts the effect concentration from sediment tests (Swartz, 1991a; DeWitt et al., 1992). Using these specifications, Tier 1 ESBs have been derived for PAH mixtures in this document, metals mixtures (U.S. EPA, 2003c) and, the nonionic organic insecticides endrin and dieldrin (U.S. EPA, 2003d, e). In comparison, the minimum requirements for a Tier 2 ESB (U.S. EPA, 2003f) are less rigorous: (1) The K_{OW} for the chemical that is used to derive the K_{OC} can be from slow-stir, generator column, shake flask, SPARC or other sources (e.g., Site 2001). (2) FCVs can be from published or draft WQC documents, the Great Lakes Initiative or developed from AQUIRE. Secondary chronic values (SCV) from Suter and Mabrey (1994) or other effects concentrations from water-only toxicity tests can be used. (3) EqP cconfirmation tests are recommended, but are not required for the development of Tier 2 ESBs. Because of these lesser requirements, there is greater uncertainty in the EqP prediction of the sediment effect concentration from the water-only effect concentration, and in the level of protection afforded by Tier 2 ESBs. Examples of Tier 2 ESBs for nonionic organic chemicals are found in U.S. EPA (2003f).

1.4 Data Quality Assurance

All data used to derive the FCV used to calculate the ESB for PAHs from water-only toxicity tests were obtained from a comprehensive literature search completed in 1995. Discussions in other sections of this document utilized literature obtained up to 2003. Data were evaluated for acceptability using the procedures in the Stephan et al. (1985): *Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses*. Data not meeting the criteria for acceptability were rejected. All calculations were made using the procedures in Stephan et al. (1985). All data and intermediate values are presented in tables or appendices in the document. Four significant figures were used in intermediate calculations to limit the effect of rounding error, and are not intended to indicate the true level of precision. The document was reviewed as part of a formal peer review and all original data were made available as part of the review process. Any errors of omission or calculation discovered during the peer review process were corrected. The document was revised according to the comments of peer reviewers and additional scientific literature and significant data identified by reviewers were incorporated into the document. Hard copies of peer-review comments and responses to these comments are available from the ORD/NHEERL Atlantic Ecology Division - Narragansett, Rhode Island. Hard copies of all literature cited in this document reside at ORD/ NHEERL Atlantic Ecology Division - Narragansett, Rhode Island.

1.5 Overview

This document presents the theoretical basis and supporting data relevant to the derivation of ESBs for mixtures of PAHs.

Section 2 of this document "Narcosis Theory: Model Development and Application for PAH Mixtures" contains an analysis of the narcosis and EqP models to demonstrate the scientific basis for the derivation of WQC and ESBs for mixtures of narcotic chemicals, including PAHs. Data are

presented that demonstrate that the toxicity of narcotic chemicals when based on concentration in water increase with their K_{OW} and that the slope of the K_{OW}-toxicity relationship is not different across species. The universal slope of this relationship (-0.945) is applicable for all narcotic chemical classes, whereas the intercept is chemical class-specific. The intercept of this slope at a K_{OW} of 1.0 predicts the tissue effect concentration. The toxicities of mixtures of narcotic chemicals in water are shown to be approximately additive, thus the toxic unit concept is applicable to mixtures. The toxicities of narcotic chemicals are shown to be limited by their solubilities in water, hence their toxicities in sediments are limited.

Section 3 of this document "Toxicity of PAHs in Water Exposure and Derivation of PAH-specific FCVs" presents an analysis of acute and chronic water-only toxicity data for freshwater and saltwater aquatic organisms exposed to individual PAHs. It examines (1) the relative sensitivities of freshwater and saltwater organisms to determine if separate FCVs are required, and (2) the relative sensitivities of benthic organisms and organisms used to derive WQC to determine if the WQC FCV should be based only on benthic organisms. These data are used with the narcosis model presented in Section 2, the EqP approach (U.S. EPA, 2003a), and the U.S. EPA National WQC (Stephan et al., 1985) to derive the FCV for individual PAHs (PAH-specific FCV).

Section 4 "Derivation of PAH $\Sigma ESBTU_{FCV}$" contains the approach used for deriving the ΣESBs for mixtures of PAHs. The $C_{OC,PAHi,FCVi}$ is derived for each individual PAH as the product of the PAH-specific FCV and the respective K_{OC} value as recommended by the EqP approach. The use of the $C_{OC,PAHiFCVi}$ value for individual PAHs is inappropriate for use as the ESB because PAHs occur as mixtures. The toxicities of mixtures of narcotic chemicals has been shown to be approximately additive, therefore, combined toxic contributions of all PAHs in the mixture can be determined by summing the quotients of the concentration of each PAH in the sediment divided by its $C_{OC,PAHi,FCVi}$ to determine the sum of these Equilibrium Partitioning Sediment Benchmark Toxic Units ($\Sigma ESBTU_{FCV}$). If the $\Sigma ESBTU_{FCV}$ is ≤1.0, the sediment benchmark for the PAH mixture is not exceeded and the PAH concentration in the sediment is protective of benthic organisms. If the $\Sigma ESBTU_{FCV}$ exceeds 1.0, the sediment benchmark for the PAH mixture is exceeded and sensitive benthic organisms may be affected by the PAHs. The $\Sigma ESBTU_{FCV}$ is derived for PAH mixtures in sediments from national monitoring programs to reveal the incidence of sediment benchmark exceedences.

Section 5 "Actual and Predicted Toxicity of PAH Mixtures in Sediment Exposures" examines the applicability of the EqP methodology for $C_{OC,PAHi,FCVi}$ and ESB derivation. The $C_{OC,PAHi,FCVi}$ and ESB are compared to (1) databases of observed sediment toxicity, and (2) amphipod abundance in sediments from the field where PAHs are the probable contaminants of concern.

Section 6 "Implementation" defines the PAHs to which the ESB apply. An example calculation is provided to explain the conversion of concentrations of individual PAHs on a dry weight basis into the benchmark. The photo-activation of PAHs in UV sunlight and teratogenicity and carcinogenicity of certain PAHs in the mixture are examined. The importance of equilibrium and the partitioning of PAHs to other organic carbon phases (e.g., soot and coal) is described. An approach for calculating PAH solubilities for temperatures or salinities at specific sites is provided.

Section 7 "Sediment Benchmark Values: Application and Interpretation" presents the sediment benchmark values and lists several factors to consider when applying and interpreting these values.

Section 8 "References" lists references cited in all sections of this document.

Appendices provide supplementary tabulated information.

Section 2

Narcosis Theory: Model Development and Application for PAH Mixtures

2.1 Section Overview

This section of the ESB document presents a model of the toxicity of narcotic chemicals to aquatic organisms that is applicable to the derivation of WQC and ESBs for mixtures of narcotic chemicals, including PAHs. Both the model and this section of the document are largely excerpted from the publications of Di Toro et al. (2000) and Di Toro and McGrath (2000) which should be consulted for information on components of the overall model that are not included in this ESB document. The narcosis model includes a scientific analysis of the toxicities of narcotic chemicals fundamental to the derivation of WQC and ESBs for their mixtures. The ESB for PAH mixtures described in Section 4 of this document is derived using this model and toxicity data exclusively for PAHs (see Section 3).

The narcosis model is used to describe the toxicity of all type I narcotic chemicals. Since PAHs are expected to be type I narcotic chemicals (Hermens, 1989; Verhaar et al., 1992), the toxicological principles that apply to them should be more accurately characterized by an analysis of the principles that apply to narcotic chemicals overall. Model development utilizes a database of LC50 values comprising 156 chemicals and 33 aquatic species, including fish, amphibians, arthropods, molluscs, annelids, coelenterates and echinoderms. The analysis detailed in this section is used to demonstrate that (1) the toxicities of narcotic chemicals, and therefore PAHs, are dependant on the chemical's K_{OW}; (2) the slope of the K_{OW}-toxicity relationship is the same for all species of aquatic organisms and classes of narcotic chemicals with the intercepts being species and chemical class-specific; (3) the species-specific LC50 values normalized to a K_{OW} =1.0 permit ranking of species sensitivities and are equivalent to the body burden LC50 on a lipid basis; and (4) the toxicities of mixtures of narcotic chemicals are additive.

The analysis of narcotic chemical toxicity data presented in this section shows that the proposed model accounts for the variations in toxicity due to differing species sensitivities and chemical differences. The model is based on the idea that the target lipid is the site of action in the organism. Further, it is assumed that target lipid has the same lipid-octanol linear free energy relationship for all species. This implies that the $\log_{10}$LC50 vs $\log_{10}K_{OW}$ slope is the same for all species. However, individual species may have varying target lipid body burdens of narcotic chemicals that cause mortality. The target lipid LC50 body burdens estimated by extrapolations from the water-only acute toxicity data and K_{OW} values are compared to measured total lipid LC50 body burdens for five species. They are essentially equal, indicating that the extrapolation in the model is appropriate for estimation of LC50 body burdens, i.e., that the target lipid concentration is equal to the total extracted lipid concentration. The precise relationship between target lipid and octanol is established.

2.2 Narcosis Model Background

A comprehensive model of type I narcosis chemicals which considers multiple species has been presented by Van Leeuwen et al. (1992).

They developed QSARs for individual species and performed species sensitivity analysis. The analysis and model presented below and in Di Toro et al. (2000) and that of Van Leeuwen et al. (1992) are similar. The key differences in the Di Toro et al. (2000) model are the use of a single universal slope for the $\log_{10}$LC50 versus $\log_{10}K_{OW}$ QSAR for all the species, the inclusion of corrections for chemical classes, such as PAHs, that are slightly more potent than reference narcotics, and the interpretation of the y-intercepts as the species-specific critical body burdens for narcosis mortality.

2.3 Body Burden Model

The initial QSAR models for narcotic toxicity relied on correlations of $\log_{10}$LC50 and $\log_{10}K_{OW}$ (Konemann, 1981; Veith et al., 1983). An interesting and important interpretation of this inverse relationship which relates the toxicity to chemical body burden has been presented by McCarty et al. (1991), and proceeds as follows. The relationship between the LC50 (mmol/L) and K_{OW} for the narcosis LC50 for fish is approximately

$$\log_{10}LC50 \simeq -\log_{10}K_{OW} + 1.7 \qquad (2\text{-}1)$$

For each LC50, a fish body burden, on a wet weight basis, corresponding to narcosis mortality can be computed using a bioconcentration factor BCF (L/kg) which is defined as the ratio of the chemical concentration in the organism C_{Org} (mmol/kg wet weight) to the chemical concentration dissolved in the water C_d (mmol/L)

$$BCF = \frac{C_{Org}}{C_d} \qquad (2\text{-}2)$$

Using the BCF, the organism concentration corresponding to the LC50, which is referred to as the critical body burden and denoted by C^*_{Org}, can be computed using

$$C^*_{Org} = BCF \times LC50 \qquad (2\text{-}3)$$

The superscript * indicates that it is a critical body burden corresponding to the LC50. The BCF also varies with K_{OW}. For fish, the relationship is

$$\log_{10}BCF \simeq \log_{10}K_{OW} - 1.3 \qquad (2\text{-}4)$$

Therefore, the critical body burden corresponding to the LC50 for fish narcosis can be computed using the narcosis LC50 and the BCF

$$\log_{10} C^*_{Org} = \log_{10}BCF + \log_{10}LC50$$

$$\simeq \log_{10}K_{OW} - 1.3 - \log_{10}K_{OW} + 1.7$$

$$\simeq 0.4 \qquad (2\text{-}5)$$

or

$$C^*_{Org} \simeq 2.5\ \mu\text{mol/g wet wt} \qquad (2\text{-}6)$$

Thus, McCarty et al. (1991) rationalized the relationship between LC50 values and K_{OW} by suggesting that mortality is caused as a result of a constant body burden of the narcotic chemical.

The reason the critical body burden is a constant concentration for all the narcotic chemicals represented by the narcosis LC50 is a consequence of the unity slopes for $\log_{10}K_{OW}$ in Equations 2-1 and 2-4. For example, if the fraction of lipid in the fish is ass umed to be 5% ($f_{Lipid} = 0.05$), then the critical body burden in the lipid fraction of the fish is

$$C^*_L = \frac{C^*_{Org}}{f_{Lipid}} = \simeq 50\ \mu\text{mol / g lipid} \qquad (2\text{-}7)$$

which is the estimate of the chemical concentration in the lipid of these fish that causes 50 % mortality. The model presented below is an extension of this idea.

2.4 Target Lipid Model

The body burden model relates the narcosis concentration to a whole body concentration using a BCF. If different species are tested, then species-specific BCFs and lipid concentrations would be required to convert the LC50 concentration to a body burden for each species. A more direct approach is to relate narcotic

lethality to the concentration of the chemical in the target tissue of the organism, rather than to the concentration in the whole organism. If the partitioning into the target tissue is independent of species, then the need for species-specific BCFs is obviated. The identity of the target tissue is still being debated (Abernethy et al., 1988; Franks and Lieb, 1990), but we assume that the target is a lipid fraction of the organism. Hence the name, target lipid.

The target lipid model is based on the assumption that mortality occurs when the chemical concentration in the target lipid reaches a threshold concentration. This threshold is assumed to be species-specific rather than a universal constant that is applicable to all organisms (e.g., 50 µmol/g lipid, see Equation 2-7). The formulation follows the body burden model (McCarty et al., 1991). The target lipid-water partition coefficient K_{LW} (L/kg lipid) is defined as the ratio of chemical concentration in target lipid, C_L (µmol/g lipid = mmol/kg lipid), to the freely-dissolved aqueous concentration C_d, (mmol/L)

$$K_{LW} = \frac{C_L}{C_d} \qquad (2\text{-}8)$$

This equation can be used to compute the chemical concentration in the target lipid phase producing narcotic mortality, i.e., the critical body burden in the lipid fraction C_L^*, when the chemical concentration in the water phase is equal to the LC50

$$C_L^* = K_{LW} \times LC50 \qquad (2\text{-}9)$$

Assuming the narcosis hypothesis is true, i.e., that 50% mortality occurs if any narcotic chemical reaches the concentration C_L^*, then the LC50 for any chemical can be calculated using the same critical target lipid concentration C_L^* and the chemical-specific target lipid-water partition coefficient

$$LC50 = \frac{C_L^*}{K_{LW}} \qquad (2\text{-}10)$$

or

$$\log_{10}LC50 = \log_{10}C_L^* - \log_{10}K_{LW} \qquad (2\text{-}11)$$

The problem is determining the K_{LW} for narcotic chemicals. It is commonly observed for many classes of organic molecules that the logarithms of the partition coefficient between two liquids are related by a straight line (Leo, 1972). For target lipid and octanol, the relationship would be

$$\log_{10}K_{LW} = a_0 + a_1 \log_{10}K_{OW} \qquad (2\text{-}12)$$

Such a relationship is called a linear free energy relationship (LFER) (Leo et al., 1971; Brezonik, 1994). Combining Equations 2-11 and 2-12 yields the following linear relationship between $\log_{10}LC50$ and $\log_{10}K_{OW}$

$$\log_{10}LC50 = \log_{10}C_L^* - a_0 - a_1 \log_{10}K_{OW} \qquad (2\text{-}13)$$

where $\log_{10}C_L^* - a_0$ is the y intercept and $-a_1$ is the slope of the line.

This derivation produces the linear relationship between $\log_{10}LC50$ and $\log_{10}K_{OW}$ which is found experimentally (see, for example, Table 6 in Hermens et al., 1984)

$$\log_{10}LC50 = m \log_{10}K_{OW} + b \qquad (2\text{-}14)$$

where m and b are the slope and intercept of the regression, respectively. In addition, it identifies the meanings of the parameters of the regression line. The slope of the line m is the negative of the slope of the LFER between target lipid and octanol, a_1. The intercept of the regression $b = \log_{10}C_L^* - a_0$ is composed of two parameters: C_L^* is the target lipid concentration at narcosis mortality, and a_0 is the constant in Equation 2-12.

The difference between the target lipid model and the McCarty et al. (1991) body burden model is that for the latter, the coefficients a_0 and a_1 for fish are assumed to be known: $a_0 = -1.3$ and $a_1 = 1.0$. It is interesting to examine the consequences of a similar assumption applied to the target lipid model. If it is assumed that the partitioning of narcotic chemicals in lipid and octanol are equal, i.e., that lipid is octanol, a common first approximation, then $a_1 = 1$ and $a_0 = 0$ and the y-intercept becomes

$$b = \log_{10}C_L^* \qquad (2\text{-}15)$$

which is the target-lipid concentration producing 50% narcosis mortality.

This result can be understood by examining Figure 2-l. The y-intercept b is the LC50 value for a chemical with a $\log_{10}K_{OW} = 0$ or $K_{OW} = 1$. The K_{OW} is the ratio of the chemical's concentration in octanol to its concentration in water. Hence, for this hypothetical chemical (an example would be 2-chloroethanol for which $\log_{10}K_{OW} = -0.048$l 0 the chemical's concentration in water is equal to its concentration in octanol. However, if the K_{LW} equals the K_{OW}, i.e., lipid is octanol, then its concentration in water must be equal to its concentration in the target lipid of the organism. Therefore, the y-intercept is the target lipid phase concentration at which 50% mortality is observed. That is

$$LC50|_{K_{ow}=1} = b = C^*_{octanol} = C^*_L \qquad (2\text{-}16)$$

Note that this interpretation is true only if $a_0 = 0$ (see Equation 2-13).

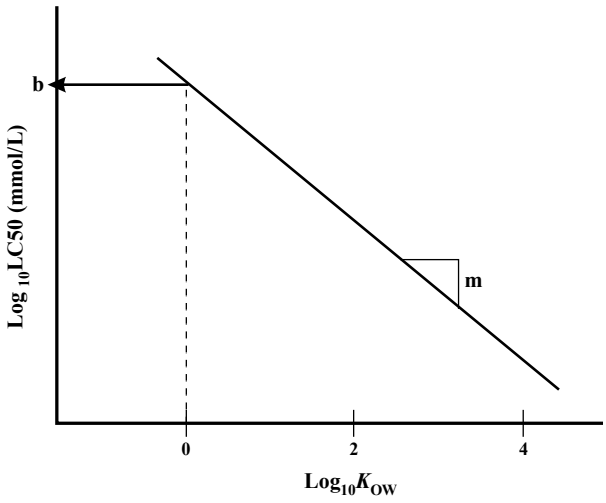


Figure 2-1. Schematic diagram of the $\log_{10}$LC50 versus $\log_{10}K_{OW}$ relationship. At $\log_{10}K_{OW} = 0$ ($K_{OW} = 1$), the concentration in water equals the concentration in octanol.

Thus, the target lipid narcosis model differentiates between the chemical and biological parameters of the $\log_{10}$LC50 - $\log_{10}K_{OW}$ regression coefficients in the following way

$$\underbrace{m}_{\text{slope}} = \underbrace{-a_1}_{\text{chemical}}$$

$$\underbrace{b}_{\text{intercept}} = \underbrace{-a_0}_{\text{chemical}} + \underbrace{\log_{10}C^*_L}_{\text{biological}} \qquad (2\text{-}17)$$

The chemical parameters a_0 and a_1 are associated with the LFER between octanol and target lipid (Equation 2-12). The biological parameter is the critical target lipid concentration C^*_L. This result is important because it suggests that the slope $m = -a_1$ of the $\log_{10}$LC50- $\log_{10}K_{OW}$ relationship should be the same regardless of the species tested since it is a chemical property of the target lipid - the slope of the LFER. Of course this assumes that the target lipid of all species have the same LFER relative to octanol. This seems to be a reasonable expectation since the mechanism of narcosis is presumed to involve the phospholipids in the cell membrane and it appears to be a ubiquitous mode of action. However, the biological component of the intercept C^*_L (Equations 2-13 and 2-17) should vary with species sensitivity to narcosis since it is commonly found that different species have varying sensitivity to the effects of exposure to the same chemical. The expectations that follow from the target lipid model - that the slope should be constant among species and that the intercepts should vary among species - is the basis for the data analysis presented below.

2.5 Acute Lethality Database Compilation

An acute lethality (LC50) database for type I narcotics from water-only toxicity tests was compiled from available literature sources. The principal criterion for acceptance was that a number of chemicals were tested using the same species so that the slope and intercept of the $\log_{10}$LC50 - $\log_{10}K_{OW}$ relationship could be estimated. The data were restricted to acute exposures and a mortality end point to limit the sources of variability. A total of 33 aquatic species including amphibians, fishes, arthropods (insects

and crustaceans), molluscs, annelids, coelenterates and protozoans were represented. Seventy-four individual datasets were selected for inclusion in the database which provided a total of 796 individual data points. Details are provided in Appendix A. The individual chemicals which comprise the database are listed in Appendix B. There are 156 different chemicals including halogenated and non-halogenated aliphatic and aromatic hydrocarbons, PAHs, alcohols, ethers, furans, and ketones.

The $\log_{10}K_{OW}$ values and aqueous solubilities of these chemicals were determined using SPARC (SPARC Performs Automated Reasoning in Chemistry) (Karickhoff et al., 1991), which utilizes the chemical's structure to estimate various properties. The reliability of SPARC was tested using $\log_{10}K_{OW}$ values measured using the slow stir flask technique (de Bruijn et al., 1989). Fifty three compounds such as phenols, anilines, chlorinated monobenzenes, PAHs, PCBs and pesticides were employed. A comparison of the $\log_{10}K_{OW}$ values measured using the slow stir flask technique to the SPARC estimates demonstrates that SPARC can be used to reliably estimate measured $\log_{10}K_{OW}$ values over nearly a seven order of magnitude range of $\log_{10}K_{OW}$ (Figure 2-2A). Note that this comparison tests both SPARC and the slow stir measurements, since SPARC is not parameterized using octanol-water partition coefficients (Hilal et al., 1994).

2.5.1 Aqueous Solubility

The toxicity data were screened by comparing the LC50 value to the aqueous solubility, S, of the chemical (Figure 2-2B). (Note: For this and other figures in this document where a large number of data points are available, the plotting procedure limits the actual number of data plotted.) Individual LC50 values were eliminated from the database if the LC50 > S, which indicated the presence of a separate chemical phase in the experiment. For these cases, mortality must have occurred for reasons other than narcosis - for example, the effect of the pure liquid on respiratory surfaces - since the target lipid concentration cannot increase above that achieved at the water solubility concentration. A total of 55 data points were eliminated, decreasing

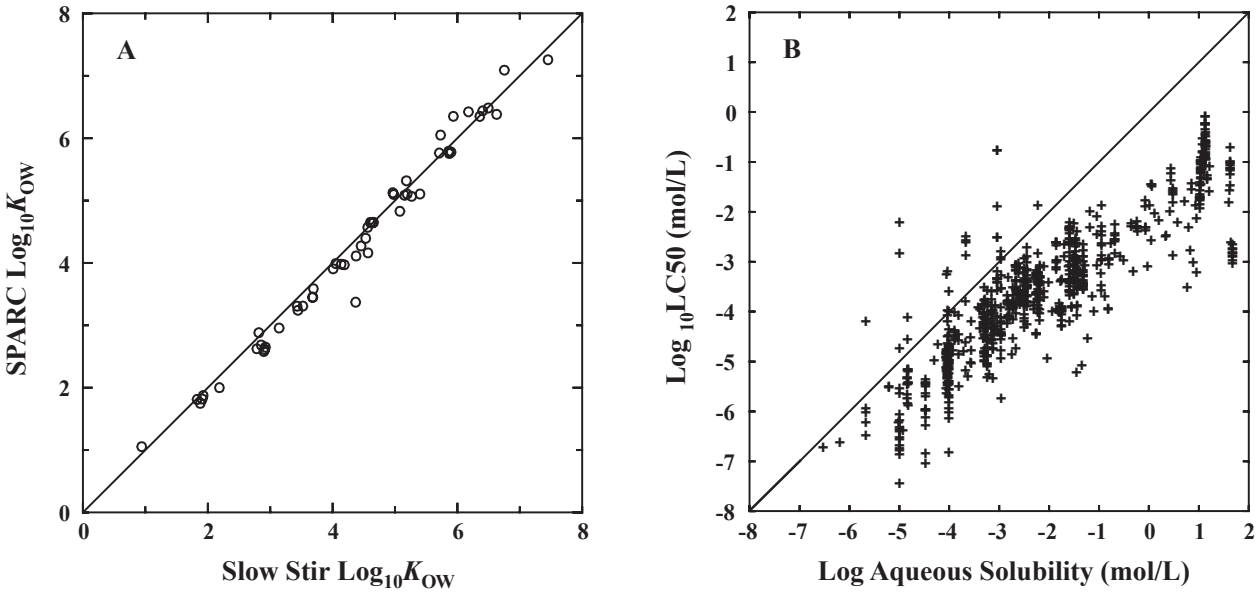


Figure 2-2. Comparisons of (A) $\log_{10}K_{OW}$ predicted by SPARC versus measured $\log_{10}K_{OW}$ using slow stir method and (B) reported $\log_{10}$LC50 values versus the aqueous solubility estimated by SPARC. The diagonal line represents equality.

the number to 736 and the number of individual chemicals to 145 (Appendix B).

2.5.2 Exposure Duration

The duration of exposure varied in the dataset from 24 to 96 hours (Appendix A). Before the data could be combined for ana ysis, the individual datasets should be adjusted to account for this difference. The required equilibration time may vary with both organism and chemical. An increase in either organism body size or chemical hyodrphobicity may increase the time to reach equilibrium.

To determine if acute lethality for narcotic chemicals varied with exposure time, cata were selected where toxicity was reported at multiple exposure times for the same organism and the same chemical. For seven fish species, data were available for 96 hours and either 24, 48 or both 24 and 48 hours of exposure. Arithmetic ratios of the LC50 values for 48 to 96 hours and for the 24 and 96 hours exposure are compared to log 10 K_{OW}. The 48 to 96 hour ratio is 1.0 for essentially all the data (Figure 2-3A). The 24 to 96 hour ratio is larger, approaching 1.4 for the higher Kow chemicals (Figure 2-3B). A linear regression is used to fit the relationship in Figure 2-3B.

$$LC50_{(24)}/LC50_{96} = 0.0988 \log_{10} K_{OW} + 0.9807 \qquad (2\text{-}18)$$

where $LC50_{24}$ and $LC50_{96}$ are the LC50 values for 24 and 96 hour exposures. Since approximately 46% of the data points in the overall database represent narcosis mortality after exposure of fish to a chemical for 24 hours, these data were converted to 96 hour LC50 values using Equation 2-18 for chemicals having $\log_{10}K_{OW}$ values of >1. No correction factor is applied to 24 hour toxicity data for invertebrates and fishes exposed to chemicals having $\log_{10}K_{OW}$ values of <1 (Di Toro et al., 2000).

2.6 Data Analysis

The analysis of the toxicity data is based on the target lipid model assumption that the slope of the $\log_{10}K_{OW}$ is the same for all species. This assumption was tested using a linear regression model to estimate the species-specific body burdens and the universal narcosis slope.

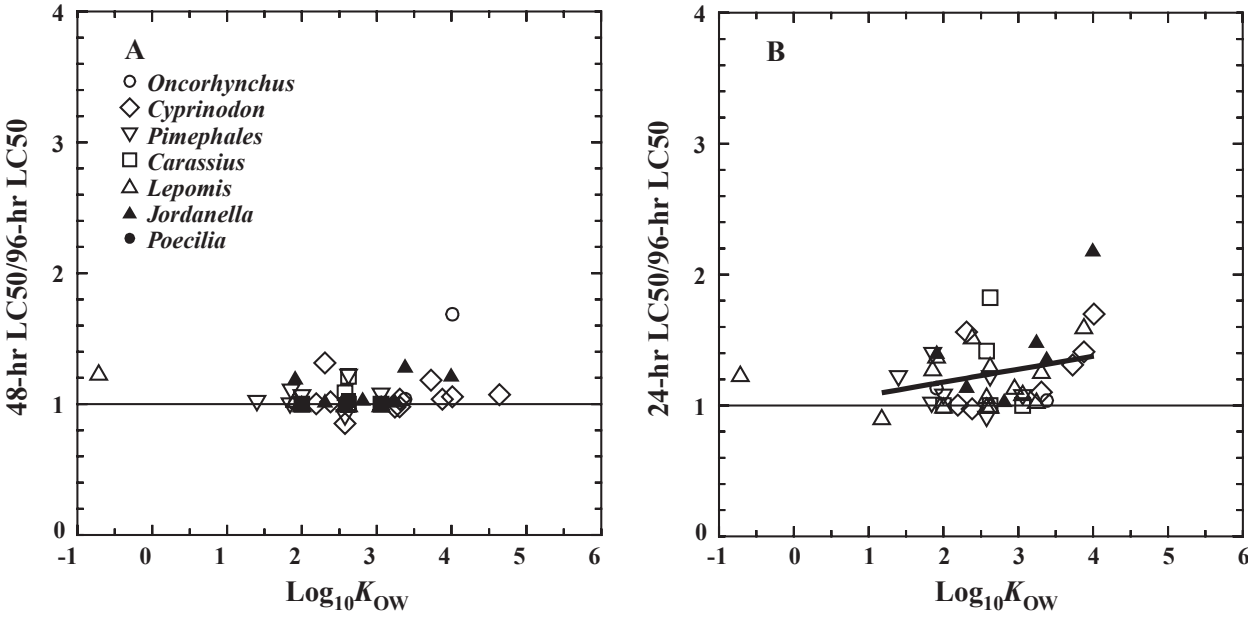


Figure 2-3. Ratios of (A) 48- to 96-hour LC50 values and (B) 24- to 96-hour LC50 values versus $\log_{10}K_{OW}$. The line in (B) is the regression used to correct the 24-hour LC50 to 96-hour LC50.

2.6.1 Regression Model

Consider a species k and a chemical j. The $LC50_{k,j}$ for that species-chemical pair is

$$\log_{10}LC50_{k,j} = \log_{10}C_L^*(k) - a_0 - a_1 \log_{10}K_{OW}(j) \quad (2\text{-}19)$$

$$= b_k - a_1 \log_{10}K_{OW}(j) \quad (2\text{-}20)$$

where

$$b_k = \log_{10}C_L^*(k) - a_0 \quad (2\text{-}21)$$

is the y-intercept. The problem to be solved is: how to include all the b_k, k = 1,...,N_S corresponding to the N_s = 33 species and a single slope a_1 in one multiple linear regression model equation.

The solution is to use a set of indicator variables $*_{ki}$ that are either zero or one depending on the species associated with the observation being considered. The definition is

$$\delta_{ki} = 1 \qquad k = i$$

$$\delta_{ki} = 0 \qquad k \neq i \quad (2\text{-}22)$$

which is the Kronecker delta (Kreyszig, 1972). The regression equation can be formulated using δ_{ki} as follows

$$\log_{10}LC50_{i,j} = a_1 \log_{10}K_{OW}(j) + \sum_{k=1}^{N_S} b_k \delta_{ki} \quad (2\text{-}23)$$

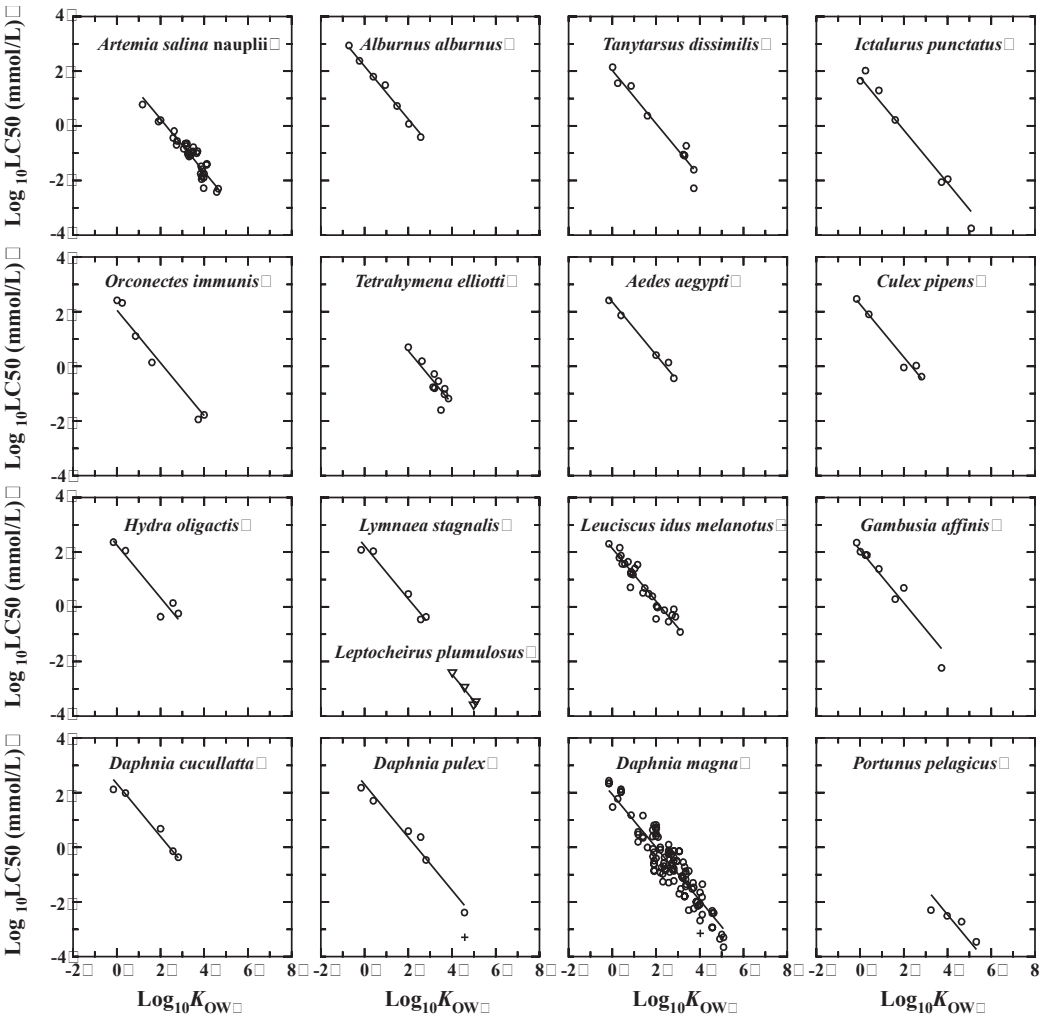


Figure 2-4. $Log_{10}LC50$ versus $log_{10}K_{OW}$ for the indicated species. The line has a constant slope of −0.945. The y-intercepts vary for each species. Outliers are denoted by a plus symbol (+).

Equation 2-23 is now a linear equation with N_s+1 independent variables: $\log_{10}K_{OW}(j)$ and δ_{ki}, k = 1,...,N_s. There are N_s+1 coefficients to be fit: a_1 and b_k, k = 1,...,N_S. For each $LC50_{ij}$ corresponding to species i and chemical j, one of the b_k corresponding to the appropriate species k = i has a unity coefficient $\delta_{ii} = 1$ while the others are zero. The way to visualize this situation is to realize that each row of data consists of the LC50 and these N_S+1 independent variables, for example for j = 1 and i = 3

$\log_{10}(LC50_{ij})$	$\log_{10}(K_{OW}(j))$	δ_{1i}	δ_{2i}	δ_{3i}	...	δ_{Nsi}
0.788	1.175	0	0	1	0	0

(2-24)

which is actually the first of the 736 records in the database. The result is that b_3 is entered into the regression equation as the intercept term associated with species i = 3 because that δ_{ki} is one for that record. By contrast, the slope term $a_1\log_{10}K_{OW}(j)$ is always included in the regression because there is always an entry in the $\log_{10}K_{OW}(j)$ column (Equation 2-24). Hence, the multiple linear regression estimates the common slope a_1 and the species-specific intercepts b_k, k = 1,...,N_s.

A graphical comparison of the results of fitting Equation 2-23 to the full dataset are shown in Figure 2-4 for each of the 33 species. The regression coefficients are tabulated and discussed subsequently after a further refinement is made to the model. The lines appear to be representative of the data as a whole. There appear to be no significant deviations from the common slope. A few outliers, which are plotted as +, were not included in the regression analysis. An outlier is identified if the difference between predicted and observed LC50 values are greater than one log unit when they are included in the regression. This decreases the total number of data points from 736 to 722.

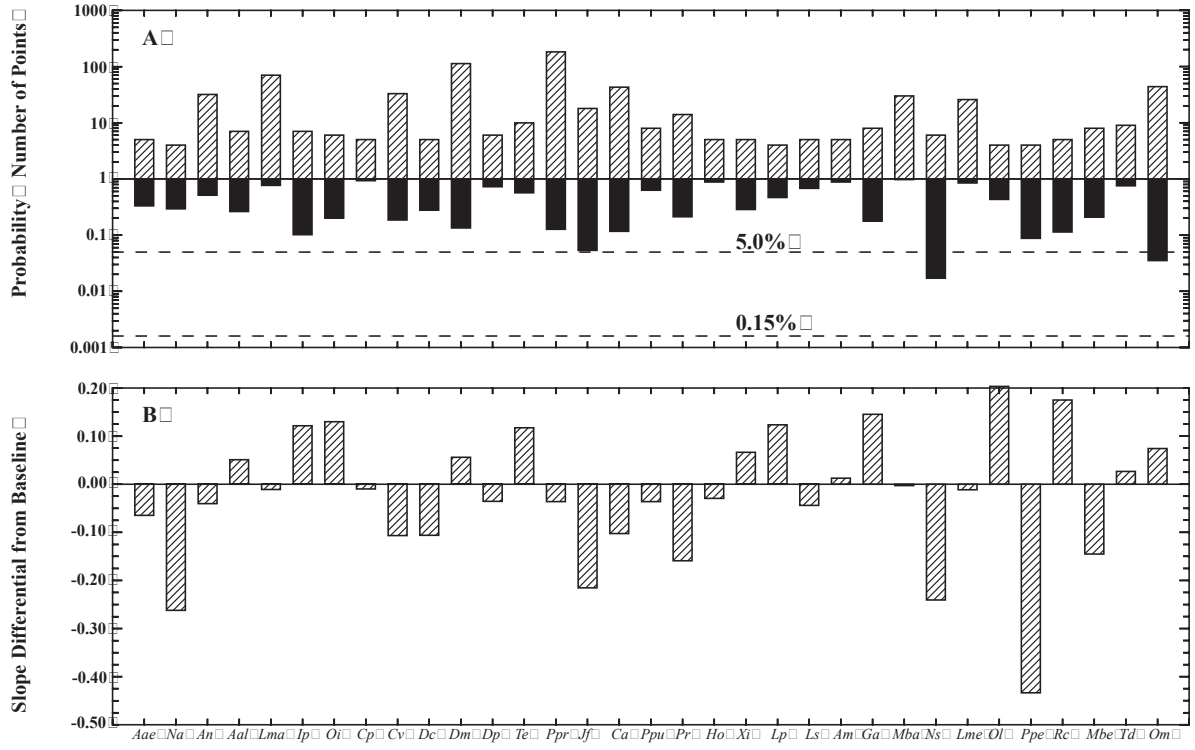


Figure 2-5 Statistical comparison of slopes fitted to individual species to the universal slope of −0.945 showing (A) the probability that the difference occurred by chance (filled bars) and number of data points in the comparison (hatched bars) for each species in the database, and (B) the deviations of the individual estimates from the universal slope.

2.6.2 Testing Model Assumptions

The adequacy of the regression model is tested by answering three questions:

1. Are the data consistent with the assumption that the slope is the same for each species tested?
2. Does the volume fraction hypothesis (Abernethy et al., 1988) provide a better fit?
3. Are there systematic variations for particular chemical classes?

The first assumption, that the slope estimated for a particular species is statistically indistinguishable from the universal slope a_1 = -0.97 without chemical class correction (see Section 2.5.4), can be tested using conventional statistical tests for linear regression analysis (Wilkinson, 1990). The method is to fit the data for each species individually to determine a species-specific slope. Then, that slope is tested against the universal slope a_1 = -0.97 without chemical class correction to determine the probability that this difference could have occurred by chance alone. The probability and the number of data points for each species are shown in Figure 2-5A. The slope deviations are shown in Figure 2-5B. Some of the slope deviations are quite large. However, only three species equal or exceed the conventional significance level of 5% for rejecting the equal slope hypothesis.

Testing at the 5% level of significance is misleading, however, because there is a one in twenty chance of rejecting one species falsely when 33 species are being tested simultaneously. The reason is that the expected number of rejections for a 5% level of significance would be 33 x 0.05 = 1.65, i.e., more than one species on average would be rejected due to statistical fluctuations even though all the slopes are actually equal. In fact, only 20 tests at 5% would, on average, yield one slope that would be incorrectly judged as different. The correct level of significance is (1/33)(1/20) = 0.152% so that the expected number of rejections is 33 x 0.00152 = 0.05 or 5% (Wilkinson, 1990). This level of significance is displayed together with the slope data presented in Figure 2-5A. As can be seen, there is no statistical evidence for rejecting the claim of equal slopes for the tested species. As would be expected, when 5% was used as the level of significance two species were identified as having unique slopes. When the current level of significance (0.00152) was used for the 33 samples none were significantly different.

2.6.3 Volume Fraction Hypothesis

The volume fraction hypothesis asserts that narcotic mortality occurs at a constant volume fraction of chemical at the target site of the organism (Abernethy et al., 1988). Basically, this involves expressing the LC50 as a volume fraction of chemical rather than a molar concentration. This is done using the molar volume of the chemicals (see column MV in Appendix B). The LC50 on a molar volume basis is

$$LC50(cm^3/L) = LC50\,(mmol/L) \times MV\,(cm^3/mmol) \quad (2\text{-}25)$$

The question is: does using molar volume as the concentration unit improve the regression analysis? The results are shown below

	Molar concentrations (mmol/L)	Molar volumes (cm^3/L)
Slope	-0.97 ±0.012	-0.90 ± 0.012
R^2	0.94	0.96

The coefficient of determination (R^2 value) for the volume fraction analysis (0.96) is slightly greater than that for the molar concentration (0.94). Because they are essentially the same, this document uses the molar concentration rather than those based on the volume fraction. Importantly, the slope for both volume and weight units of concentration is not unity.

2.6.4 Chemical Classes

The analysis presented above assumes that all of the 145 chemicals listed in Appendix B are narcotic chemicals. That is, the only distinguishing chemical property that affects their toxicity is K_{OW}.

A criteria has been suggested that can be used to determine whether a chemical is a narcotic (Bradbury et al., 1989), namely that it demonstrates additive toxicity with a reference narcotic. However, it is not practical to test each possible chemical. The more practical test is whether the toxicity can be predicted solely from the $\log_{10}LC50$ - $\log_{10}K_{OW}$ regression. In fact, this is used in methods that attempt to discriminate reference narcotics from other classes of organic chemicals (Verhaar et al., 1992).

Using this approach, differences in toxicity among chemical classes would be difficult to detect if differing species were aggregated or different slopes were allowed in the regression analysis. However, with the large dataset employed above, these differences can be seen by analyzing the residuals grouped by chemical class.

The criteria for choosing the relevant classes are not obvious without a detailed understanding of the mechanism of narcotic toxicity. Hence, the conventional organic chemical classes based on structural similarities, e.g. ethers, alcohols, ketones, etc., are used. The results are shown in Figure 2-6A. The means ±2 standard error (SE) of the means are shown for each class. Although not a rigorous test, the ±2 SE range does not encompass zero for certain classes. Thus, it is likely that there are statistically significant chemical class effects.

2.6.4.1 Statistical Analysis of K_{OW}-Toxicity Relationships

A rigorous test is conducted by including correction constants for each of the chemical classes in a manner that is analogous to Equation 2-23. The model equation is formulated using N_C - 1 corrections, Δc_ℓ, corresponding to the $\ell = 1,...,N_C$ - 1 chemical classes. These are interpreted as corrections relative to the reference class which is chosen to be aliphatic non-halogenated hydrocarbons. The regression equation is formulated as before with a variable $\xi_{\ell j}$ that is one if chemical j is in chemical class ℓ and zero otherwise

$\xi_{\ell j} = 1$ if chemical j is in class ℓ

$\xi_{\ell j} = 0$ otherwise (2-26)

The regression equation that results is

$$\log_{10}LC50_{i,j} = a_1 \log_{10}K_{OW}(j) + \sum_{k=1}^{N_S} b_k \delta_{k_i} + \sum_{\ell=1}^{N_C-1} \Delta c_\ell \xi_{\ell j} \quad (2\text{-}27)$$

Each data record now contains t he dependent variable $\log_{10}LC50_{i,j}$, the independent variables $\log_{10}K_{OW}(j)$, and the δ_{ki}, $k = 1,...,N_s$ and $\xi_{\ell j}$, $\ell = 1,...,N_C$ - 1 indicator variables which are 0 or 1 depending on which species and which chemical class is represented by the $LC50_{i,j}$.

Only N_C - 1 chemical class corrections are required because including N_C class corrections under-determines the equation set with one too many unknowns. The reason is that every equation would have one b_i and one Δc_ℓ for species i and chemical j in chemical class ℓ. Since this condition would occur in every equation there is no unique solution for the b_k and the Δc_ℓ values. One of these constants could be adjusted by an arbitrary amount and the rest could then be adjusted to compensate while still achieving the same fit of the data. Thus, a reference chemical class is chosen: non-halogenated aliphatic hydrocarbons for which $\Delta c_\ell = 0$. The remaining regression constants Δc_ℓ, $\ell = 1,...,N_c$ - 1 are then the differential toxicity of chemical class ℓ relative to the reference chemical class. This is the reason for the Δc notation.

The requirement for a chemical class correction is decided using a statistical test that compares the Δc_ℓ values that result from the regression to the hypothesis $\Delta c_\ell = 0$. For the classes which are not statistically different, they are included in the reference class and the parameters are re-estimated. This is continued until all the remaining Δc_ℓ values are statistically different from zero. After a number of trials, it was found that treating halogen substitutions as a separate additive correction gave the least number of statistically significant class corrections. Thus, chemical class corrections are applied to the base structure, if necessary, and an additional correction is made if any substitute is a halogen. Therefore,

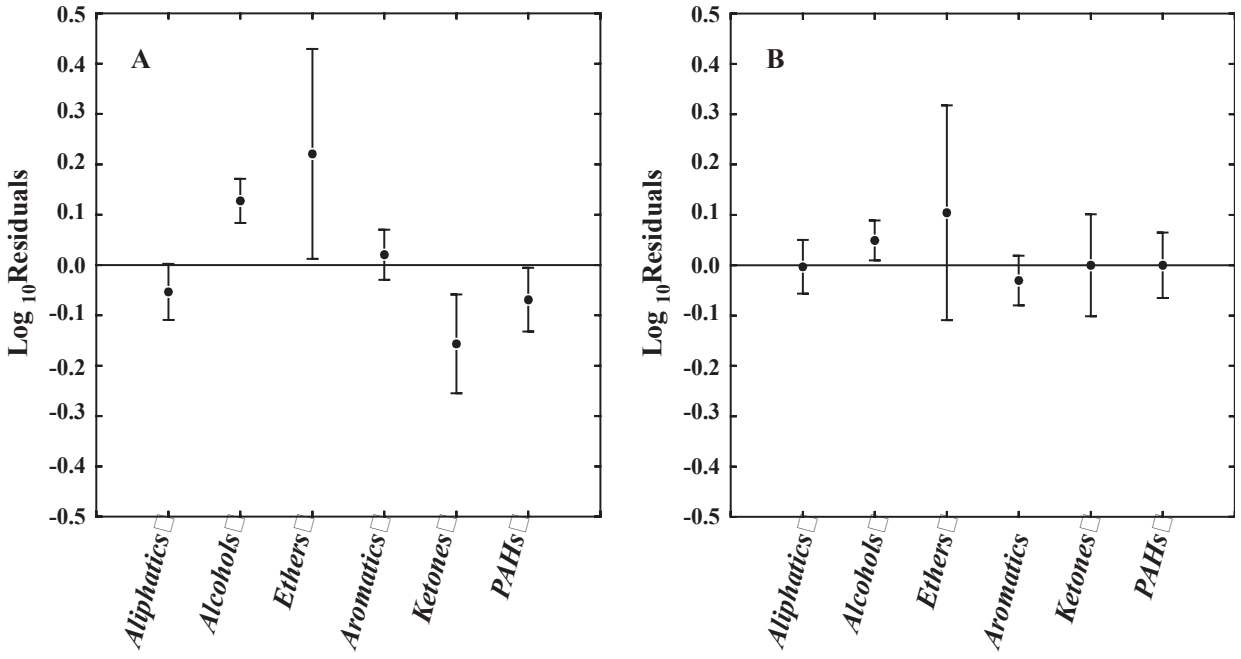


Figure 2-6. Chemical class comparisons of residuals from the regression grouped by class with (A) mean ± 2 standard errors and (B) chemical class corrections included in the regression.

for halogenated chemicals it is possible that two $\xi_{ij} = 1$ in Equation 2-27. The chemical classes are listed in Appendix B.

The results of the final regression analysis are listed in Table 2-1. Both the logarithmic b_i and arithmetic 10^{b_i} values of the intercepts are included together with their standard errors. Chemical classes which demonstrate higher potency than the reference class are ketones and PAHs. Halogenation increases the potency as well. After accounting for different potencies in the chemical classes, the mean residuals are statistically indistinguishable from zero (Figure 2-6B).

2.6.4.2 Standard Errors and Residuals

The standard errors of the body burdens $SE(b_i)$ found from the regression (Equation 2-27) are in an almost one-to-one correspondence with the number of data points for that species. Thus, the b_i for *Pimephales promelas* (fathead minnow) with 182 data points has a 10% coefficient of variation, $CV(b_i) = SE(b_i)/b_i$, while the b_i for *Neanthes arenaceodentata* (polychaete worm) with 4 data points has a 50% coefficient of variation (Table 2-1). The relationship of the sample size (N) to the coefficient of variation of the estimated critical body burden, $CV(b_i)$, is shown in Figure 2-7A.

The residuals are log normally distributed (Figure 2-7B) and exhibit no trend with respect to K_{OW} (Figure 2-7C) which confirms the assumption underlying the use of regression analysis. The reason they are restricted to ±1 order of magnitude is that 14 data points outside that range were originally excluded as outliers (for some values previously less than ± one order of magnitude, chemical class corrections produced values slightly greater than one order of magnitude as shown in Figure 2-7C).

2.6.4.3 Chemical Class Corrections

The corrections due to chemical classes reduce the critical body burden by a factor of approximately one-half for ketones and PAHs. Correction for halogenation reduces it further by

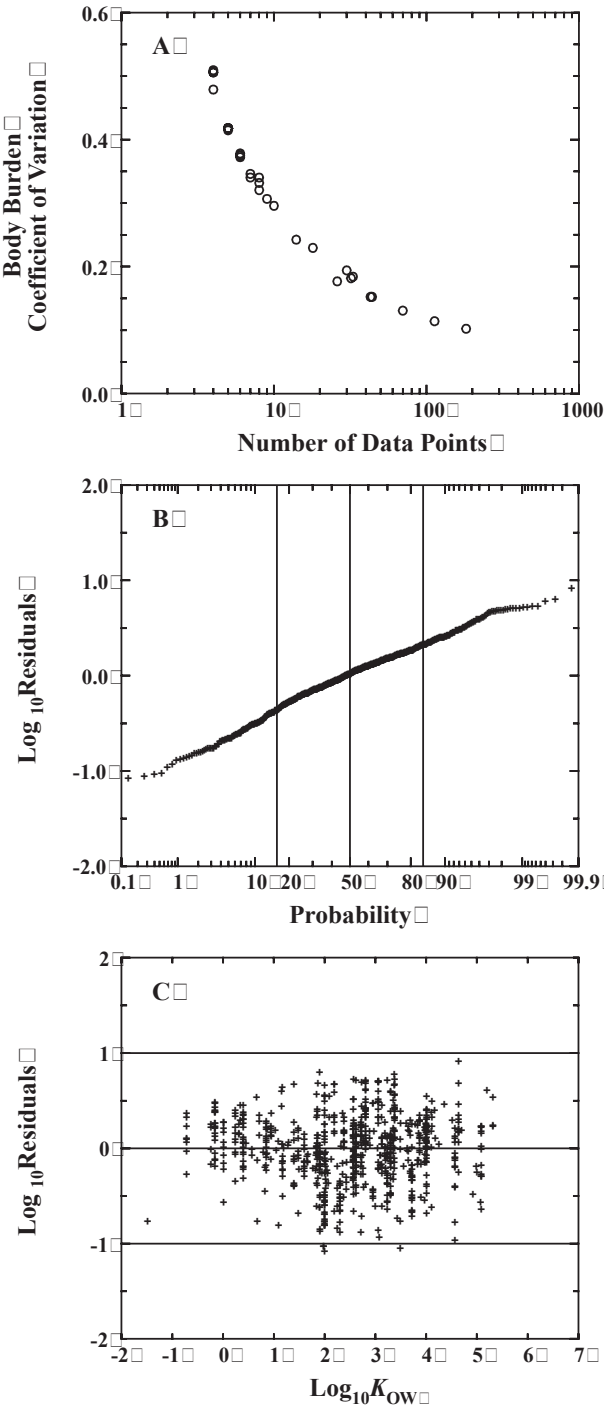


Figure 2-7. The coefficient of variation of the estimated species-specific body burdens versus (A) the number of data points for that species (B), the log probability plot of the residuals, and (C) the residuals versus $\log_{10}K_{OW}$.

0.570 (Table 2-1). Thus, a chlorinated PAH would exhibit a critical body burden of approximately one-third of a reference narcotic. The coefficients of variation for these corrections are approximately 10%.

The chemical class differences among the type I narcotics affect the LC50-K_{OW} relationship. The model no longer predicts a single straight line for the $\log_{10}$LC50-$\log_{10}K_{OW}$ relationship for all narcotic chemicals. What is happening is that the y-intercepts are changing due to the changing $)c_R$ values. The model (Equation 2-27) when applied to a single species k is

$$\log_{10}LC50_{k,j} = a_1 \log_{10}K_{OW}(j) + b_k + \sum_{\ell=1}^{N_C-1} \Delta c_\ell \xi_{\ell j} \qquad (2\text{-}28)$$

This is a straight line if only reference narcotics are considered $\Delta c_\ell = 0$ or if only one chemical class correction is involved, e.g., all halogenated reference narcotics. Otherwise, more than one Δc_ℓ, enters into Equation 2-28 and the line is jagged. Figure 2-8 presents three examples. The deviations from the reference narcosis straight line are caused by the different chemical class potencies.

2.7 Universal Narcosis Slope

The universal narcosis slope: m = -0.945±0.014 which results from the final analysis that includes chemical class corrections (Table 2-1) is smaller than that determined above without chemical class corrections (-0.97±0.012). It is close to unity, a value commonly found (Hansch and Leo, 1995), and larger than the average of individual slopes (-0.86±0.14) reported by Van Leeuwen et al. (1992), but comparable with a recent estimate for fathead minnows of -0.94 (Di Toro et al., 2000).

The fact that the slope is not exactly one suggests that octanol is not quite lipid. However, it is also possible that for the more hydrophobic chemicals in the database, the exposure time may not have been long enough for complete equilibration of water and lipid to have occurred. To test this hypothesis, the regression analysis is

restricted to successively smaller upper limits of $\log_{10}K_{OW}$. The results are listed below

Maximum $\log_{10}K_{OW}$	3.5	4.0	4.5	5.0	5.5
Slope	-0.959	-0.970	-0.958	-0.950	-0.945
Standard Error	0.018	0.015	0.015	0.014	0.015

The variation is within the standard errors of estimation, indicating that there is no statistically significant difference if the higher $\log_{10}K_{OW}$ data are removed from the regression. This suggests that the universal narcosis slope is not minus one but is actually -0.945 ± 0.014.

One consequence of the use of a universal narcosis slope is that the species sensitivity ranking derived from comparing either the water-only LC50 values or the critical body burdens of various species are the same. This occurs because the critical body burden is calculated from the LC50 value and the universal slope (Equations 2-14 and 2-15)

$$\log_{10}C_L^* = \log_{10}LC50 + 0.945\log_{10}K_{OW} \qquad (2\text{-}29)$$

If this were not the case, then the species sensitivity order could be reversed if LC50 values or C_L^* were considered.

Equation 2-29 is important because it can be used to compute the critical body burden of any type I narcotic chemical. Thus it predicts what the critical body burden should be for a particular species at its LC50 value. This would be the concentration that would be compared to a directly measured critical body burden. It can be thought of as a normalization procedure that corrects type I narcotics for the varying K_{OW} and places them on a common footing, namely, the critical body burden.

The motivation for the development of the target lipid model was to apply it to mixtures of PAHs and other persistent narcotic chemicals in sediments. The narcosis database used to determine the universal narcosis slope and the critical body burdens consists of 145 chemicals, of which 10 are un-substituted and substituted PAHs (Di Toro et al., 2000). A comparison of the LC50 data for just these chemicals and the target lipid

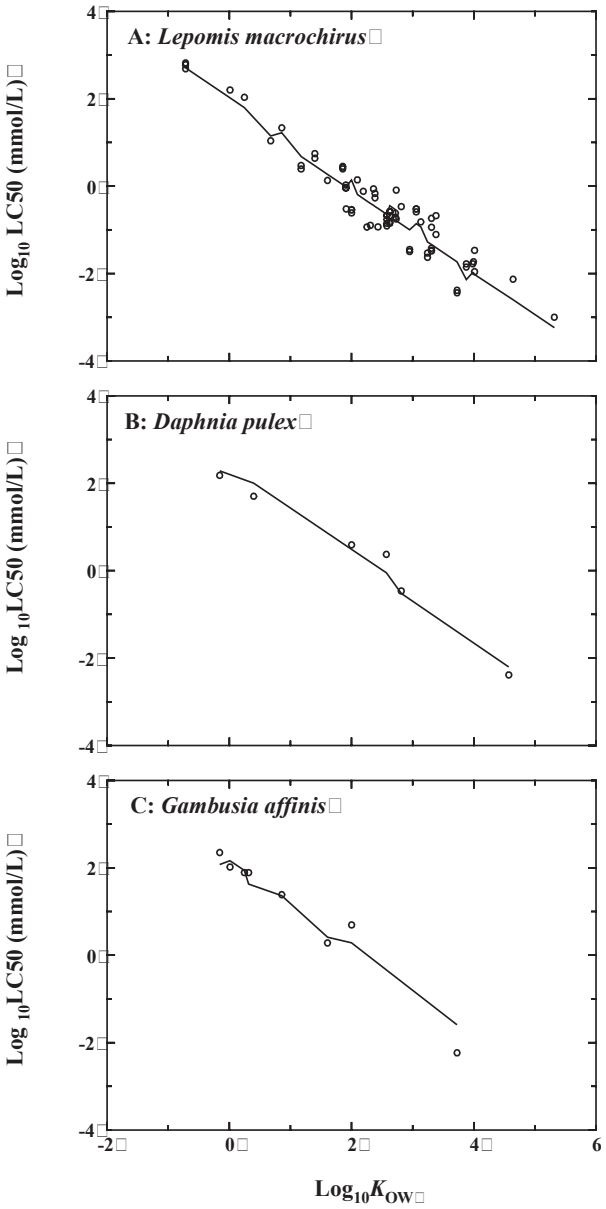


Figure 2-8. $\log_{10}$LC50 versus $\log_{10}K_{OW}$ for (A) *Lepomis macrochirus*, (B) *Daphnia pulex*, and (C) *Gambusia affinis*. The line connects the individual estimates of the $\log_{10}$LC50 values, including the chemical class correction.

model is shown in Figure 2-9. The solid $\log_{10}LC50 - \log_{10}K_{OW}$ lines are computed using the universal narcosis slope and the appropriate body burdens for PAHs for each organism listed. The dotted lines apply to the chloronaphthalenes which have a slightly lower critical body burden due to the halogen substitution. The lines are an adequate fit of the data, although the scatter in the *D. magna* data is larger than some of the other

Table 2-1. Regression results: y-intercepts and chemical class corrections‡ (Table from Di Toro et al., 2000).

Species i	N	b_i	SE(b_i)	10^{b_i} (μmol/g octanol)	SE(10^{b_i}) (μmol/g octanol)
Americamysis bahia	30	1.54	0.082	34.3	6.7
Portunus pelagicus	4	1.56	0.19	36.1	18.2
Leptocheirus plumulosus	4	1.56	0.191	36.2	18.4
Palaemonetes pugio	8	1.68	0.137	48.2	16.4
Oncorhynchus mykiss	44	1.79	0.065	61.7	9.4
Jordanella floridae	18	1.82	0.096	66.1	15.2
Ictalurus punctatus	7	1.87	0.139	74.8	25.9
Pimephales promelas	182	2.02	0.044	105	10.8
Lepomis macrochirus	70	2.03	0.0056	108	14.1
Daphnia magna	113	2.04	0.049	111	12.6
Cyprinodon variegatus	33	2.05	0.078	111	20.5
Oryzias latipes	4	2.05	0.182	112	53.9
Carassius auratus	43	2.13	0.065	134	20.5
Rana catesbian	5	2.13	0.162	135	55.9
Tanytarsus dissimilis	9	2.14	0.125	137	42
Orconectes immunis	6	2.14	0.149	139	52.3
Alburnus alburnus	7	2.16	0.137	144	49.1
Nitocra spinipes	6	2.17	0.148	147	54.7
Gambusia affinis	8	2.17	0.13	149	47.9
Leucisus idus melanotus	26	2.18	0.075	152	26.8
Neanthes arenaceodentata	4	2.23	0.19	168	85
Artemia salina nauplii	32	2.26	0.077	181	32.8
Lymnaea stagnalis	5	2.29	0.163	195	81.5
Xenopus laevis	5	2.33	0.163	213	88.9
Hydra oligactis	5	2.33	0.163	214	89.5
Culex pipiens	5	2.34	0.163	216	90.4
Poecilia reticulata	14	2.36	0.101	228	55.2
Menidia beryllina	8	2.37	0.134	233	77.3
Daphnia pulex	6	2.38	0.15	240	91

Table 2-1. Continued

Species i	N	b_i	SE(b_i)	10^{b_i} (μmol/g octanol)	SE(10^{b_i})
Ambystoma mexicanum	5	2.39	0.163	245	103
Daphnia cucullata	5	2.4	0.163	249	104
Aedes aegypti	5	2.42	0.163	261	109
Tetrahymena elliotti	10	2.46	0.121	286	85
Chemical Class ℓ	N	Δc_ℓ	SE(Δc_ℓ)	$10^{\Delta c_\ell}$	SE($10^{\Delta c_\ell}$)
Aliphatics	215	0	-	1	-
Ethers	13	0	-	1	-
Alcohols	134	0	-	1	-
Aromatics	241	0	-	1	-
Halogenated	319	-0.244	0.033	0.57	0.044
Ketones	49	-0.245	0.059	0.569	0.078
PAHs	84	-0.263	0.057	0.546	0.073
Slope		-0.945	0.014		

‡See Equation (2-27).
N = Number of data points.
b_i = y-intercept.
SE(b_i)=Standard error of b_i.
Δc_ℓ =chemical class correction to the y-intercept.
SE(Δc_ℓ)=standard error of Δc_ℓ.
†=Standard errors of 10^{b_i} and $10^{\Delta c_\ell}$ are based on the assumption that the estimation errors for b_i and Δc_ℓ are gaussian. The formulas follow from the standard error of a log normally distributed random variable (Aitchison and Brown, 1957). For $x = b_i$ or Δc_ℓ, $\mu_\ell = 2.303x$, $\sigma_\ell = 2.303$ SE(x), and SE(10^x)= SE($e^{2.303x}$) = $e^{\mu_\ell}\sqrt{e^{2\sigma_\ell^2} - e^{2\sigma_\ell^2}}$.

species with multiple sources of data and there is a clear outlier for *Americamysis bahia*. It is for this reason that the slope representing all data for narcosis chemicals is used to derive the target lipid concentration from water-only toxicity data for PAHs in Section 3 of this document.

2.8 Comparison to Obsered Body Burdens

The target lipid model predicts the concentration in octanol (the y-intercept) that causes 50% mortality in 96 hours. The question is: how do these compare to measured critical body burdens? The species-specific y-intercepts, b_i, are related to the target lipid concentration by the relationship

$$\text{y-intercept} = b_i = \log_{10} C_L^*(i) - a_0 \qquad (2\text{-}30)$$

or, with chemical class corrections,

$$\text{y-intercept} = b_i + \Delta c_\ell = \log_{10} C_L^*(i) - a_0 \qquad (2\text{-}31)$$

for species i and chemical class ℓ, where a_0 is the parameter in the LFER between octanol and target lipid (Equation 2-12).

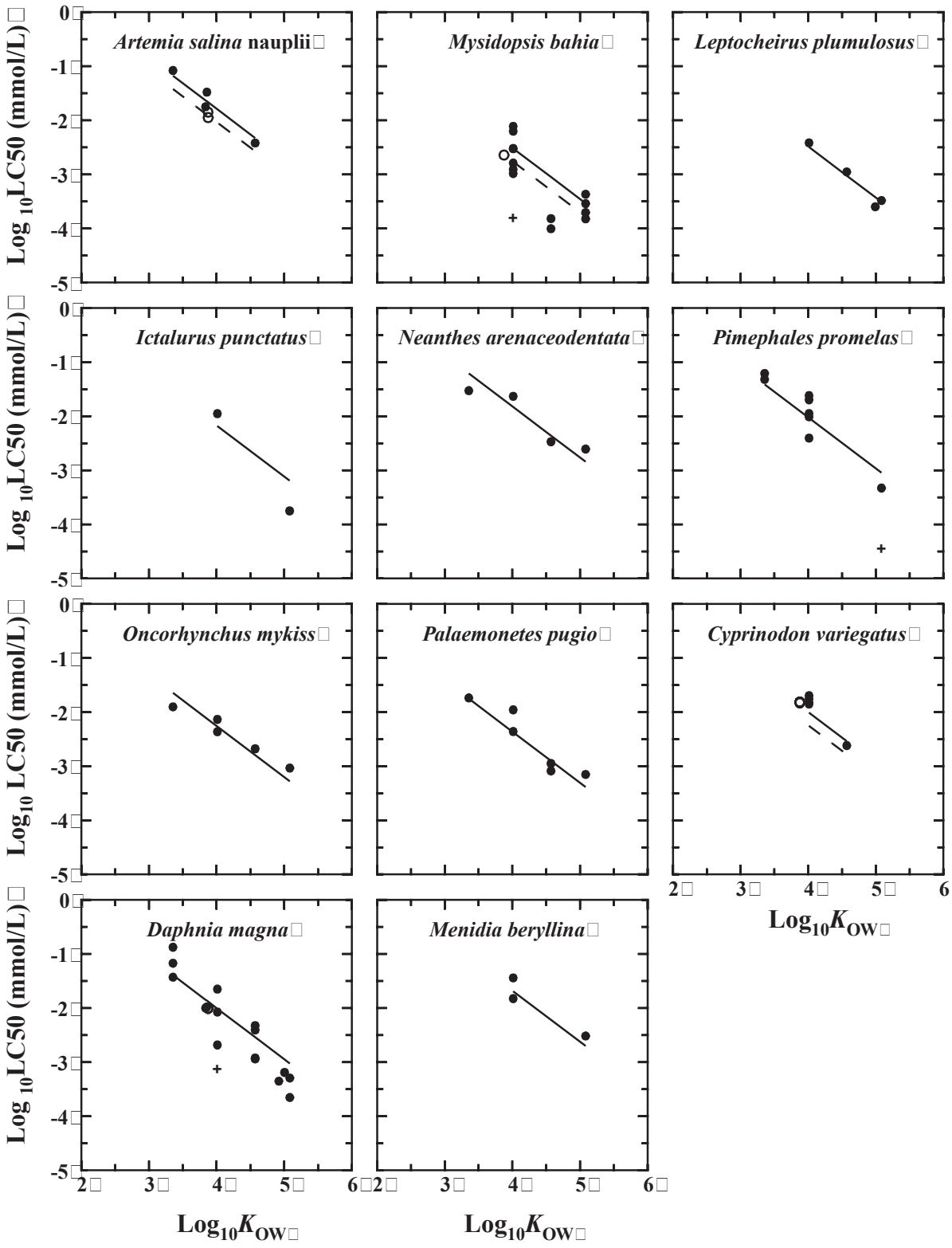


Figure 2-9. Comparison of target lipid model, line-of-fit and observed LC50 data for individual PAHs, by species.

The relationship between the predicted concentration in octanol, $b_i + \Delta c_\ell$, to the concentration measured in extracted lipid, $\log_{10}C_L^*$, is examined in Table 2-2 which lists observed LC50 body burdens (µmol/g lipid) and predicted critical body burdens (µmol/g octanol) for organisms in the database for which measured lipid-normalized critical body burdens were available. Three fish species: *Gambusia affinis* (mosquito fish), *Poecilia reitculata* (guppy) and *P. promelas*, and a crustacean: *Portunus pelagicus* (crab) are compared in Figure 2-10. The predicted and measured body burdens differ by less than a factor of 1.6. The fish were observed to have higher critical body burdens than the crustacean, which the model reproduces.

The apparent near equality between the estimated and measured critical body burdens, which come from two independent sets of data, strongly suggest that in fact

$$a_0 = 0 \qquad (2\text{-}32)$$

so that

$$\log_{10}C_L^*(i) = b_i + \Delta c_\ell = \text{y-intercept} \qquad (2\text{-}33)$$

This relationship implies that the target lipid is the lipid measured by the extraction technique used in the body burden datasets. This is an important practical result since it suggests that body burdens normalized to extracted lipid are expressed relative to the appropriate phase for narcotic toxicity. Since the intercepts appear to be the organism's lipid concentration, the y-intercepts ($b_i + \Delta c_\ell$) in the discussion presented below are referred to as body burden lipid concentrations although the units (µmol/g octanol) are retained since these are, in fact, the actual units of the intercepts.

2.9 Mixtures and Additivity

Narcotic chemicals, including PAHs, occur in the environment as mixtures, therefore, their mixture effects need to be appropriately resolved. If the toxicity of mixtures is additive, mixture effects can be assessed using the concept of toxic units. A toxic unit (TU) is defined as the ratio of the concentration in a medium to the effect concentration in that medium.

The additivity of the toxicity of narcotic chemicals in water has been demonstrated by a number of investigators. The results of mixture experiments which employed a large enough number of narcotic chemicals so that non-additive behavior would be detected is presented in Figure 2-11 as adopted from Hermens (1989). Three of the four experiments demonstrated essentially additive behavior and the fourth, a chronic exposure, was almost additive.

2.10 Aqueous Solubility Constraint

The existence of the need for a solubility cut-off for toxicity was suggested by Veith et al. (1983) based on data from fathead minnows (*P. promelas*) and guppies (*P. reticulata*). The highest dissolved concentration in water that can be achieved by a chemical is its aqueous solubility (S). Therefore, the maximum lipid concentration that can be achieved is limited as well. It is for this reason that the LC50 database is limited to chemicals with $\log_{10}K_{OW} \leq 5.3$. This is also the reason that the LC50 database that was used to generate the FCVs for specific PAHs in Section 3 of this document, was screened initially for LC50 values ≤ S, using the solubilities from Mackay et al. (1992), rather than $\log_{10}K_{OW} \leq 5.3$ used by Di Toro et al. (2000).

For sediments, a solubility constraint should be applied as well. This is readily calculated using the relationship between interstitial water and the organic carbon-normalized sediment concentration. Since the interstitial water concentration is limited by S, the sediment concentration should be limited by the concentration in sediment organic carbon that is in equilibrium with the interstitial water at the aqueous solubility. Therefore, observed sediment concentrations are limited by the condition

$$C_{OC} \leq C_{OC,max} = K_{OC}S \qquad (2\text{-}34)$$

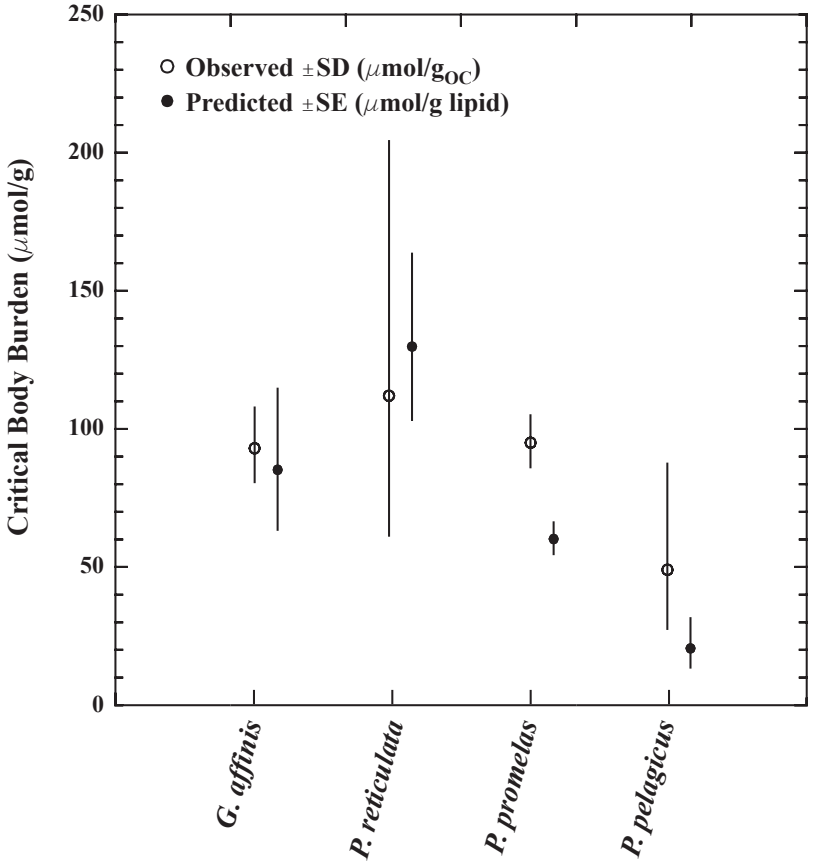


Figure 2-10. Predicted and observed body burdens for four species.

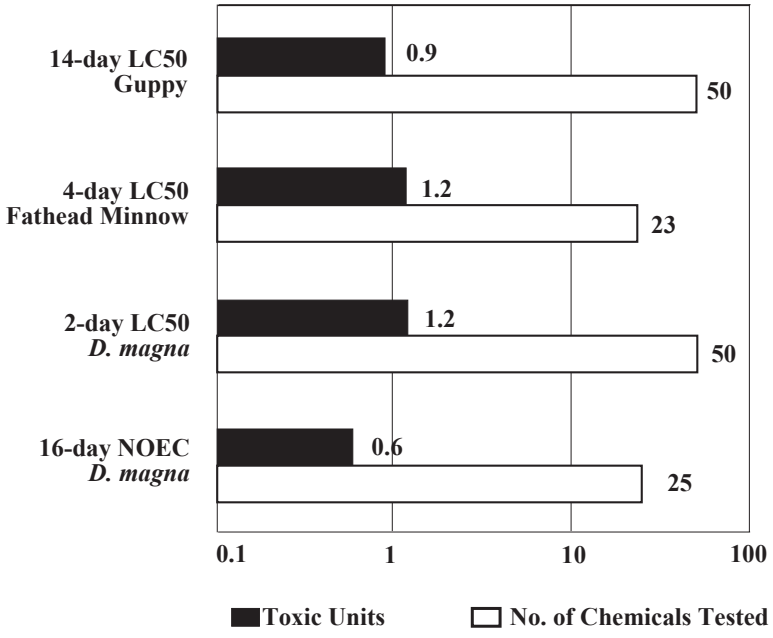


Figure 2-11. Additivity of type I narcosis toxicity. Comparison of the observed TU concentrations calculated from four studies to the predicted TU of 1.0.

Table 2-2. Comparison of body burdens observed in aquatic organisms acutely exposed to narcotic chemicals and body burdens predicted from target lipid narcosis theory (Table from Di Toro et al., 2000).

				C^*O_{rg}			
				Obs	Mean	Pred.	
Organism	Chemical	log K_{OW}	time (hr)	(µmol/ g lipid)		(µmol/g octanol)	References
Mosquitofish,	1,4-dibromonbenzene	3.55	96	85.0			Chaisuksant and
Gambusia affinis	1,2,3-trichlorobenzene	3.98	"	140.0			Connell, 1997
	1,2,4-trichlorobenzene	4.00	"	92.0			
	pentachlorobenzene	5.32	"	69.0	93.2	85.3	
Guppy,	1,4-difluorobenzene	2.11	1.5	444.0			Sijm et al., 1993
Poecilia reticulata	1,2-dichlorobenzene	3.31	91	34.0			
	1,4-dichlorobenzene	3.24	41	400.0			
	1,2-dibromobenzene	3.56	4	24.0			
	1,4-dibromobenzene	3.55	6	120.0	110	130	
Fathead minnow,	1,2-dichlorobenzene	3.31	18	78.0			Sijm et al., 1993
Pimephales promelas	1,4-dichlorobenzene	3.24	10	68.0			
	1,2-dibromobenzene	3.56	7	60.0			
	1,4-dibromobenzene	3.55	10	54.0			
	1,2,4-trichlorobenzene	4.00	50.2				van Wezel et al.,
	1,1,2,2-tetrachlorobenzene	2.31	57.2				1995
	dichlorobenzene	3.27	75.5				
	dichlorobenzene	3.27	129				
	1,2-dichlorobenzene	3.31	62.3				
	1,2-dichlorobenzene	3.31		98.9			van Wezel et al.,
	1,4-dichlorobenzene	3.24		173			1996
	1,4-dichlorobenzene	3.24		121			
	1,2-1,4-dichlorobenzene			107			
	1,2-1,4-dichlorobenzene			110			
	1,2-1,4-dichlorobenzene			138			
	1,2-1,4-dichlorobenzene			150			
	naphthalene	3.36		123			de Maagd et al.,
	1,2,4-trichlorobenzene	4.00		215	95	59.9	1996
Crab,	1,4-dichlorobenzene	3.24	96	9.6			Mortimer and
Portunus pelagicus	1,2,3-trichlorobenzene	3.98	96	45.0			Connell, 1994
	1,2,3,4-tetrachlorobenzene	4.64	96	119			
	pentachlorobenzene	5.32	96	111	49.9	20.6	

Section 3

Toxicity of PAHs in Water Exposures and Derivation of PAH-Specific FCVs

3.1 Narcosis Theory, EqP Theory and WQC Guidelines: Derivation of PAH-Specific FCVs for Individual PAHs

Polycyclic aromatic hydrocarbons occur in the environment as mixtures. Therefore, in order to adequately protect aquatic life the approach used to derive a WQC FCV or sediment benchmark for PAHs must account for their interactions as a mixture. In this section, we present an approach for deriving FCVs for individual PAHs which can be used to derive the ESB for mixtures of PAHs.

Concepts developed by Di Toro et al. (2000) and presented in Section 2 of this document provide the technical framework for screening and analyzing aquatic toxicity data on PAHs (Tables 3-1, 3-2). In particular, Section 2 demonstrated that: (1) the universal slope of the K_{OW}-toxicity relationship for narcotic chemicals is the same for all aquatic species; and (2) the intercept of the slope at a K_{OW} of 1.0 for each species provides the LC50/EC50 in µmol/g octanol that indicates the critical body burden in and relative sensitivities of each species.

These concepts permit the use of the U.S. EPA National WQC Guidelines (Stephan et al., 1985) to derive WQC FCVs for individual PAHs and PAH mixtures. The universal slope is used with PAH-specific LC50/EC50 values to derive test-specific K_{OW} normalized reference acute values at a K_{OW} of 1.0. This normalization was performed to put the data on the toxicities of narcotic chemicals on an internally consistent scale. This was also performed using hardness when WQC were derived for metals. These K_{OW} normalized reference acute values are used to calculate species mean acute values (SMAVs) and genus mean acute values (GMAVs): (1) because only acute and chronic toxicity data from water-only tests with freshwater and saltwater species exposed to individual PAHs are used, a PAH chemical class correction is not needed; (2) the data are screened for acceptability following the requirements for use of species resident to North America, test durations, test quality, etc. of the U.S. EPA National WQC Guidelines (Stephan et al., 1985); (3) the PAH-specific species mean acute values (PAH-specific SMAVs) from Appendix C are adjusted using the universal slope of the K_{OW}-toxicity relationship from the narcotic chemical analysis that was shown to apply to all aquatic species in Section 2 (Equation 2-29) to derive the acute value for that species at a K_{OW} of 1.0 (K_{OW} normalized PAH-specific SMAV) (Appendix C); (4) the intercept of the slope at a K_{OW} of 1.0 provides the LC50/EC50 in µmol/g octanol that indicates the relative sensitivity of each tested species and PAH, which was used to calculate SMAVs and GMAVs in µmol/g octanol, which are indicative of critical tissue concentrations in organisms on a µmol/g lipid basis. The GMAVs are used to calculate the final acute value (FAV) applicable to PAHs at a K_{OW} of 1.0 (Stephan et al., 1985). This FAV at a K_{OW} of 1.0, when divided by the Final Acute-Chronic Ratio (FACR), becomes the FCV at a K_{OW} of 1.0. Importantly, the FCV for any specific PAH can then be derived by back calculating using FAV at a K_{OW} of 1.0, the K_{OW} of the specific PAH and the universal narcosis slope. When the PAH-specific FCV exceeds the known solubility of that PAH, the maximum contribution of that PAH to the toxicity of the mixture is set at the K_{OC} multiplied by the solubility of that PAH.

Table 3-1. Summary of the chronic sensitivity of freshwater and saltwater organisms to PAHs; test specific data.

Common Name, Species	Test[A]	Habitat[B]	PAH	Duration	NOEC[C] (µg/L)	OEC[D] (µg/L)	Observed Effects (Relative to Controls)	Chronic Value (µg/L)	Reference
Cladoceran, *Daphnia magna*	LC	W	Anthracene	21d		2.1	5.3% fewer broods	<2.1	Holst and Giesy, 1989
						4	8.0% fewer broods		
						8.2	13.8% fewer broods		
Cladoceran, *Daphnia magna*	LC	W	Fluoranthene	21d	6.9-17	35	17% reduction in length	24.5	Spehar et al., 1999
						73	25% reduction in length, 37% fewer young/adult		
						148	No survival		
Cladoceran, *Daphnia magna*	LC	W	Phenanthrene	21d	46-57	163	Survival reduced 83%, 98% fewer broods	96.39	Call et al., 1986
Midge, *Paratanytarsus sp.*	LC	B	Acenaphthene	26d	32-295	575	Survival reduced ~90%, ~60% reduction in growth, no reproduction	411.8	Northwestern Aquatic Sciences, 1982
Midge, *Paratanytarsus sp.*	LC	B	Acenaphthene	26d	27-164	315	Survival reduced ~20%, ~30% reduction in growth	227.3	Northwestern Aquatic Sciences, 1982; Thursby, 1991a
						676	Survival reduced ~60%		
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32d	50	109	5% reduction in growth	73.82	Academy of Natural Sciences, 1981; Thursby, 1991a
						410	26% reduction in growth, Survival reduced 45%		
						630	No survival		

Table 3-1. Continued

Common Name, Species	Test[A]	Habitat[B]	PAH	Duration	NOEC[C] (µg/L)	OEC[D] (µg/L)	Observed Effects (Relative to Controls)	Chronic Value (µg/L)	Reference
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32d	50-109	410	20% reduction in growth, Survival reduced 66%	211.4	Academy of Natural Sciences, 1981; Thursby, 1991a
						630	No survival		
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32-35d	67-332	495	54% reduction in growth	405.4	Cairns and Nebeker, 1982
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32-35d	197-345	509	30% reduction in growth	419	Cairns and Nebeker, 1982
						682	52% reduction in growth, Survival reduced 45%		
						1153	87% reduction in growth, Survival reduced 97%		
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32d	64	98	Survival reduced 24%	79.2	ERCO, 1981
						149	Survival reduced 65%		
						271	Survival reduced 75%		
						441	Survival reduced 80%		
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32d	50-91	139	Survival reduced 20%	112.5	ERCO, 1981
						290	Survival reduced 50%		
						426	Survival reduced 52%		
Fathead minnow, *Pimephales promelas*	ELS	W	Fluoranthene	32d	3.7-10.4	21.7	Survival reduced 67%, 50% reduction in growth	15.02	Spehar et al., 1999
Rainbow trout, *Oncorhynchus mykiss*	ELS	B/W	Phenanthrene	90d	5	8	Survival reduced 41%, 33% reduced growth	6.325	Call et al., 1986
						14	Survival reduced 48%, 44% reduced growth		
						32	Survival reduced 52%, 75% reduced growth		
						66	No survival		

Table 3-1. Continued

Common Name, Species	Test[A]	Habitat[B]	PAH	Duration	NOEC[C] (µg/L)	OEC[D] (µg/L)	Observed Effects (Relative to Controls)	Chronic Value (µg/L)	Reference
Mysid, *Americamysis bahia*	LC	B/W	Acenaphthene	35d	100-240	340	93% reduction in young	285.7	Horne et al., 1983
						510	No survival		
Mysid, *Americamysis bahia*	LC	B/W	Acenaphthene	25d	20.5-44.6	91.8	91% reduction in young	63.99	Thursby et al., 1989b
						168	No reproduction, 34% reduction in growth		
						354	Survival reduced 96%, no reproduction		
Mysid, *Americamysis bahia*	LC	B/W	Fluoranthene	28d	35926	21	Survival reduced 26.7%, 91.7% reduction in young	15.87	U.S. EPA, 1978
						43	No survival		
Mysid, *Americamysis bahia*	LC	B/W	Fluoranthene	31d	0.41-11.1	18.8	Survival reduced 23%, no reproduction	14.44	Spehar et al., 1999
Mysid, *Americamysis bahia*	LC	B/W	Phenanthrene	32d	1.5-5.5	11.9	No survival	8.129	Kuhn and Lussier, 1987
Mysid, *Americamysis bahia*	LC	B/W	Pyrene	28d	3.82	5.37	46% reduction in young	4.53	Champlin and Poucher, 1992b
						6.97	47% reduction in young		
						9.82	73% reduction in young		
						15.8	85% reduction in young		
						20.9	90% reduction in young, Survival reduced 37%		
						38.2	No survival		

Table 3-1. Continued

Common Name, Species	Test[A]	Habitat[B]	PAH	Duration	NOEC[C] (µg/L)	OEC[D] (µg/L)	Observed Effects (Relative to Controls)	Chronic Value (µg/L)	Reference
Sheepshead minnow, *Cyprinodon variegatus*	ELS	B/W	Acenaphthene	28d	240-520	970	Survival reduced 70%	710.2	Ward et al., 1981
						2000	No survival		
						2800	No survival		

[A] Test: LC = life-cycle, PLC = partial life-cycle, ELS = early life-stage
[B] Habitat: I = infauna, E = epibenthic, W = water column
[C] NOEC = Concentrations where no significant effects were detected.
[D] OEC = Concentrations where significant effects were detected on survival, growth, or reproduction.

Table 3-2. Summary of acute and chronic values, acute-chronic ratios and derivation of the final acute values, final acute-chronic values and final chronic values.

Common Name, Species	PAH Tested	Value (µg/L)	Value (µg/L)	Chronic Ratio	PAH-Specific Mean Acute-Chronic Ratio	Species Mean Acute-Chronic Ratio	Reference
FRESHWATER SPECIES							
Cladoceran, *Daphnia magna*	Anthracene	-	<2.1	-	-	-	Holst and Giesy, 1989
Cladoceran, *Daphnia magna*	Fluoranthene	117	24.5	4.78	4.78	-	Spehar et al., 1999
Cladoceran, *Daphnia magna*	Phenanthrene	117	96.4	1.21	1.21	2.41	Call et al., 1986
Midge, *Paratanytarsus* sp.	Acenaphthene	2,040[A]	411	4.96	-	-	Northwestern Aquatic Sciences, 1982
Midge, *Paratanytarsus* sp.	Acenaphthene	2,040[A]	227	9	6.68	6.68	Northwestern Aquatic Sciences, 1982; Thursby,1991a
Fathead Minnow, *Pimephales promelas*	Acenaphthene	608	405	1.5	-	-	Cairns and Nebeker, 1982; Thursby, 1991a
Fathead Minnow, *Pimephales promelas*	Acenaphthene	608	419	1.45	1.48	-	Cairns and Nebeker, 1982
Fathead Minnow, *Pimephales promelas*	Acenaphthene	-	73.82	-	-	-	Academy of Natural Sciences, 1981
Fathead Minnow, *Pimephales promelas*	Acenaphthene	-	211	-	-	-	Academy of Natural Sciences, 1981
Fathead Minnow, *Pimephales promelas*	Acenaphthene	-	79.2	-	-	-	ERCO, 1981
Fathead Minnow, *Pimephales promelas*	Acenaphthene	-	112	-	-	-	ERCO, 1981
Fathead Minnow, *Pimephales promelas*	Fluoranthene	69[C]	15	4.6	4.6	2.61	Spehar et al., 1999
Rainbow trout, *Oncorhynchus mykiss*	Phenanthrene	50[C]	6.32	7.9	7.9	7.9	Call et al., 1986

Table 3-2. Continued

Common Name, Species	PAH Tested	Value (µg/L)	Value (µg/L)	Chronic Ratio	PAH-Specific Mean Acute-Chronic Ratio	Species Mean Acute-Chronic Ratio	Reference
SALTWATER SPECIES							
Mysid, *Americamysis bahia*	Acenaphthene	466	286	1.63	-	-	Horne et al., 1983
Mysid, *Americamysis bahia*	Acenaphthene	460	64	7.19	3.42	-	Thursby et al., 1989b
Mysid, *Americamysis bahia*	Fluoranthene	40	15.9	2.52	-	-	U.S. EPA, 1978
Mysid, *Americamysis bahia*	Fluoranthene	31	14.4	2.15	2.33	-	Spehar et al., 1999
Mysid, *Americamysis bahia*	Phenanthrene	27.1	8.13	3.33	3.33	-	Kuhn and Lussier, 1987
Mysid, *Americamysis bahia*	Pyrene	28.3	4.53	6.24	6.24	3.59	Champlin and Poucher, 1992b
Sheepshead minnow, *Cyprinodon variegatus*	Acenaphthene	3,100[B]	710	4.36	4.36	4.36	Ward et al., 1981

[A] Geometric mean of two flow-through measured tests from the same laboratory as conducted the life-cycle tests.
[B] LC50 concentration slightly greater than acenaphthene's water solubility.
[C] EC50 based on immobilization used as the acute value instead of the LC50.

Final Acute Value = 9.31 µmol/g octanol
Final Acute-chronic Ratio = 4.16
Final Chronic Value = 2.24 µmol/g octanol

The FCV at a K_{OW} of 1.0 for PAHs derived in this section of the document differs slightly from that which would be derived for other narcotic chemicals according to Di Toro et al. (2000) in that it: (1) is derived using only acute and chronic toxicity data from water-only tests with freshwater and saltwater species exposed to individual PAHs, therefore, the data do not require the PAH chemical class correction; (2) the data are rigorously screened for acceptability following the requirements for the use of species resident to North America, test durations, test quality, etc. of the U.S. EPA National WQC Guidelines (Stephan et al., 1985). All other steps in the derivation of FCVs are the same as those used by Di Toro et al. (2000).

3.2 Acute Toxicity of Individual PAHS: Water Exposures

3.2.1 Acute Toxicity of PAHs

One hundred and four acute water-only toxicity tests with 12 different PAHs have been conducted on 24 freshwater species from 20 genera that meet the requirements of the U.S. EPA National WQC Guidelines (Stephan et al., 1985, see Appendix C). The tested life-stages of 15 of the genera were benthic (infaunal or epibenthic). The most commonly tested freshwater species were the cladocerans (*Daphnia magna* and *D. pulex*), rainbow trout (*O. mykiss*), fathead minnow (*P. promelas*) and bluegill (*Lepomis macrochirus*). The most commonly tested PAHs with freshwater organisms were acenaphthene, fluoranthene, fluorene, naphthalene, phenanthrene and pyrene.

Seventy-seven acute water-only toxicity tests with 8 different PAHs have been conducted on 30 saltwater species from 29 genera (Appendix C). The tested life-stages of 21 of the genera were benthic (infaunal or epibenthic). The most commonly tested saltwater species were the annelid worm (*N. arenaceodentata*), mysid (*Americamysis bahia*), grass shrimp (*Palaemonetes pugio*), pink salmon (*Oncorhynchus gorbuscha*), and sheepshead minnow (*Cyprinodon variegatus*). The most commonly tested PAHs with saltwater organisms were acenaphthene, fluoranthene, naphthalene, phenanthrene and pyrene.

3.2.2 Acute Values at a K_{OW} of 1.0

The rules for test acceptability of the National WQC Guidelines (Stephan et al., 1985) were used to identify the LC50 values or EC50 (µg/L) values from individual acute aquatic toxicity tests (Appendix C) and these values were used to derive the K_{OW} normalized GMAV (µmol/g octanol) in the following manner. The goal of this process was to convert individual LC50 or EC50 values that vary for a species across PAHs into a PAH-specific GMAV normalized to a K_{OW} of 1.0. The use of normalizing factors in FCV derivation is not unique to this ESB document. The use of K_{OW} to normalize the toxicity of PAHs to put the toxicity data on an internally consistent scale is analogous to the hardness normalization applied to the freshwater WQC for cadmium, copper, lead, nickel and zinc and the pH and temperature normalization applied to the freshwater WQC for ammonia. For multiple PAHs tested against one species, the K_{OW} normalization should result in similar PAH-specific SMAVs. The first step in the analysis of published LC50 or EC50 values was to compare them to the known solubility in water of the PAH tested. If the LC50 or EC50 concentration exceeded the solubility of the tested PAH, the published LC50/EC50 is in parentheses in Appendix C, the solubility is listed in bold in Appendix C as a "greater than" acute value to indicate that the actual toxicity of the dissolved PAH was unknown. For these tests, this greater than solubility value, and not the published LC50 or EC50 value, was used in further calculations only when there were no acute values for that species at concentrations less than the solubility. Next, the LC50, EC50 or greater than solubility value was converted to mmol of the tested PAH/L. When the same PAH was tested more than once against a species, the geometric mean of all LC50 or EC50 values was calculated to determine the PAH-specific SMAV using the rules in Stephan et

al. (1985). The -0.945 universal slope of the toxicity/K_{OW} relationship (Equation 2-29) was applied to the PAH-Specific SMAVs (µmol/L) to calculate the PAH-specific SMAV (µmol/g octanol) at a K_{OW}=1.0. The SMAV for all tested PAHs is the geometric mean of the PAH-Specific SMAVs at a K_{OW} of 1.0. The GMAV (µmol/g octanol) at a K_{OW} of 1.0 is the geometric mean of the SMAVs at a K_{OW} of 1.0.

The SMAVs at a K_{OW} of 1.0 were similar for multiple PAHs (Appendix C). For 18 freshwater and saltwater species, two to nine different PAHs were tested. The ratios of the highest to lowest acute values for multiple PAHs tested against an individual species before normalization was 1.37 to 1170; an average ratio of 105. In contrast, the range in the ratios of the highest to lowest PAH-specific SMAVs at a K_{OW} of 1.0 was 1.4 to 12.2; average ratio of 4.27. For 10 of the 18 (56%) species tested against multiple PAHs, the ratio of high to low SMAVs at a K_{OW} of 1.0 was 4.0 or less. This compares favorably with the factor of four or less difference in the acute values for 12 of 19 (63%) of the same species in multiple tests with the same PAH. Therefore, the variability of SMAVs at a K_{OW} of 1.0 across PAHs is similar to the variability inherent for these data in acute toxicity testing with only one PAH. This suggests that the GMAVs provide data across PAHs that indicate the relative sensitivity of that species that can be used to describe species at risk and to calculate the FAV.

The K_{OW}-normalized GMAVs (not including values greater than the solubility of the tested PAH) range from 7.63 µmol/g octanol for *Americamysis* to 187 µmol/g octanol for *Tanytarsus*, a factor of only 24. Saltwater genera constitute four of the five genera with GMAVs at a K_{OW} of 1.0 within a factor of two of the most sensitive genus (*Americamysis*). Of the 49 genera, the most sensitive one-third include a freshwater hydra, two amphipods, an insect, saltwater fish, a crab, two mysids, two shrimp, and three saltwater amphipods. All of these 16 genera have GMAVs at a K_{OW} of 1.0 that are within a factor of three, and 14 of the genera are benthic. Benthic and water column genera are distributed throughout the sensitivity distributions indicating that they have similar sensitivities. Genera that are benthic have been tested more frequently than water column genera.

3.3 Applicability of the WQC as the Effects Concentration for Benthic Organisms

The use of the FAV or FCV as the effects concentration for calculation of ESBs assumes that benthic (infaunal and epibenthic) species, taken as a group, have sensitivities similar to all aquatic (benthic and water column) species used to derive the WQC FCV. The data supporting the reasonableness of this assumption over all chemicals for which there were published or draft WQC documents were presented in Di Toro et al. (1991) and U.S. EPA (2003a). The conclusion of similarity of sensitivity was supported by comparisons between (1) acute values for the most sensitive benthic species and acute values for the most sensitive water column species for all chemicals; (2) acute values for all benthic species and acute values for all species in the WQC documents across all chemicals after normalizing the LC50 values; (3) FAVs calculated for benthic species alone and FAVs in the WQC documents; and (4) individual chemical comparisons of benthic species versus all species. The following analysis examines the data on the similarity of sensitivity of benthic and all aquatic species for PAHs.

For PAHs, benthic life-stages were tested for 15 of 20 freshwater genera and 21 out of 29 saltwater genera (Appendix C). An initial test of the difference between the freshwater and saltwater FAVs for all species (water column and benthic) exposed to PAHs was performed using the Approximate Randomization (AR) Method (Noreen, 1989). The AR Method tests the significance level of a test statistic when compared to a distribution of statistics generated from many random sub-samples. The test statistic in this case was the difference between the freshwater FAV (computed from the GMAVs at a K_{OW} of 1.0 for combined water column and benthic organisms) and the saltwater FAV (computed from the GMAVs at a K_{OW} of 1.0 for combined water

column and benthic organisms) (Appendix C). In the AR Method, the freshwater and the saltwater GMAVs at a K_{OW} of 1.0 were combined into one dataset. The dataset was shuffled, then separated back so that randomly generated "freshwater" and "saltwater" FAVs could be computed. The LC50 values were re-separated such that the number of GMAVs at a K_{OW} of 1.0 used to calculate the sample FAVs were the same as the number used to calculate the original FAVs. These two FAVs were subtracted and the difference used as the sample statistic. This was done iteratively so that the sample statistics formed a probability distribution representative of the population of FAV differences (Figure 3-1A). The test statistic was compared to this distribution to determine its level of significance. The null hypothesis was that the GMAVs at a K_{OW} of 1.0 that comprise the freshwater and saltwater data bases were not different. If this was true, the difference between the actual freshwater and saltwater FAVs should be common to the majority of randomly generated FAV differences. For PAHs, the test-statistic occurred at the 93.5 percentile of the generated FAV differences (Table 3-3). This percentile suggests that saltwater genera may be somewhat

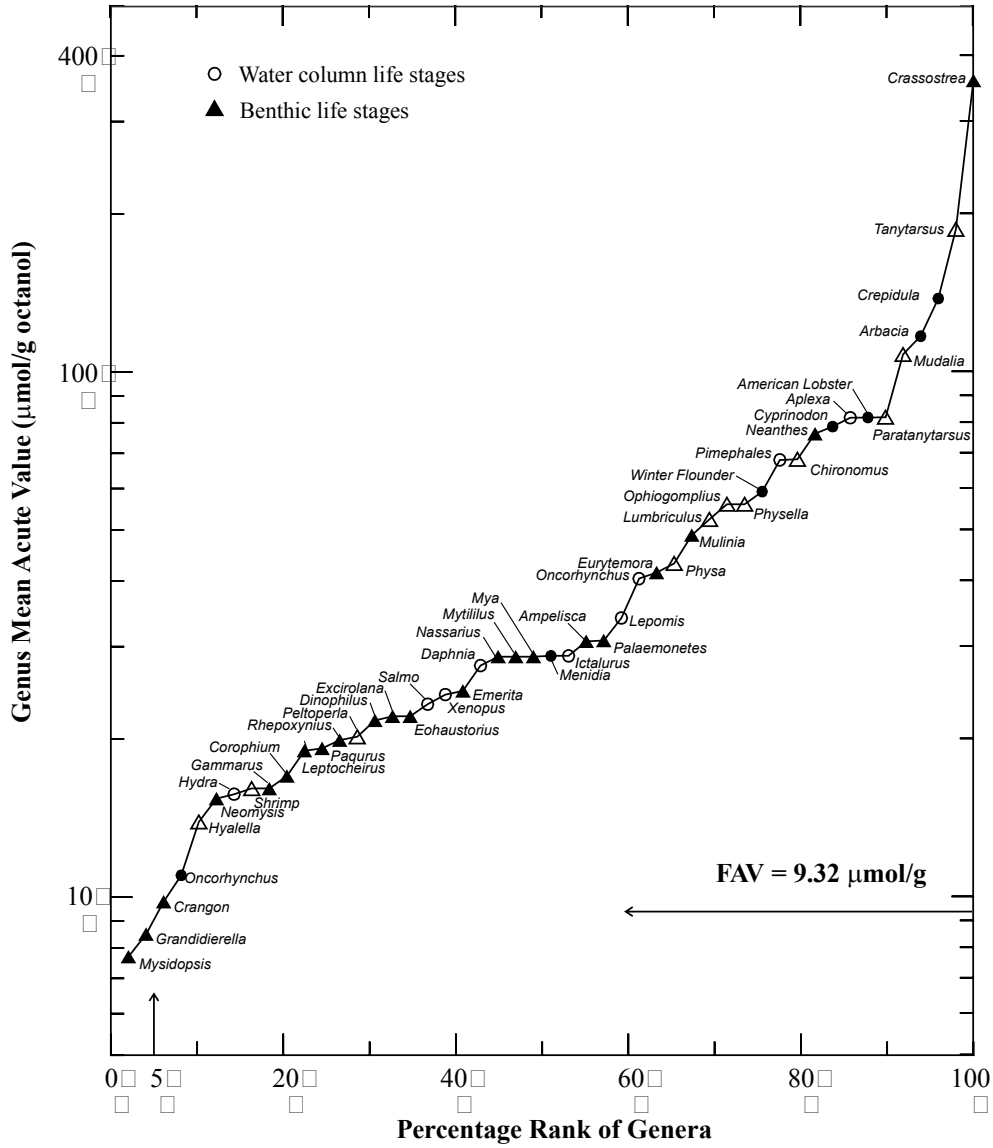


Figure 3-1 Probability distributions of FAV difference statistics to compare water-only toxicity data from (A) freshwater versus saltwater genera and (B) benthic versus WQC.

Table 3-3. Results of approximate randomization (AR) test for the equality of the freshwater and saltwater FAV distributions at a K_{OW} of 1.0 and AR test for the equality of benthic and combined benthic and water column FAVs for freshwater and saltwater distributions.

Comparison	Habitat or Water Type[A]		AR Statistic[B]	Probability[C]
Fresh vs Salt	Fresh (20)	Salt (29)	5.746	93.5
Freshwater: Benthic vs WQC[D]	WQC (49)	Benthic (33)	0.862	82.8

[A] Values in parantheses are the number of GMAVS at a K_{OW} of 1.0 used in the comparison.
[B] AR statistic = FAV difference between original compared groups.
[C] Probability that the theoretical AR statistic ≤ the observed AR statistic given that all samples came from the same population.
[D] Combined freshwater and saltwater.

more sensitive than freshwater genera as illustrated in Figure 3-2 and Appendix C. However, since the probability was less than 95% in the AR analysis, the null hypothesis of no significant difference in sensitivity for freshwater and saltwater species was accepted (Table 3-3).

Since freshwater and saltwater species showed no significant differences in sensitivity, the AR Method was applied jointly for the analysis of the difference in sensitivity for benthic and all aquatic organisms (benthic and water column species are always combined to derive WQC, therefore, the complete GMAV dataset is hereafter referred to as "WQC"). Using the criteria in U.S. EPA (2003a), each life stage of each test organism, hence each GMAV at a K_{OW} of 1.0, was assigned a habitat (Appendix C). The test statistic in this case was the difference between the WQC FAV, computed from the WQC GMAVs at a K_{OW} of 1.0, and the benthic FAV, computed from the benthic organism GMAVs at a K_{OW} of 1.0. The approach used to conduct this analysis was slightly different than that used in the previous test for freshwater and saltwater GMAVs. The difference was that freshwater and saltwater GMAVs in the first test represented two separate groups. In this test, the GMAVs at a K_{OW} of 1.0 for benthic organisms were a subset of the GMAVs at a K_{OW} of 1.0 in the entire WQC dataset. In the AR analysis for this test, the number of data points coinciding with the number of benthic organisms were selected from the WQC dataset to compute each "benthic" FAV. The original WQC FAV and the "benthic" FAV were then used to compute the difference statistic. This was done iteratively and the distribution that results was representative of the population of FAV difference statistics. The test statistic was compared to this distribution to determine its level of significance. The probability distributions of the computed FAV differences are shown in Figure 3-1B. The test statistic for this analysis occurred at the 82.8 percentile and the null hypothesis of no difference in the sensitivities between benthic species and species used to derive the WQC FCV was accepted (Table 3-3). This analysis supports the derivation of the FCV for PAHs based on all GMAVs at a K_{OW} of 1.0.

3.4 Derivation of the FAV at a K_{OW} of 1.0

The FAV is an estimate of the concentration corresponding to a cumulative probability of 0.05 in the GMAVs at a K_{OW} of 1.0. The analysis above demonstrates that the acute sensitivities of freshwater and saltwater genera and the sensitivities of benthic and benthic plus water column genera do not differ. Therefore, for calculation of the FAV, the GMAVs at a K_{OW} of 1.0 for all freshwater and saltwater genera can be grouped together to represent the relative sensitivities of all benthic organisms (Figure 3-2). The FAV at a K_{OW} of 1.0 is calculated using the procedure in Stephan et al. (1985), the GMAVs at

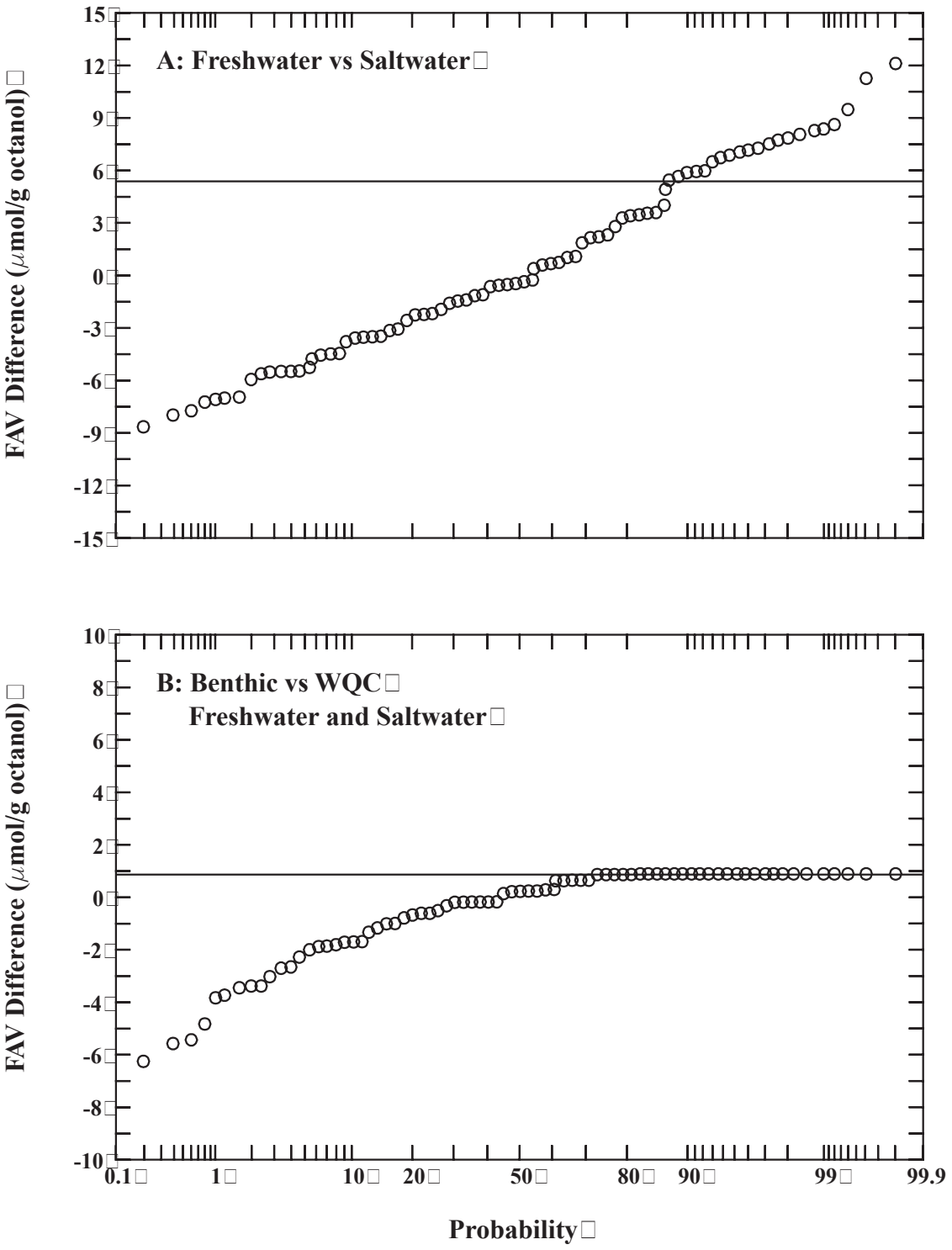


Figure 3-2. GMAVs at a $\log_{10}K_{OW}$ of 1.0 from water-only acute toxicity tests using freshwater and saltwater genera versus percentage rank of their sensitivity.

a K_{OW} of 1.0 of 7.63 µmol/g octanol for *Americamysis*, 8.51 µmol/g octanol for *Grandidierella*, 9.83 µmol/g octanol for *Crangon*, 11.0 µmol/g octanol for *Oncorhynchus* and the total number of genera tested (N = 49). The FAV at a K_{OW} of 1.0 is 9.31 µmol/g octanol. This FAV is greater than the GMAVs of the two most acutely sensitive genera as would be expected given the calculation procedure and the presence of 31 GMAVs.

3.5 Chronic Toxicity of Individual PAHs: Water Exposures

3.5.1 Acenaphthene

Chronic life-cycle toxicity tests have been conducted with acenaphthene with the freshwater midge (*Paratanytarsus* sp.) and the saltwater mysid (*A. bahia*), and early life-stage tests have been conducted with the fathead minnow (*P. promelas*) and sheepshead minnow (*C. variegatus*) (Table 3-1). For each of these species, one or more benthic life-stages were exposed. Other chronic toxicity tests have been conducted with the freshwater chironomid (*Paratanytarsus* sp.) and *P. promelas* (Lemke et al.,1983; Lemke, 1984; Lemke and Anderson, 1984) but insufficient documentation is available to permit use of these results (Thursby, 1991a).

Two acceptable life-cycle toxicity tests have been conducted with *Paratanytarsus* sp. (North-western Aquatic Sciences, 1982). In the first test, 575 µg/L reduced survival 90%, reduced growth 60%, and all eggs failed to hatch (Table 3-1). No adverse effects occurred at acenaphthene concentrations up to 295 µg/L acenaphthene. In the second test, survival was reduced 20% and growth 30% at 315µg/L. Egg hatchability was not affected in the highest concentration of 676 µg/L; although survival of hatched larvae was reduced ~60%. No significant effects were observed at acenaphthene concentrations up to 164 µg/L.

A total of six early life-stage toxicity tests have been conducted with *P. promelas* as part of a round-robin test series; two each from three laboratories (Table 3-1) (Academy of Natural Sciences, 1981; ERCO, 1981; Cairns and Nebeker, 1982). The lowest observed effect concentrations (LOEC) across laboratories and tests ranged from 98 to 509 µg/L, a factor of 5.2. Growth (dry weight), survival, or both growth and survival were reduced. Only one of these test pairs had a suitable measured acute value that allowed calculation of an ACR (Cairns and Nebeker, 1982). The concentration-response relationships were similar for the two tests of Cairns and Nebeker (1982). In the first test, the early life-stages of this fish were unaffected in acenaphthene concentrations ranging from 67 to 332 µg/L, but 495 µg/L reduced growth 54% relative to control fish. In the second test, growth was reduced 30% at 509 µg/L, but no effects were detected in fish exposed to 197 to 345 µg/L.

Data from saltwater chronic toxicity tests with acenaphthene are available for *A. bahia* and *C. variegatus*. Reproduction of *A. bahia* was affected by acenaphthene in two life-cycle tests from two different laboratories. In the first test (Horne et al., 1983), 340 µg/L reduced reproduction 93% relative to controls and all *A. bahia* died at 510 µg/L. No effects were observed on the parental generation at 100 to 240 µg/L and second generation juveniles were not affected at ≤ 340 µg/L. In the second test (Thursby et al., 1989b), no effects were observed at ≤ 44.6 µg/L, but a concentration of 91.8 µg/L reduced reproduction 91%. No reproduction occurred at higher concentrations, and growth was reduced 34% at 168 µg/L and survival 96% at 354 µg/L.

A test with early life-stages of *C. variegatus* showed that 240 to 520 µg/L had no effects, but that concentrations of 970, 2,000 and 2,800 µg/L reduced survival of embryos and larvae by ≥70% (Table 3-1; Ward et al., 1981).

In general, the above results show that the difference between acute and chronic toxicity of acenaphthene is small and differed minimally between species (Table 3-2). Species mean acute-chronic ratios for acenaphthene are 6.68 for *Paratanytarsus* sp., 1.48 for *P. promelas*, 3.42 for *A. bahia* and 4.36 for *C. variegatus*.

3.5.2 Anthracene

A single life-cycle toxicity test has been conducted with *D. magna* exposed to only three concentrations of anthracene (Holst and Geisy, 1989). Minimal decreases were observed on the number of broods produced in all three of the concentrations tested: 2.1 mg/L (5.3%), 4.0 µg/L (8.0%) and 8.2 µg/L (13.8%). No acute toxicity tests were conducted by the authors. Therefore, an ACR could not be derived for anthracene.

3.5.3 Fluoranthene

Fluoranthene has been tested in life-cycle toxicity tests with the freshwater cladoceran, *D. magna* (Spehar et al., 1999) and the saltwater mysid, *A. bahia* (U.S. EPA, 1978, Spehar et al., 1999), and early life-stage tests have been conducted with the fathead minnow (Spehar et al., 1999) (Table 3-1). No effects were observed with *D. magna* at ≤17 µg/L, but growth was reduced 17% at 35 µg/L and 25% at 73 µg/L. There were 37% fewer young per adult at 73 µg/L and no daphnids survived at 148 µg/L. An early life-stage toxicity test conducted with the fathead minnow showed no effects at ≤10.4 µg/L, but reduced survival (67%) and growth (50%) at 21.7 µg/L.

Saltwater mysids (*A. bahia*) were tested in two life-cycle toxicity tests. In the first test, the mysids were exposed to fluoranthene for 28 days (U.S. EPA, 1978). There was no effect on survival or reproduction (growth was not measured) in concentrations ranging from 5-12 µg/L. At a fluoranthene concentration of 21 µg/L, survival was reduced 26.7% and reproduction 91.7%, relative to the controls. At the highest concentration of fluoranthene, 43 µg/L, all *A. bahia* died. In the second test, *A. bahia* were exposed to fluoranthene for 31 days (Spehar et al., 1999). Effect concentrations were similar to those in the U.S. EPA (1978) test. *A. bahia* were not affected at fluoranthene concentrations from 0.41-11.1 µg/L. At the highest concentration tested, 18.8 µg/L, survival was reduced 23% relative to controls and there was no reproduction. Reproduction was reduced by 77% in 11.1 µg/L, but this was not significantly different from controls even at α=0.1.

The difference between acute and chronic sensitivity to fluoranthene varied minimally between species (Table 3-2). Three species mean ACRs are available for fluoranthene: 4.78 for *D. magna*, 4.60 for *P. promelas*, and 2.33 for *A. bahia*.

3.5.4 Phenanthrene

Phenanthrene has been tested in life-cycle toxicity tests with *D. magna* and *A. bahia* and an early life-stage test has been conducted with rainbow trout (*O. mykiss*) (Table 3-1). There were no effects of phenanthrene on *D. magna* at ≤57 µg/L, but survival was reduced 83% and reproduction 98% at 163 µg/L (Call et al., 1986). In a test with *O. mykiss*, no effects were observed at 5 µg/L. The percentage of abnormal and dead fry at hatch was significantly increased at the highest exposure concentration of 66 µg/L and survival of hatched fry was reduced with increase in exposure concentration (Call et al., 1986). Mortality was 41, 48, 52 and 100% at 8, 14, 32, and 66 µg/L, respectively. Wet weight was reduced 33, 44, and 75% at 8, 14 and 32 µg/L, respectively.

A life-cycle toxicity test with *A. bahia* exposed to phenanthrene showed that the effect concentrations were similar to those that affected *O. mykiss* (Kuhn and Lussier, 1987) (Table 3-1). Survival, growth and reproduction were not affected at ≤5.5 µg/L. However, at the highest test concentration of phenanthrene (11.9 µg/L), all mysids died.

The difference between acute and chronic sensitivity to phenanthrene varied minimally between *D. magna* (PAH-specific ACR= 1.21), *O. mykiss* (ACR=7.90) and *A. bahia* (ACR= 3.33). The ACR for *O. mykiss* (Call et al., 1986) was derived using the EC50 for immobilization (50 µg/L) and not the 96-hour LC50 of 375 µg/L as was required in Stephan et al. (1985).

3.5.5 Pyrene

A life-cycle toxicity test with *A. bahia* exposed to pyrene was conducted by Champlin and Poucher (1992b). There were no effects at 3.82 µg/L, but

20.9 µg/L reduced survival 37% and no mysids survived at the next higher concentration of 38.2 µg/L (Table 3-1). Reproduction was significantly reduced in ≥5.37 µg/L. The ACR from this test was pyrene is 6.24.

3.5.6 Naphthalene

Fathead minnows were exposed to naphthalene in an early life-stage toxicity test (DeGraeve et al., 1982). Hatching of fry was significantly reduced in 4.38 and 8.51 µg/L and none were alive in these concentrations at the end of the 30-day test. Weight and length of fish surviving the test were significantly reduced in 0.85 and 1.84 µg/L. No significant effects were detected in concentrations ≤0.45 µg/L. Control survival was only 42%, which does not meet requirements according to the American Society of Testing and Materials (ASTM, 1998). Also, the carrier methanol was absent from the control. These data are summarized in the text for completeness, but the ACR of 12.7, chronic value of 0.62 µg/L, and 96-hour LC50 of 7.9 µg/L for naphthanlene are not included in Tables 3-1 and 3-2.

The calanoid copepod (*Eurytemora affinis*) was exposed indvidually to 14.21 µg/L naphthalene, 15.03 µg/L 2-methylnaphthalene, 8.16 µg/L 2,6-dimethylnaphthalene and 9.27 µg/L 2,3,5-trimethylnaphthalene in life-cycle toxicity tests (Ott et al., 1978). Survival and reproduction were affected by each of the naphthalenes, but ACRs could not be derived because the duration of the acute test was too short (24 hours) according to WQC Guidelines (Stephan et al., 1985), and no other concentrations were tested chronically.

3.5.7 Derivation of the Final Acute Chronic Ratio

The FACR for the six PAHs is 4.16. This FACR is the geometric mean of all species mean ACRs for *Daphnia* (2.41), *Paratanytarsus* (6.68), *Pimephales* (2.61), *Oncorhynchus* (7.90), *Americamysis* (3.59), and *Cyprinodon* (4.36) (Table 3-2).

3.6 Derivation Of FCVs

3.6.1 Derivation of the FCV at a K_{OW} of 1.0

The FCV is the value that should protect 95% of the tested species. The FCV is the quotient of the FAV and the FACR for the substance. The FAV at a K_{OW} of 1.0 is 9.31 mmol/g octanol. It is an estimate of the acute LC50 or EC50 concentration corresponding to a cumulative probability of 0.05 for the GMAVs at a K_{OW} of 1.0. The FACR of 4.16 is the mean ratio of acute to chronic toxicity for six species exposed exposed both acutely and chronically to one or more of six individual PAHs in 15 experiments. (For more information on the calculation of ACRs, FAVs, and FCVs see the U.S. EPA National WQC Guidelines (Stephan et al., 1985.))

The FAV at a K_{OW} of 1.0 of 9.31 µmol/g octanol is divided by the FACR of 4.16 to obtain a FCV at a K_{OW} of 1.0 of 2.24 µmol/g octanol (Table 3-3). Because nonionic organic chemicals partition similarly into octanol and lipid of organisms, the FCV at a K_{OW} of 1.0 in µmol/g octanol approximately equals tissue-based "acceptable" concentration of about 2.24 µmol/g lipid.

3.6.2 Derivation of the PAH-Specific FCVs

The PAH-specific FCVs (mg/L) (Table 3-4, Appendix D) are calculated from the FCV at a K_{OW} of 1.0 (µmol/g octanol), the slope of the K_{OW}-K_{OC} relationship, the universal narcotic slope of the K_{OW}-acute toxicity relationship, and the PAH-specific K_{OW} values (Equation 3-1, 3-2, and 3-3).

$$\log_{10}\text{PAH-specific FCV} = (\text{slope})\log_{10}K_{OW} + \log_{10}\text{FCV at a } K_{OW} \text{ of 1.0} \quad (3\text{-}1)$$

$$\log_{10}\text{PAH-specific FCV} = -0.945\log_{10}K_{OW} + \log_{10}(2.24) \quad (3\text{-}2)$$

$$\text{PAH-specific FCV (mmol/L)} = 1000(\text{antilog}(-0.945\log_{10}K_{OW} + 0.3502)) \quad (3\text{-}3)$$

Table 3-4. $C_{OC,PAHi,FCVi}$ concentrations and properties required for their derivation[A].

PAH[B]	SPARC[C] $\log_{10}K_{ow}$	$\log_{10}K_{oc}$	FCV_i (µmol/g octanol)	PAH specific FCV_i (µmol/L)	PAH specific FCV_i (µg/L)	$C_{OC,PAHi,FCVi}$ (µg/goc)	$C_{OC,PAHi,MAXi}$[D] (µg/goc)
indan	3.158	3.105	2.24	2.322	274.5	349	127200
naphthalene	3.356	3.299	2.24	1.509	193.5	385	61700
C1-naphthalenes	3.8	3.736	2.24	0.5744	81.69	444	-
1-methylnaphthalene	3.837	3.772	2.24	0.53	75.37	446	165700
2-methylnaphthalene	3.857	3.792	2.24	0.5074	72.16	447	154800
acenaphthylene	3.223	3.168	2.24	2.016	306.9	452	24000
acenaphthene	4.012	3.944	2.24	0.3622	55.85	491	33400
1-ethylnaphthalene	4.221	4.15	2.24	0.2298	35.91	507	142500
2-ethylnaphthalene	4.283	4.21	2.24	0.2008	31.37	509	129900
C2-naphthalenes	4.3	4.227	2.24	0.1935	30.24	510	-
1,4-dimethylnaphthalene	4.3	4.227	2.24	0.1935	30.24	510	192300
1,3-dimethylnaphthalene	4.367	4.293	2.24	0.1673	26.13	513	157100
2,6-dimethylnaphthalene	4.373	4.299	2.24	0.1651	25.79	513	33800
2,3-dimethylnaphthalene	4.374	4.3	2.24	0.1647	25.74	513	49900
1,5-dimethylnaphthalene	4.378	4.304	2.24	0.1633	25.52	514	62400
fluorene	4.208	4.137	2.24	0.2364	39.3	538	26000
C3-naphthalenes	4.8	4.719	2.24	0.0652	11.1	581	-
2,3,5-trimethylnaphthalene	4.858	4.776	2.24	0.05747	9.785	584	-
1,4,5-trimethylnaphthalene	4.872	4.789	2.24	0.05575	9.488	584	129300
anthracene	4.534	4.457	2.24	0.1163	20.73	594	1300
phenanthrene	4.571	4.494	2.24	0.1073	19.13	596	34300
C1-fluorenes	4.72	4.64	2.24	0.0776	13.99	611	-
1-methylfluorene	4.739	4.659	2.24	0.07445	13.42	612	49700
C4-naphthalenes	5.3	5.21	2.24	0.02197	4.048	657	-
2-methylanthracene	4.991	4.906	2.24	0.04303	8.273	667	2420
1-methylanthracene	4.998	4.913	2.24	0.04238	8.148	667	-
9-methylanthracene	5.006	4.921	2.24	0.04165	8.007	668	21775
2-methylphenanthrene	5.029	4.944	2.24	0.03961	7.616	669	-
1-methylphenanthrene	5.037	4.952	2.24	0.03893	7.485	670	24100
C1-phenanthrene/anthracenes	5.04	4.955	2.24	0.03868	7.436	670	-
9-ethylfluorene	4.973	4.889	2.24	0.04475	8.693	673	-
C2-fluorenes	5.2	5.112	2.24	0.02731	5.305	686	-
pyrene	4.922	4.839	2.24	0.05	10.11	697	9090
fluoranthene	5.084	4.998	2.24	0.03515	7.109	707	23870
2-ethylanthracene	5.357	5.266	2.24	0.0194	4.003	739	-
C2-phenanthrene/anthracenes	5.46	5.367	2.24	0.01551	3.199	746	-
9,10-dimethylanthracene	5.494	5.401	2.24	0.0144	2.971	748	14071
3,6-dimethylphenanthrene	5.515	5.422	2.24	0.01376	2.838	749	-
C3-fluorenes	5.7	5.603	2.24	0.009199	1.916	769	-
C1-pyrene/fluoranthenes	5.287	5.197	2.24	0.0226	4.887	770	-
2,3-benzofluorene	5.539	5.445	2.24	0.01306	2.824	787	558

Table 3-4. Continued

PAH[B]	SPARC[C] $\log_{10}K_{ow}$	$\log_{10}K_{oc}$	FCV_i (µmol/g octanol)	PAH specific FCV_i (µmol/L)	PAH specific FCV_i (µg/L)	$C_{OC,PAHi,FCVi}$ (µg/goc)	$C_{OC,PAHi,MAXi}$[D] (µg/goc)
benzo(a)fluorene	5.539	5.445	2.24	0.01306	2.824	787	12500
C3-phenanthrene/anthracenes	5.92	5.82	2.24	0.0057	1.256	829	-
naphthacene	5.633	5.538	2.24	0.01064	2.43	838	207
benz(a)anthracene	5.673	5.577	2.24	0.009756	2.227	841	4153
chrysene	5.713	5.616	2.24	0.008943	2.042	844	826
triphenylene	5.752	5.654	2.24	0.008215	1.875	846	19400
C4-phenanthrenes/anthracenes	6.32	6.213	2.24	0.002387	0.5594	913	-
C1-benzanthracene/chrysenes	6.14	6.036	2.24	0.003531	0.8557	929	-
C3-pyrene/fluoranthenes	6.284	6.177	2.24	0.002581	0.6307	949	-
benzo(a)pyrene	6.107	6.003	2.24	0.003794	0.9573	965	3840
perylene	6.135	6.031	2.24	0.00357	0.9008	967	431
benzo(e)pyrene	6.135	6.031	2.24	0.00357	0.9008	967	4300
benzo(b)fluoranthene	6.266	6.16	2.24	0.002685	0.6774	979	2169
benzo(j)fluoranthene	6.291	6.184	2.24	0.002542	0.6415	981	3820
benzo(k)fluoranthene	6.291	6.184	2.24	0.002542	0.6415	981	1220
C2-benzanthracene/chrysenes	6.429	6.32	2.24	0.001883	0.4827	1008	-
9,10-dimethylbenz(a)anthracene	6.567	6.456	2.24	0.001395	0.3575	1021	124200
7,12-dimethylbenz(a)anthracene	6.575	6.464	2.24	0.00137	0.3513	1021	145300
7-methylbenzo(a)pyrene	6.537	6.426	2.24	0.001489	0.3965	1058	-
benzo(ghi)perylene	6.507	6.397	2.24	0.001589	0.4391	1095	648
C3-benzanthracene/chrysenes	6.94	6.822	2.24	0.0006194	0.1675	1112	-
indeno(1,2,3-cd)pyrene	6.722	6.608	2.24	0.0009953	0.275	1115	-
dibenz(a,h)anthracene	6.713	6.599	2.24	0.001015	0.2825	1123	2389
dibenz(a,j)anthracene	6.713	6.599	2.24	0.001015	0.2825	1123	47680
dibenz(a,c)anthracene	6.78	6.665	2.24	0.0008773	0.2442	1129	7400
C4-benzanthracene/chrysenes	7.36	7.235	2.24	0.0002483	0.07062	1214	-
C1-dibenz(a,h)anthracenes	7.113	6.992	2.24	0.0004251	0.1243	1221	-
coronene	6.885	6.768	2.24	0.0006981	0.2097	1230	821
C2-dibenz(a,h)anthracenes	7.513	7.386	2.24	0.000178	0.05454	1325	-
C3-dibenz(a,h)anthracenes	7.913	7.779	2.24	0.0000746	0.02389	1435	-

[A] Four significant figures are used even when fewer are appropriate for the parameter to limit the effects of rounding error when calculating $\Sigma ESBTU_{FCV}$ which has two significant figures.

[B] See Appendix E for solubilities.

[C] For C#-PAHs, reported $\log_{10}K_{OW}$ values are the average $\log_{10}K_{OW}$ values of all structures.

[D] $C_{OC,PAHi,Maxi}$ is based on solubility; if $C_{OC,PAHi,FCVi}$ is > $C_{OC,PAHi,Maxi}$, then $C_{OC,PAHi,Maxi}$ may be used to calculate ESB toxic units (see Section 6).

Section 4

Derivation of PAH ESB_{FCVS}

4.1 Derivation of Potencies for Individual PAHs in Sediments ($C_{OC,PAHi,FCVi}$)

The critical concentration of a PAH in sediment ($C_{OC,PAHi,FCVi}$) that is related to the FCV is derived following the EqP method (U.S. EPA, 2003a; Di Toro et al., 1991) because the interstitial water-sediment partitioning of PAHs follows that of other nonionic organic chemicals. Therefore, a sediment effects concentration for any measure of effect can be derived from the product of the water-only effects concentration for that effect and the K_{OC} for that particular PAH. The use of K_{OC} to derive a sediment effects concentration for PAHs is applicable because partitioning for these chemicals is primarily determined by the organic carbon concentration of the sediment.

The partitioning equation between the organic carbon-normalized sediment concentration, C_{OC} ($\mu mol/g_{OC} = \mu mol/kg_{OC}$), and the free interstitial water concentration, C_d (mmol/L), is given by the equation

$$C_{OC} = K_{OC}\, C_d \qquad (4\text{-}1)$$

where K_{OC} (L/kg_{OC}), defined above, can be calculated from a K_{OW} obtained from SPARC (Hilal et al., 1994) using the following equation from Di Toro (1985)

$$\log_{10}K_{OC} = 0.00028 + 0.983 \log_{10}K_{OW} \qquad (4\text{-}2)$$

$C_{OC,PAHi,FCVi}$ for individual PAHs are then calculated using Equation 4-1 with the FCV as the water concentration

$$C_{OC,PAHi,FCVi} = K_{OC}\, FCV_i \qquad (4\text{-}3)$$

Since K_{OC} is presumed to be independent of sediment type for nonionic organic chemicals, so also is $C_{OC,PAHi,FCVi}$.

Table 3-4 contains the $C_{OC,PAHi,FCVi}$ ($\mu g/g_{OC}$) for 74 PAHs found in sediments, including the 34 PAHs (in bold) analyzed by the U.S. EPA in their EMAP program (U.S. EPA, 1996a,b; 1998). $C_{OC,PAHi,FCVi}$ values for PAHs not in Table 3-4 can be calculated in a similar manner (see Section 7.2 for discussion on the PAHs to which the ESB applies). The range in the $C_{OC,PAHi,FCVi}$ values for the 74 PAHs listed in Table 3-4, which were derived using only data for PAHs, is from 349 to 1435 $\mu g/g_{OC}$. In contrast, the range of the same value, termed the $C_{S,OC}$ by Di Toro and McGrath (2000), was about the same (655 to 1940 $\mu g/g_{OC}$) for the 23 PAHs commonly measured when derived using the database for narcotic chemicals with a PAH correction.

4.2 Derivation of the ESB_{FCV} for PAH Mixtures

The correct derivation of the ESB for a mixture of PAHs is based on the approximate additivity of narcotic chemicals in water and tissue (Di Toro et al., 2000; Section 2.8 of this document) and in sediment (Section 5.2). Because WQC and ESBs are based on FCVs they are not intended to cause toxicity in water or sediments to most species, the term toxic unit could be misleading. Therefore, we refer to the quotient of the concentration of a specific chemical in water and its WQC FCV as water quality criteria toxic units ($WQCTU_{FCVi}$). Similarly, the quotient of the sediment concentration for a specific PAH ($C_{OC,PAHi}$) and the $C_{OC,PAHi,FCVi}$ in sediments should be termed equilibrium partitioning sediment benchmark toxic unit ($ESBTU_{FCVi}$). Thus, the ESB for the mixture of PAHs is the sum of the $ESBTU_{FCVi}$ for all of the PAHs in the particular sediment termed the $\Sigma ESBTU_{FCV}$

$$\Sigma ESBTU_{FCV} = \sum_{i} \frac{C_{OC,PAHi,}}{C_{OC,PAHi,FCVi}} \qquad (4\text{-}4)$$

For a particular sediment, if the $\Sigma ESBTU_{FCV}$ for "total PAHs" is less than or equal to 1.0, the concentration of the mixture of PAHs in the sediment is acceptable for the protection of benthic organisms (see Section 7.2 for the technical basis for defining total PAH as the $\Sigma ESBTU_{FCV}$ for the 34 PAHs monitored in the U.S. EPA EMAP). The equilibrium partitioning sediment benchmark is given by the equation

$$ESB = \Sigma ESBTU_{FCV} \leq 1.0 \quad (4\text{-}5)$$

For a particular sediment, if the $EESBTU_{FCV}$ is > 1.0, the concentration of the mixture of PAHs in the sediment may not acceptable for the protection of benthic organisms

$$ESB = \Sigma ESBTU_{FCV} > 1.0 \quad (4\text{-}6)$$

4.3 Aqueous Solubility Constraint

A solubility constraint is applied to sediment concentrations when computing their individual contributions to the effect of the PAH mixture because the $C_{OC,PAHi,FCVi}$ derived for each PAH is solubility limited, i.e., the interstitial water concentration of the PAH is limited by the solubility S. Therefore, $C_{OC,PAHi,FCVi}$ is limited by the concentration in sediment organic carbon that is in equilibrium with the interstitial water at the aqueous solubility (Equation 4-7). This is termed the maximum $C_{OC,PAHi,Max}$ (Table 3-4)

$$C_{OC,PAHi,FCVi} \leq C_{OC,PAHi,Maxi} = K_{OC}S \quad (4\text{-}7)$$

Thus, only the contribution up to the maximum $C_{OC,PAHi,Maxi}$ is counted in the $\Sigma ESBTU_{FCV}$ for the PAH mixture.

Narcosis theory suggests that highly insoluble PAHs should contribute fractional toxic units and $ESBTU_{FCVi}$, limited by the solubility constraint, to the sum of the effects of the mixture when these PAHs are present in mixtures. If so, then this points out the importance of knowing the aqueous solubility of these PAHS so that Equations 4-4 and 4-5 can be applied correctly.

The question of whether highly insoluble chemicals that are not by themselves acutely or chronically toxic, e.g., high molecular weight PAHs, contribute fractional toxic units to the total toxicity when present as mixtures is discussed in Section 5.2.8 of this document and in Spehar et al. (*In preparation*). Spehar et al. (*In preparation*) demonstrate that high K_{OW} PAHs do contribute to the total toxicity of the PAH mixture.

4.4 Comparison of the $\Sigma ESBTU_{FCV}$ for Mixtures of PAHS in Estuarine Sediments

Coastal and estuarine monitoring data were compiled from eight sources to obtain a preliminary assessment of the $\Sigma ESBTU_{FCV}$ values for PAHs in the sediments of the Nation's water bodies (NOAA, 1991; Adams et al., 1996; Anderson et al., 1996; Fairey et al., 1996; U.S. EPA, 1996a,b, 1998; Hunt et al., 1998). Data sources which were identified had measured concentrations for the 23 PAHs (18 parent and 5 alkylated groups) (see Table 6-2) as well as the corresponding sediment organic carbon measurements. Sediments analyzed were from randomly selected and specifically targeted locations, samples of surficial grabs and vertical profiles, and studies where the relative frequency and intensities of sampling varied. This analysis is presented as an aid in assessing the range of reported PAH concentrations, and the extent to which they may exceed 1.0 $\Sigma ESBTU_{FCV}$. The sediments analyzed were not randomly selected from the entire United States. Therefore, this analysis is not intended to reflect expected occurrence nationwide or at any specific site of concern. Sediments where 23 PAHs were analyzed will underestimate the $\Sigma ESBTU_{FCV}$ if 34 PAHs had been analyzed. $\Sigma ESBTU_{FCV}$ values were computed by summing the $ESBTU_{FCVi}$ for each PAH measured in the sediment sample. For insoluble PAHs, the $C_{OC,PAHi,Maxi}$ (Table 3-4) was used to calculate $\Sigma ESBTU_{FCV}$.

The probability distribution for the $\Sigma ESBTU_{FCV}$ data are shown on Figure 4-1. The number of data points used to generate each distribution is provided in the lower right hand corner of each graph. For visual effect, only non-

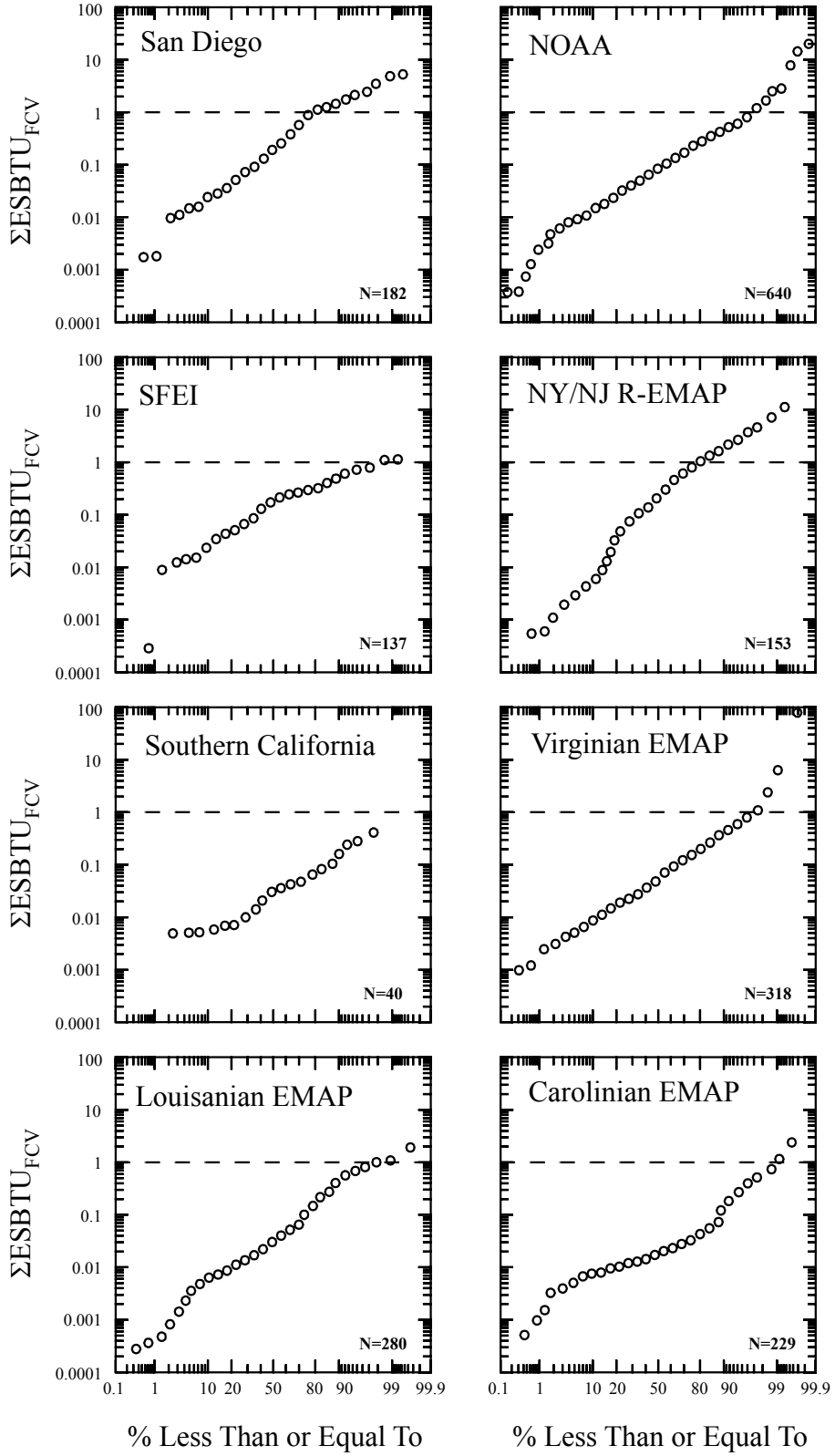


Figure 4-1. Probability distribution of the $\Sigma ESBTU_{FCV}$ for PAH mixtures in sediments from individual coastal and estuarine locations in the United States.

overlapping data are shown. For comparison purposes, a line indicating 1.0 $\Sigma ESBTU_{FCV}$ is also shown. Data presented are from sediments with 0.201 to 15.2% organic carbon. With the exception of the Louisianian and Carolinian Province EMAP datasets, all of the datasets had only 23 PAHs measured. The Louisianian and Carolinian Province EMAP datasets had a total of 34 measured PAHs (18 parent and 16 alkylated groups). The PAHs in addition to the 23 were the C1 through C4 alklyated forms of some of the parent PAHs. To assess the total number of PAHs, a C1-PAH series was considered as one PAH. Computed $\Sigma ESBTU_{FCV}$ values are based on the total number of PAHs measured. The distributions across the different locations are relatively similar. With the exception of the Southern Californian data, all of the datasets had $\Sigma ESBTU_{FCV}$ values greater than 1.0 at the 95th percentile. Although the $\Sigma ESBTU_{FCV}$ from the Lousianian and Carolinian Province EMAP data are computed from 34 PAHs, these sediments do not contain greater $\Sigma ESBTU_{FCV}$ values than sediments from the other studies which measured only 23 PAHs.

A single probability distribution using all of the data is shown in Figure 4-2. The total number of sediments is 1979. $\Sigma ESBTU_{FCV}$ values computed from 23 PAHs are denoted by open circles, and for the 34 PAHs, by open squares. The median $\Sigma ESBTU_{FCV}$ was about 0.06. Approximately 6% of the samples (109 sediments) had $\Sigma ESBTU_{FCV}$ values greater than 1.0.

Although the EqP-based ESBs for nonionic organic chemicals are not intended for use with largely sandy sediments having <0.2% TOC, the EMAP Lousianian and Carolinian Provinces (34 PAHs) and the Elliot Bay (31 PAHs) monitoring databases were examined to determine the frequency of ESB exceedences. A total of 115 of the 654 sediments in these databases had <0.2% TOC. Only two of these sediments (1.7 percent) exceeded the ESB of >1.0 $\Sigma ESBTU_{FCV}$.

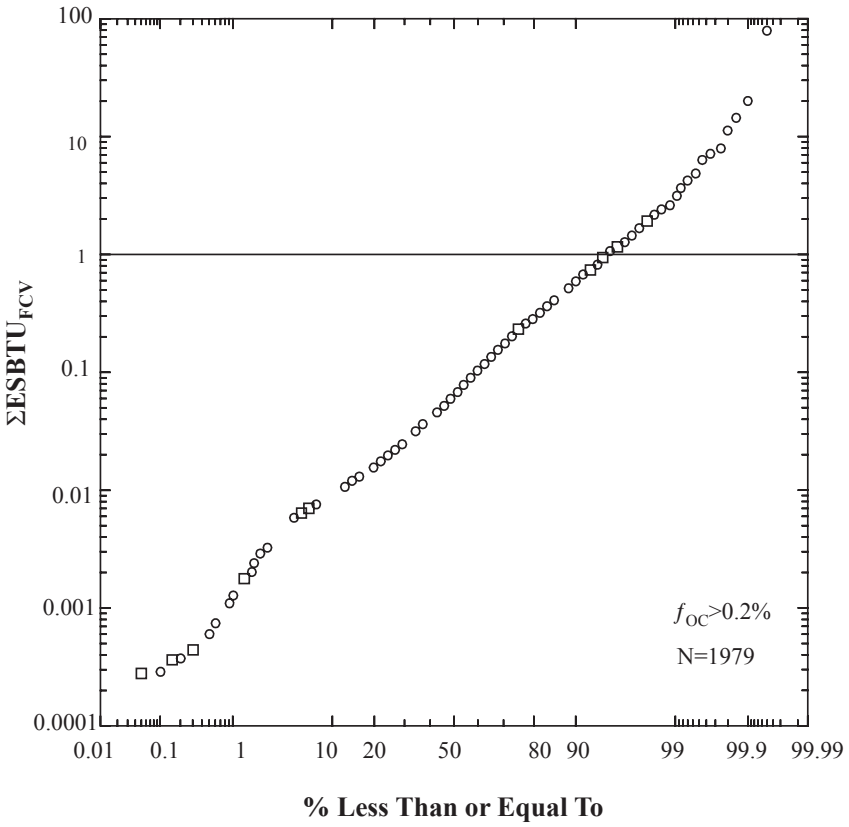


Figure 4-2. Probability distribution of the $\Sigma ESBTU_{FCV}$ for PAH mixtures in sediments from all of the coastal and estuarine locations in the United States from Figure 4-1.

Section 5

Actual and Predicted Toxicity of PAH Mixtures in Sediment Exposures

5.1 Introduction

The $C_{OC,PAHi,FCVi}$ for individual PAHs and ESBs for their mixtures were derived using water-only toxicity data (Appendix C) and both equilibrium partitioning (U.S. EPA, 2003a; Di Toro et al., 1991) and narcosis theory (Di Toro et al., 2000; Di Toro and McGrath, 2000). This section examines data from toxicity tests with spiked and field sediments contaminated with individual PAHs and their mixtures to demonstrate the strength of the technical approach used to derive ESBs and the applicability of ESBs to sediments from the field.

5.2 Spiked Sediment Toxicity Tests

5.2.1 Interstitial Water Concentrations and Sediment Toxicity: Relevance to Water-Only Toxicity Tests and WQC FCVs

The key hypothesis in the derivation of ESBs from EqP and narcosis theory is that effects concentrations from water-only aquatic toxicity tests data using benthic species are similar to effects concentrations in sediment toxicity tests based on interstitial water concentrations or sediment concentrations predicted to be toxic using EqP. This hypothesis has been tested in two ways: 1) by comparing LC50 values determined in water-only experiments to interstitial water LC50 values determined in spiked-sediment exposures, and 2) by comparing organic carbon-normalized sediment LC50 values observed in spiked-sediment exposures with those predicted from water-only LC50 values multiplied by the K_{OC} using the equilibrium partitioning model (Di Toro et al, 1991).

The interstitial water and water-only LC50 values for 28 experiments with a variety of PAHs and several freshwater and marine species are listed in Appendix F (Swartz et al., 1990; Swartz, 1991a; DeWitt et al., 1992; Suedel et al., 1993; Driscoll et al., 1997a,b, 1998). The mean ratio of the water-only LC50 to interstitial water LC50 from 20 experiments with definitive LC50 values was 1.60, indicating agreement generally within less than a factor of two. Interstitial water LC50 values almost always slightly exceeded water-only LC50 values. Three factors may contribute to that result: 1) some test species, especially epibenthic or tube-dwelling organisms, frequently encounter unspiked, overlying water and, thus, are not exclusively exposed to interstitial water; 2) interstitial water near the sediment surface may be slowly diluted by overlying water because of bioturbation and other transport processes; and 3) chemical analyses of interstitial water may include a portion of the non-bioavailable PAH fraction that is bound to dissolved organic matter. Despite these limitations, the interstitial water and water-only LC50 values are remarkably close, especially for sensitive, free-burrowing, infaunal species like *R. abronius*. These data support the evaluation of the risks of sediment-associated chemicals by comparisons between dissolved concentrations in interstitial water and water concentrations of concern from water-only toxicity tests.

A more comprehensive evaluation of the degree to which the response of benthic organisms can be predicted from contaminant concentrations in interstitial water can be made utilizing organism responses in each treatment from toxicity tests with sediments spiked with various

chemicals, including acenaphthene (Swartz, 1991a), phenanthrene (Swartz, 1991a), fluoranthene (Swartz et al., 1990; DeWitt et al., 1992), endrin (Nebeker et al., 1989; Schuytema et al., 1989), dieldrin (Hoke, 1992), DDT (Nebeker et al., 1989; Schuytema et al., 1989) or kepone (Adams et al., 1985) (Figure 5-1). Interstitial Water Toxic Units (IWTU) are calculated by dividing the concentration of a chemical in the interstitial water (µg/L) of a treatment by the water-only LC50 (µg/L). Theoretically, 50% mortality should occur at 1.0 IWTU. Mortality should be <50% at interstitial water concentrations < 1.0 IWTU, and > 50% at concentrations > 1.0 IWTU. Figure 5-1 presents the percent mortality in individual treatments for each chemical versus the IWTUs. Mortality was generally low at concentrations <1.0 IWTU, and increased sharply at ≥1.0 IWTU as would be expected if interstitial water concentrations account for the bioavailability of nonionic organic chemicals across sediments and water-only LC50 values are surrogates for interstitial water LC50 values.

5.2.2 Sediment Toxicity: Prediction Using Water-Only Toxicity and K_{OC}

The equilibrium partitioning model predicts the organic carbon-normalized sediment PAH concen-tration (PAH_{OC}) as the product of the PAH-specific partition coefficient between organic carbon and water (K_{OC}) and the water-only effect concentration for the PAH in water (example, 10-day LC50 or FCV)(Di Toro et al., 1991).

$$\text{Predicted LC50 } (\mu g/g_{OC}) = \text{water-only LC50 } (\mu g/L) \times K_{OC}\ (L/kg_{OC}) \quad (5\text{-}1)$$

Equation 5-1 was used with the water-only LC50 values in table 5-1 and the K_{OC}s in table 3-4 to predict the sediment LC50s ($\mu g/g_{OC}$) for 22 combinations of a variety of PAHs and test species (Table 5-1). Corresponding LC50 values were also determined for each combination in standard sediment toxicity tests. The mean ratio of observed/predicted LC50 values was 2.07, indicating that Equation 5-1 predicts PAH LC50 values $\mu g/g_{OC}$ in sediment with an accuracy within a factor of two (Table 5-1). This result is essentially equal to the ratio of the interstitial water and water-only LC50 values and may be the result of the same factors listed previously.

As in the case of IWTU, predicted sediment toxic units (PSTU) can be estimated by dividing the measured PAH concentration in sediments from individual treatments of spiked-sediment toxicity tests ($\mu g/g_{OC}$) by the predicted LC50 ($\mu g/g_{OC}$). This standardization allows a comprehensive analysis of the efficacy of the EqP prediction of a sediment effect concentration from the product of the K_{OC} and water-only effects data for that chemical and duration of exposure. Figure 5-2 combines PSTU-response data for diverse chemicals including acenaphthene (Swartz, 1991a), phenanthrene (Swartz, 1991a), fluoranthene (Swartz et al., 1990; DeWitt et al., 1992), endrin (Nebeker et al., 1989; Schuytema et al., 1989), dieldrin (Hoke, 1992) or kepone (Adams et al., 1985) (Figure 5-2). As with the IWTU plot, 50% mortality should occur at about 1.0 PSTU. Figure 5-2 shows that mortality was generally low at PSTU < 1, increased rapidly at PSTU ≈ 1, and was high for most samples with PSTU > 1.

These analyses support the concept that water-only LC50 values and K_{OC}s can be used to predict the sediment concentrations on an organic carbon basis that are toxic to benthic organisms. It seems probable that this EqP prediction of sediment effect concentrations from water-only effect data is applicable to other measures of aquatic toxicity, including WQC final chronic values. Therefore, an FCV for a specific PAH multiplied by its K_{OC} value should be applicable to the derivation of a value analogous to the FCV, but based on a sediment concentration. This concentration is the ESB.

5.2.3 Toxicity of Individual PAHs

Spiked-sediment toxicity tests have provided an important tool for investigating the effects of sediment-associated PAHs and the applicability of the EqP approach for the derivation of sediment benchmark concentrations. The toxicity test

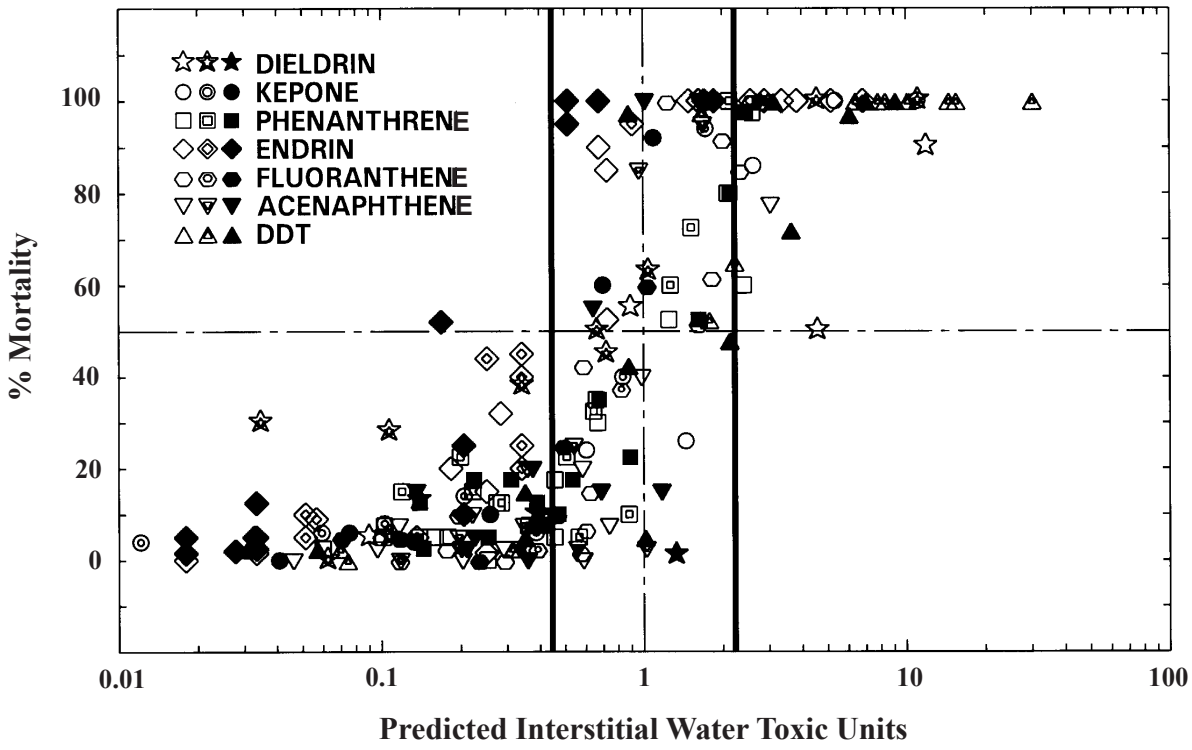


Figure 5-1. Percent mortality versus predicted interstitial water toxic units for seven chemicals and three sediments per chemical (each sediment represented by unique symbol).

method involves: 1) addition and thorough mixing of the PAH into a reference sediment that contains little or no background contamination and is not toxic, by itself, to the test species; 2) storage of the spiked sediment for up to 28d to allow the PAH to reach an equilibrium of the partitioning of the PAH between interstitial water and dissolved and particulate sedimentary materials; 3) conduct of a sediment toxicity test following standard U.S. EPA (1994) or ASTM (1993) procedures; and 4) analytical measurements, typically of the sediment/interstitial water concentration of the PAH, organic carbon, and other sediment variables. The method yields a dataset on the relation between the measured PAH concentration and the toxicity response, from which a LC50, IWTU, PSTU, and other statistical parameters can be calculated.

Sediment contaminant concentrations of nonionic organic chemicals are typically normalized to either the dry weight or organic carbon content of the sediment. To facilitate comparisons among the four PAHs from spiked sediment toxicity tests with *R. abronius,* PAH concentrations in sediments from each treatment in each spiked sediment toxicity test are normalized in this section to the PAH-specific $C_{OC,PAHi,FCVi}$ (see Table 3-4). This ratio is termed the $ESBTU_{FCVi}$, which is the ratio of the measured PAH concentration in sediments from the toxicity tests ($\mu g/g_{OC}$) to the $C_{OC,PAHi,FCVi}$ concentration ($\mu g/g_{OC}$) for that PAH, i.e., the fraction of $ESBTU_{FCV}$ represented by the observed PAH concentration in sediment. The $C_{OC,PAHi,FCVi}$ normalization does not alter the original variability in concentration-response but allows comparison of PAH effects among species, compounds, and response criteria. For example, the $C_{OC,PAHi,FCVi}$-normalized raw data for effects of individual PAHs on the amphipod, *R. abronius*, indicates similar patterns of concentration-response for acenaphthene, phenanthrene, fluoranthene, and pyrene (Figure 5-3). The individual LC50 values for the four PAHs ranged from 3.3 to 4.5 $ESBTU_{FCVi}$ (mean = 3.8)

Table 5-1. Water-only and spiked-sediment LC50 values used to test the applicability of narcosis and equilibrium partitioning theories to the derivation of ESBs for PAHs. See Appendix F for water-only and interstitial water LC50s (µg/L).

Chemical *Test Species*	Method[A]	Ratio: Interstitial Water LC50/Water-only LC50	Organic Carbon-Normalized LC50 ($\mu g/g_{OC}$) Observed	Predicted[B]	LC50 Ratio Obs/Pred	Reference
Freshwater						
Fluoranthene						
Diporeia sp.	FT,M/10	-	-	-	-	Driscoll et al., 1997a,b
Hyalella azteca	FT,M/10	> 0.58	-	-	-	Driscollet al., 1997a,b
Hyalella azteca	S,M/10	1.02[C]	500	4490	0.11[C]	Suedel et al., 1993
Hyalella azteca	S,M/10	5.27[C]	1480	4490	0.33[C]	Suedel et al., 1993
Hyalella azteca	S,M/10	2.17[C]	1250	4490	0.28[C]	Suedel et al., 1993
Chironomus tentans	S,M/10	2.86[C]	1587	3190	0.50[C]	Suedel et al., 1993
Chironomus tentans	S,M/10	7.87[C]	1740	3190	0.55[C]	Suedel et al., 1993
Chironomus tentans	S,M/10	2.37[C]	682	3190	0.21[C]	Suedel et al., 1993
Saltwater						
Acenaphthene						
Eohaustorius estuarius	FT,M/10	2.14	4330	2152	2.01	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	1.63	1920	2152	0.89	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	1.45	1630	2152	0.76	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	> 2.54	>23,500	3900	> 6.02	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	2.08	7730	3900	1.98	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	2.2	11200	3900	2.87	Swartz, 1991a
Fluoranthene						
Leptocheirus plumulosus	S/10	-	>21,200	3900	>5.44	Driscoll et al., 1998
Phenanthrene						
Eohaustorius estuarius	FT,M/10	1.05	4050	3778	1.07	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	1.06	3920	3778	1.04	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	1.11	3820	3778	1.01	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	2.09	8200	5335	1.54	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	1.65	6490	5335	1.22	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	1.95	8200	5335	1.54	Swartz, 1991a
2,6-dimethylnaphthlene						
Rhepoxynius abronius	S,M/10	-	8120	-	-	Ozretich et al., 2000a
2,3,5-trimethylnaphthlene						
Rhepoxynius abronius	S,M/10	-	3190	-	-	Ozretich et al., 2000a
1-methylfluorene						
Rhepoxynius abronius	S,M/10	-	1950	-	-	Ozretich et al., 2000a
2-methylphenanthrene						
Rhepoxynius abronius	S,M/10	-	2270	-	-	Ozretich et al., 2000a
9-methylanthracene						
Rhepoxynius abronius	S,M/10	-	6840	-	-	Ozretich et al., 2000a
Acenaphthene						
Rhepoxynius abronius	S,M/10	-	2110	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	2310	-	-	Swartz et al., 1997

Table 5-1. Continued

Test Species	Method[A]	Ratio: Interstitial Water LC50/Water-only LC50	Organic Carbon-Normalized LC50 (µg/g_{OC}) Observed	Predicted[B]	LC50 Ratio Obs/Pred	Reference
Phenanthrene						
Rhepoxynius abronius	S,M/10	-	3080	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	2220	-	-	Swartz et al., 1997
Pyrene						
Rhepoxynius abronius	S,M/10	-	1610	-	-	Ozretich et al., 2000a
Rhepoxynius abronius	S,M/10	-	1220	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	2810	-	-	Swartz et al., 1997
Fluoranthene						
Rhepoxynius abronius	S,M/10	-	2320	1390	1.66	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	3310	1390	2.38	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	1.63	1890	1390	1.36	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	2.12	2100	1390	1.51	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	1.74	2230	1390	1.6	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	> 22.66[D]	>4360	1390	4.04[D]	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	1.01	4410	1390	3.17	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	1.91	3080	1390	2.22	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	1.38	3150	1390	2.26	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	0.67	2790	1390	2.01	DeWitt et al., 1992
	Mean LC50 ratio =	1.6		Mean LC50 ratio =	2.07	

[A] Test conditions for water-only toxicity tests: S = static, FT = flow-through, M = measured, 10 = 10-d duration.
[B] Predicted LC50 ($\mu g/g_{OC}$) = water-only LC50 ($\mu g/L$) K_{OC} (L/kg_{OC}) 1 $kg_{OC}/1000g_{OC}$.
[C] Sediments spiked with fluoranthene by Suedel et al. (1993) were not at equilibrium, therefore, are not included in the mean.
[D] Source of organic carbon was fresh plant material, not naturally aged organic matter, therefore, value was not included in the mean.
[E] 10-day LC50 value from R. Swartz, Environmental Consultant (personal communication).

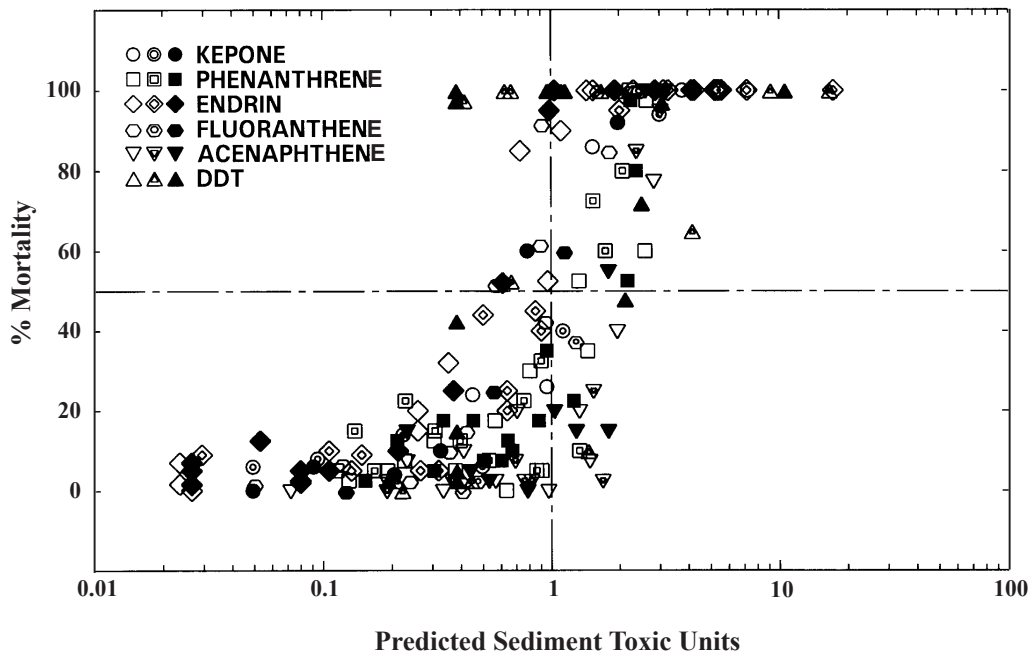


Figure 5-2. Percent mortality versus predicted sediment toxic units for six chemicals and three sediments per chemical (each sediment represented by unique symbol).

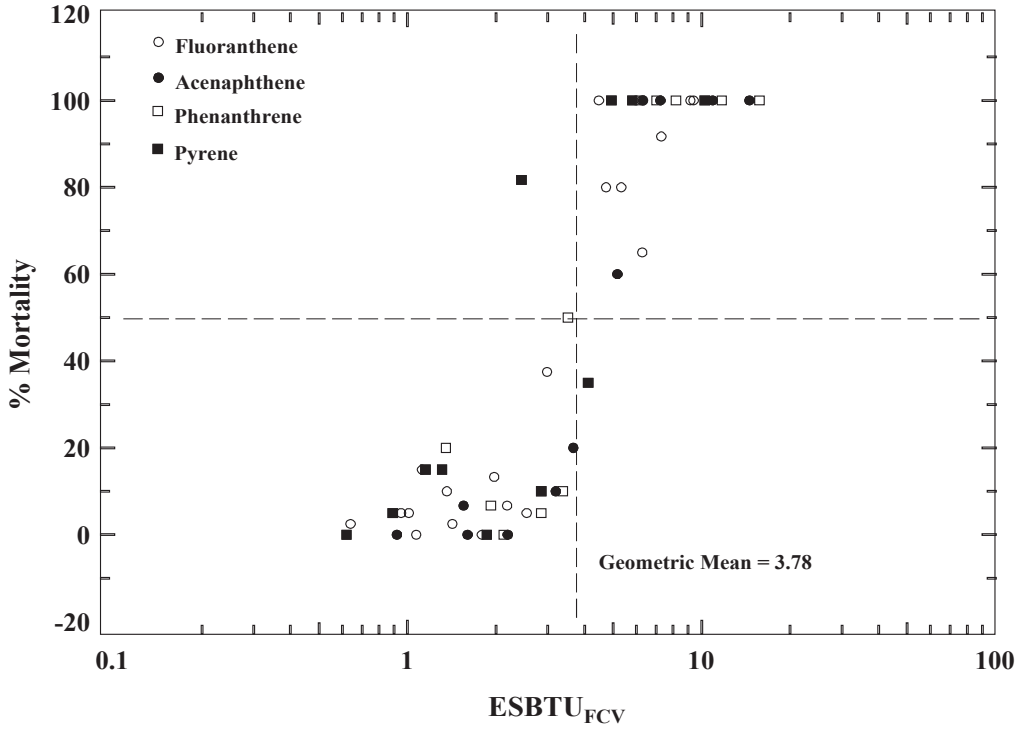


Figure 5-3. Percent mortality of *Rhepoxynius abronius* in sediments spiked with acenaphthene, phenanthrene, fluoranthene, or pyrene concentrations in sediment normalized to ESBTU$_{FCVi}$.

indicating that sediment concentrations would have to exceed the $C_{OC,PAHi,FCVi}$ by about a factor of four to cause 50% mortality in this amphipod during a 10-day exposure. The presence of mortality only at PAH concentrations in excess of the $C_{OC,PAHi,FCVi}$ would be expected.

5.2.4 Comparison of Sediment Toxicity to C_{OC,PAH_i,FCV_i}

The degree to which ESBs derived from narcosis and EqP theory and FCVs derived from water-only toxicity databases are appropriately protective of benthic organisms can be independently tested using data from spiked-sediment toxicity tests. The individual PAH concentrations in sediment (C_{OC}) affecting benthic organisms in toxicity tests were divided by the $C_{OC,PAHi,FCVi}$ to determine the $ESBTU_{FCVi}$. If most benthic organisms are sensitive at the $ESBTU_{FCVi}$ greater than 1.0 then the ESB for the PAH mixture may be appropriately protective of benthic organisms (see Section 4.2).

A review of the literature on spiked-sediment toxicity tests yielded 54 estimates of LC50, EC50 or EC25 (concentration affecting 25% of the test organisms) values for four individual PAHs (acenaphthene, phenanthrene, fluoranthene, pyrene; Appendix F). The duration of most of the tests was 10 days, but a few were longer-term tests that measured sublethal effects on reproduction or emergence (sediment avoidance). Over all the data, there was a substantial range (500 to 147,000 $\mu g/g_{OC}$) in the estimates of the median response concentrations. For example, the relative sensitivity of marine amphipods in this dataset was *Rhepoxynius abronius* > *Eohaustorius estuarius* > *Leptocheirus plumulosus*. This range in median response concentrations reflects differences in species sensitivity, PAH bioavailability and probably, most importantly, specific experimental conditions.

The data from some of the toxicity tests with individual PAHs spiked into sediments needed to be modified or not included in further analyses. Some tests with *Diporeia* sp., *Lumbriculus variegatus*, *Limnodrilus hoffmeisteri* and *Hyalella azteca* were conducted at concentra-tions in the sediment that could not have been at equilibrium with the concentration of the PAH at solubility in interstitial water (Kukkonen and Landrum, 1994; Landrum et al., 1994; Lotufo and Fleeger, 1996; Driscoll et al., 1997a,b). The reported median effect concentration is in parenthesis and maximum sediment concentration at water solubility (given in Table 3-4) for each PAH is indicated in bold in Appendix D. To facilitate comparisons of species sensitivity and to account for bioavailability, median response concentrations were divided by the $C_{OC,PAHi,FCVi}$ values to obtain the test-specific $ESBTU_{FCVi}$ values. Then PAH-specific SMAVs and GMAVS across PAHs were calculated only for 10-day lethality tests. The maximum solubility-limited sediment concentration was used to calculate the test-specific $ESBTU_{FCVi}$ and PAH-specific SMAVs and GMAVs only if there insufficient no data from tests that lacked this solubility constraint. Some tests were conducted with newly spiked sediments where time was likely insufficient to permit equilibrium to be achieved between the interstitial water and organic carbon and other sediment partitioning phases (Suedel et al., 1993). Data from these tests were not used because the median effect concentration in sediments would be lower than that expected if sediments and interstitial water were at equilibrium.

For the seven species tested acceptably against one or more PAH, the 43 test-specific $ESBTU_{FCVi}$ ranged from 1.47 to 57.8, a factor of 39.3, with no values below 1.0 $ESBTU_{FCVi}$ (Figure 5-4; Appendix D). Within each individual species, the range of test-specific $ESBTU_{FCVi}$ across multiple tests with one or more PAH, based on 10-day LC50 values, was within only a factor of 1.5 to 4.1 (mean 3.0). For the three saltwater amphipods tested against multiple PAHs the range of PAH-specific SMAVs was within a factor of 1.4 to 2.0 (mean 1.7). These observations indicate that the species tested differed in their sensitivities to PAHs, but that within a species there was a similarity of response across tests with the same or multiple PAHs. The range and frequency

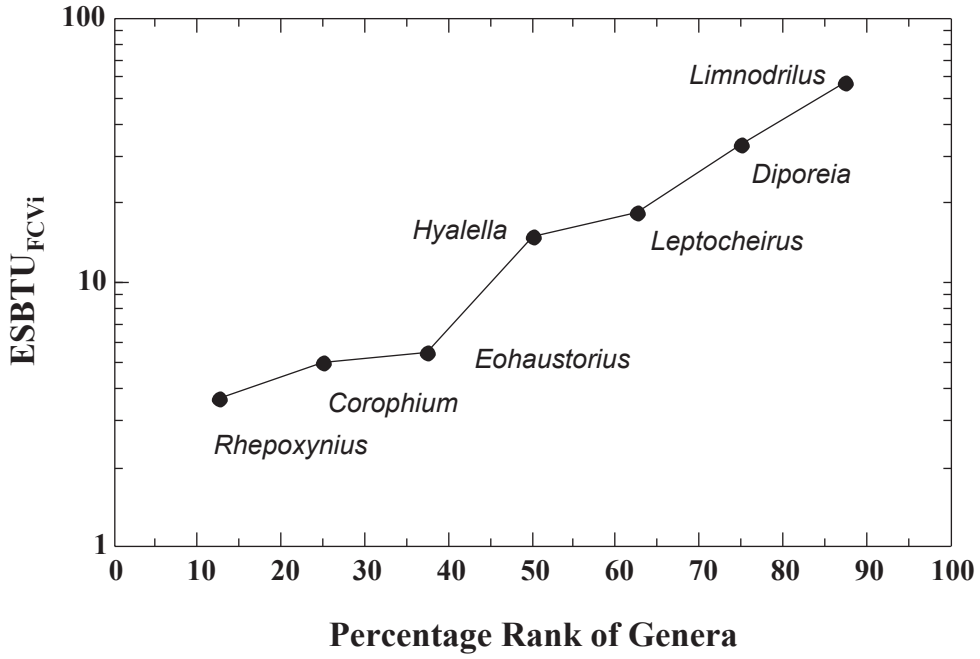


Figure 5-4. Percentage rank, based on $ESBTU_{FCVi}$, of the sensitivities of genera of benthic organisms from spiked sediment toxicity tests.

distribution of contaminant sensitivity among aquatic species is comparable to that of benthic species in water-only tests (see Section 3.4).

This analysis of data from spiked-sediment toxicity tests with individual PAHs supports the conclusion that the $C_{OC,PAHi,FCVi}$ derived from water-only toxicity tests, narcosis theory and national WQC are appropriately protective of benthic organisms. These comparisons between sediments spiked with individual PAHs and their respective $C_{OC,PAHi,FCVi}$ have value in suggesting the validity of the EqP and narcosis approaches. However, PAHs occur in nature not as individual compounds, but as mixtures.

5.2.5 PAH Mixtures

Sediments spiked with PAH mixtures have been used to resolve two issues that are relevant to the validation of the ESB for PAHs (Swartz et al., 1997; Landrum et al., 1991; Boese et al., 1999; Burgess et al., 2000b; Spehar et al., In preparation). The first concerns the toxicological additivity of the effects of the individual components of the mixture. If effects are additive, relatively simple models can be used to predict the effects of mixtures. The second issue concerns the low solubility of PAHs with high octanol-water partitioning coefficients (i.e., PAHs with $K_{OW} > 5.5$). The predicted LC50 of many high K_{OW} compounds exceeds their solubility limit. Accordingly, experimental attempts to establish the LC50 for individual high K_{OW} PAHs spiked into sediment have observed little or no acute or chronic toxicity. High K_{OW} PAH mixtures have been recently tested to see if individual high K_{OW} PAHs contribute fractional toxic units that are additive with effects of other PAHs (Spehar et al., *In preparation*).

5.2.6 Additivity of PAH Mixtures

There is a wealth of aquatic toxicological data that supports the additivity of PAHs and other narcotic chemicals in water (Konemann, 1980;

Hermens et al., 1984; Broderius and Kahl, 1985; Fig. 2-11). The additivity of sediment-associated contaminants is less well documented, although several publications indicate that PAHs in sediment are either additive or slightly less than additive (Swartz et al., 1995, 1997; Landrum et al., 1991, 1994). Landrum et al. (1991) found that the effects of a mixture of 11 sediment-associated PAHs on the freshwater amphipod, *Diporeia* sp. were "approximately additive with no overt evidence of synergism or antagonism." Landrum et al. (1991) also noted that additivity is further supported by the fact that LD50 values, expressed as PAH molar concentration in amphipod tissue, were the same for a single compound (pyrene) and the mixture of 11 compounds.

The results from some of the above 10-day studies were analyzed by dividing the concentrations of each of the PAHs in the sediments by the $C_{OC,PAHi,FCVi}$ and summing the quotients to derive the $\Sigma ESBTU_{FCV}$ for the mixture (Table 5-2). No acute toxicity was observed with *Diporeia* exposed to a $\Sigma ESBTU_{FCV}$ for all PAHs up to 3.08 (Landrum et al., 1991), but none would be expected given the 10-day LC50 value of >34.0 $\Sigma ESBTU_{FCV}$ for this species (Table 5-2). Toxicity to *R. abronius* was absent in several tests with mixtures of PAHs in treatments from 1.42 to 27.8 $\Sigma ESBTU_{FCV}$ and occurred in treatments with 5.80 and 10.3 $\Sigma ESBTU_{FCV}$ (Swartz et al., 1997; Boese et al., 1999). For *R. abronius,* the GMAV from 10-day spiked sediment tests with individual PAHs was 3.67 $\Sigma ESBTU_{FCV}$ (Table 5-2). This suggests a less than additive toxicity of the PAH mixtures tested. The amphipod *A. abdita* was exposed to a total of 2.58 and 6.05 $\Sigma ESBTU_{FCV}$ by Burgess et al. (2000b). Toxicity was absent from both treatments, and none probably should have been expected given the 4-day LC50 at 13.8 $\Sigma ESBTU_{FCV}$ (Table 5-2).

Additivity of mixtures of 13 PAHs was assumed in the development of the ΣPAH model that was used to accurately classify PAH-contaminated, field-collected sediment as toxic or not toxic (Swartz et al., 1995). Swartz et al. (1997) concluded that sediment spiked with a mixture of acenaphthene, phenanthrene, fluoranthene and pyrene caused effects on *R. abronius* that were slightly less than additive. Di Toro and McGrath (2000) reanalyzed these data and concluded that the mixture was additive (also see Section 5.2.7). Even if PAH interactions are slightly less than additive, the potential error introduced by the assumption of additivity in the derivation of an ESB for PAH mixtures would be relatively small and would be environmentally protective (i.e., the toxicity of mixtures would be slightly over-estimated).

5.2.7 PAH Additivity Demonstrated Using the Universal Narcosis Slope

The additivity of mixtures of PAHs spiked into sediments was tested using narcosis theory to calculate PAH-specific 10-day LC50 values in sediments for *R. abronius*. The experimental data from Swartz et al., (1997) was reexamined using predicted PAH-specific 10-day sediment LC50 values for *R. abronius*. The narcosis methodology was used to test additivity, rather than the actual sediment LC50 values as was presented above and by Swartz et al., (1997). This is because the predicted sediment LC50 values were derived using data from many tests with a variety of PAHs. Also, because sediment LC50 values could be predicted for the 31 or 34 PAHs analyzed from field sediments used in 10-day toxicity tests with data from toxicity tests with *R. abronius* to test narcosis and EqP predictions (See Section 5.3.1).

Interstitial water concentrations were used in place of water-only LC50 values in this process because water-only toxicity data were not available. This is justified because interstitial water and water-only LC50 values have been shown to be nearly the same (see Section 5.2.1). The 10-day interstitial water LC50 values were for eight PAHs (fluoranthene, naphthalene, pyrene, 1-methylfluorene, 2-methylphenanthrene, 9-methylanthracene, 2,6-dimethylnaphthlene, and 2,3,5-trimethylnaphthlene) tested in separate experiments. The interstitial water LC50 values for fluoranthene were from seven separate experiments (mean LC50 = 19.5 µg/L) (Swartz et al., 1990; DeWitt et al., 1992), whereas the LC50

Table 5-2. Percent mortality of benthic invertebrates in relation to the ΣESBTU$_{FCV}$ values of mixtures of polycyclic aromatic hydrocarbons spiked into sediment.

Species[A]	ΣESBTU$_{FCV}$ PAH K_{ow} <5.5	ΣESBTU$_{FCV}$ PAH K_{ow} >5.5	ΣESBTU$_{FCV}$ All PAHs	Percent Mortality	PAH Mixture[B]	Reference
Diporeia sp.	0.01	0.02	0.03	3	fluor, phen, anthr, flu, pyr, chry, b(b)flu, b(e)pyr, b(a)pyr, pery, b(ghi)pery	Landrum et al., 1991
Diporeia sp.	0.21	0.36	0.57	10	fluor, phen, anthr, flu, pyr, chry, b(b)flu, b(e)pyr, b(a)pyr, pery, b(ghi)pery	Landrum et al., 1991
Diporeia sp.	0.49	0.6	1.1	0	fluor, phen, anthr, flu, pyr, chry, b(b)flu, b(e)pyr, b(a)pyr, pery, b(ghi)pery	Landrum et al., 1991
Diporeia sp.	1.37	1.71	3.08	12	fluor, phen, anthr, flu, pyr, chry, b(b)flu, b(e)pyr, b(a)pyr, pery, b(ghi)pery	Landrum et al., 1991
R. abronius	10.32	0	10.3	100	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	5.8	0	5.8	38	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	5.12	0	5.12	8	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	3.25	0	3.25	11	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	2.5	0	2.5	4	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	1.8	0	1.8	2	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	1.42	0	1.42	3	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	2.77	0	2.77	5	anthr; flu	Boese et al., 1999
R. abronius	4.91	5.02	9.93	3	b(a)anthr; flu	Boese et al., 1999
R. abronius	5.88	0	5.88	5	2-methylanthr; flu	Boese et al., 1999
R. abronius	5.71	0	5.71	2	9,10-dimethylanth; flu	Boese et al., 1999
R. abronius	2.71	2.23	4.94	3	b(b)flu; flu	Boese et al., 1999
R. abronius	2.06	0.79	2.84	2	chr; flu	Boese et al., 1999
R. abronius	0.63	1.57	2.2	1	3,6-dimethylphen; flu	Boese et al., 1999
R. abronius	1.91	25.89	27.8	4	anthr; b(a)anthr; 2-methylanthr; b(b)flu; chr; 3,6-dimethylphen	Boese et al., 1999
R. abronius	0.58	8.03	8.61	5	anthr; b(a)anthr; 2-methylanthr; b(b)flu; chr; 3,6-dimethylphen	Boese et al., 1999
R. abronius	1.55	8.03	9.58	9	anthr; b(a)anthr; 2-methylanthr; b(b)flu; chry; 3,6-dimethylphen; flu	Boese et al., 1999
R. abronius	0.9	3.4	4.3	0	anthr; b(a)anthr; 2-methylanthr; b(b)flu; chry; 3,6-dimethylphen; flu	Boese et al., 1999
A. abdita	5.41	0.64	6.05	7	9,10-dimethylanthr; chry	Burgess et al., 2000b
A. abdita	0	2.58	2.58	7	b(a)pyr; cor	Burgess et al., 2000b
A. abdita	5.41	3.22	8.63	10	9,10-dimethylanthr; chry; b(a)pyr; cor	Burgess et al., 2000b
A. bahia	5.41	0.64	6.05	3	9,10-dimethylanthr; chry	Burgess et al., 2000b
A. bahia	0	2.58	2.58	7	b(a)pyr; cor	Burgess et al., 2000b
A. bahia	5.41	3.22	8.63	7	9,10-dimethylanthr; chry; b(a)pyr; cor	Burgess et al., 2000b

[A] Test Species: amphipods: *Diporeia* sp., *Rhepoxynius abronius*, *Ampelisca abdita*; mysids: *Americamysis bahia*
[B] PAH Code: ace - acenaphthene; anthr - anthracene; b(a)anthr - benz(a)anthracene; b(a)pry - benzo(a)pyrene; b(ghi)pery - benzo(ghi)perylene; b(b)flu - benzo(b)fluoranthene; chry - chrysene; cor - coronene; 9,10-dimethylanth - 9,10-dimethylanthracene; 3,6-dimethylphen - 3,6dimethylphenanthrene; flu - fluoranthene; fluor-fluorene; 2-methylanthr - 2-methylanthracene; pery - perylene; phen - phenanthrene; pyr - pyrene.

$$\log_{10}\text{PAH-specific LC50}_{R.\ abronius} = -0.945 \log_{10}K_{OW} + \log_{10}(15.8\ \mu\text{mol/g octanol}) \quad (5\text{-}2)$$

The PAH-specific $LC50_{R.\ abronius}$ is used to calculate the PAH-specific sediment LC50 ($\mu g/_{OC}$) for *R. abronius* (equation 5-3).

$$\text{PAH-specific sediment LC50}_{R.\ abronius} = K_{OC} \times \text{PAH-specific LC50}_{R.\ abronius} \quad (5\text{-}3)$$

values for the remaining seven PAHs are from single experiments (Ozretich et al., 1997) (Table 5-1). The individual LC50 values, and mean value for fluoranthene, were normalized to a K_{OW} of 1.0 using the universal narcosis slope (Equation 2-29). The geometric mean of these LC50 values at a K_{OW} of 1.0 is the critical body burden of 15.8 µmol/g octanol (octanol serves as a surrogate for lipid). The critical body burden is used to calculate the PAH-specific 10-day LC50 values (µg/L) for *R. abronius* (Equation 5-2). This equation is analogous to Equation 3.2 which is used to calculate the PAH-specific WQC.

The mortality of *R. abronius* in the standard 10-day sediment tests where the sediments were spiked individually (acenaphthene, fluoranthene, phenanthrene or pyrene (open symbols)), or a mixture of these four PAHs (solid circles), is compared to the predicted sediment toxic units (PSTU) to test the utility of this approach to normalize the toxicity of individual PAHs and, most importantly, to test the additivity of the PAH mixture experiment of Swartz et al. (1997) (Figure 5-5A). PSTUs are the quotients of the concentration of each PAHs measured in the individual spiked sediment treatments divided by the predicted PAH-specific 10-day sediment LC50 values for *R. abronius*. For the mixture, PSTUs were summed to obtain the total toxic unit contribution (in Section 5.3.1, sediments from the field are similarly analyzed; Figure 5-5B). The percent mortality-PSTU relationship is similar for the individual PAHs and the mixture. Apparent LC50 values are approximately within a factor of two of 1.0 PSTU. This analysis based on the universal narcosis slope and a similar analysis for narcotic chemicals in water-only experiments (Section 2.10), suggests that the assumption of near additivity of mixtures of PAHs is a reasonable approximation.

5.2.8 Additivity of Mixtures of High K_{OW} PAHs

The solubility of PAHs in water generally decreases with increasing K_{OW}, while the water column toxicity of PAH increases with increasing K_{OW}. Although the solubility of individual PAHs are a function of their structure and polarity rather than just K_{OW}, the general relationship between solubility and K_{OW} is such that solubility decreases with increasing K_{OW} slightly faster than toxicity increases. The net result of this relationship is that PAHs with high K_{OW} (roughly $\log_{10}K_{OW}$ of 5.5 and higher) have solubilities below their predicted LC50. This has led to the conventional wisdom that high K_{OW} PAHs are not toxic (at least on an acute basis) because they are insufficiently soluble to cause toxicity. For example, high K_{OW} PAHs are generally not toxic in water-only toxicity tests (Appendix C).

This argument is founded, however, on the basis of single chemicals. PAHs do not occur as single chemicals in the environment, and available experimental evidence indicates that their toxicities are additive, or slightly less than additive, when present in mixtures. This has special significance for the higher K_{OW} PAHs; although they may be too insoluble to cause toxicity individually, they could still contribute fractional toxic units to the overall toxicity of PAH mixtures.

Historically, toxicity experiments with mixtures have been conducted by testing the toxicity of individual chemicals to determine their potency, then testing mixtures of these chemicals to determine the potency of the mixture. Comparing the toxicity of the mixture to the toxic units contributed by each chemical allows evaluation of the interactive toxicity of the mixture. In the case of high K_{OW} PAHs, this experimental approach cannot be used, because the toxicity of the individual chemicals cannot be measured. Use of the narcosis model, however, allows prediction of toxicity for the mixture components and can be used to evaluate the overall toxicity of the mixture.

Spehar et al. (*In preparation*) conducted a

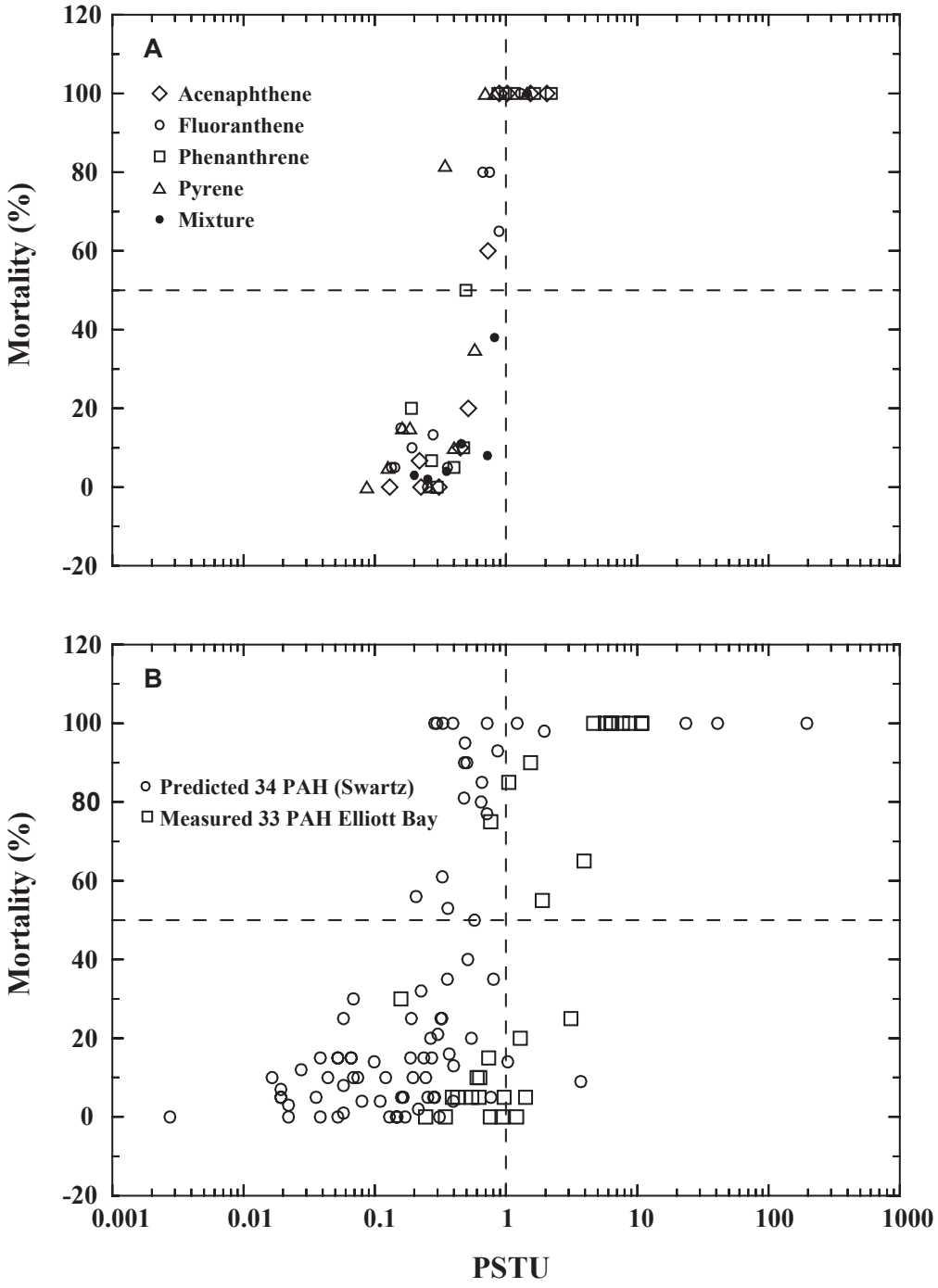


Figure 5-5. Mortality of the amphipod, *Rhepoxynius abronius*, from 10-day spiked sediment toxicity tests with four parent PAHs separately (open symbols) and in combination (closed circles) (A) and in tests with sediments from the field (B) versus predicted sediment toxic units (PSTUs). PSTUs are the quotients of the concentration of each PAH measured in sediments from the individual spiked sediment treatments, or individual sediments from the field, divided by the predicted PAH-specific 10-day sediment LC50 values for *R. abronius.* The predicted PAH-specific 10-day sediment LC50 values for *R. abronius*is were calculated using the critical body burden of 15.8 Fmol/g octanol and Equation 5-2. PSTUs were summed to obtain the total toxic unit contribution of the mixture of PAHs in spiked or field sediments.

series of sediment toxicity tests using a mixture of 13 PAHs with $\log_{10}K_{OW}$ ranging from 5.36 to 6.76 (Table 5-3). Potency of each chemical was predicted using an earlier version of the narcosis model, and the concentration for each chemical in the highest concentration of the mixture was established at an estimated 0.5 TU for *Hyalella azteca* (re-analysis using current models and the *H. azteca* GMAV from Appendix C predicts more than 0.5 TU for most PAHs). For some of these chemicals, solubility would be expected to limit their TU contribution (Table 5-3). The PAH mixture was spiked into a clean freshwater sediment at several concentrations, and into a clean marine sediment at the highest concentration only.

Several toxicity tests were conducted. A 42-day survival, growth, and reproduction study with *H. azteca* (Spehar et al., *In preparation*) was conducted in a flow-through system (2x daily renewal of overlying water) using four concentrations of the PAH mixture. In this study, chemical analysis of the bulk sediment showed that about 80% of the nominal PAH spike was measured in the sediment at the start of the exposure, and concentrations of PAH in the interstitial water were generally within a factor of 2 of the concentrations predicted from K_{OC} and solubility. After 10 days of exposure, significant effects on the dry weight of the amphipods were observed in the three highest concentrations of the PAH mixture (Figure 5-6), but there were no effects on survival. After 28 days of exposure, survival was significantly reduced in the two highest treatments, although the growth effects observed at day 10 were no longer present (Figure 5-7). As per the test protocol, organisms were removed from the sediment at day 28 and held for 14 more days in clean water to assess reproduction. No further effects on survival, growth, or reproduction were observed between days 28 and 42.

Toxicity of the PAH mixture was lower than would have been predicted based on narcosis

Table 5-3. Chemicals included in the high K_{OW} PAH mixture experiment (Spehar et al., *In preparation*).

Chemical Name	Molecular Weight (g/mol)	$\log_{10} K_{OW}$[A]	$\log_{10} K_{OC}$[B]	Estimated Solubility[C] (µg/L)	Nominal Sediment Concentration (µmol/g_{OC})	Estimated porewater concentration (µg/L) Nominal[D] (Sed. Conc./K_{OC})	Estimated porewater concentration (µg/L) Limited by Solubility
2-Ethylanthracene	206.29	5.36	5.27	59.62	39.32	43.94	43.94
3,6 Dimethylphenanthrene	206.29	5.52	5.42	77.98	42.38	33.12	33.12
2,3 Benzofluorene	216.28	5.54	5.44	25.30	42.88	33.27	25.30
Benzo(a)anthracene	228.29	5.67	5.58	12.28	45.80	27.70	12.28
Triphenylene	228.3	5.75	5.65	5.11	47.66	24.11	5.11
2-(tert-butyl)anthracene	234.34	5.88	5.78	33.04	50.91	19.78	19.78
Benzo(a)pyrene	252.31	6.11	6.00	2.88	57.46	14.38	2.88
Benzo(b)fluoranthene	252.32	6.27	6.16	8.28	62.75	10.96	8.28
Benzo(k)fluoranthene	252.32	6.29	6.18	8.35	63.64	10.50	8.35
9-Phenylanthracene	254.33	6.31	6.2	3.64	64.22	10.30	3.64
7-Methylbenzo(a)pyrene	266.35	6.54	6.43	1.46	73.37	7.32	1.46
7,12Dimethylbenz(a)anthracen	256.35	6.58	6.46	13.41	75.04	6.62	6.62
3-Methylcholanthrene	268.38	6.76	6.64	3.11	83.92	5.1	3.11
TOTAL PAH					749.4	247.1	173.9

[A] Predicted by SPARC.
[B] Predicted from Di Toro et al. (1991).
[C] Predicted by SPARC in distilled water at 25°C.
[D] Nominal concentration predicted by K_{OC}, regardless of solubility limits; highest concentration only.

theory. However, concentrations of PAH measured in the tissue of exposed *Hyalella* were considerably lower than would be in equilibrium with interstitial water, suggesting that the *Hyalella* may have avoided the test sediment, thereby reducing their exposure. Avoidance of toxic sediments by *Hyalella* has been reported previously (e.g., Whiteman et al., 1996). When 10-day growth and 28-day survival responses are compared on the basis of measured tissue burden, the thresholds for response fall in the same range as is predicted by narcosis theory (Figure 5-8). Thus, although *Hyalella* had lower uptake of these PAHs, they did show a response to the high K_{OW} PAHs suggesting that these chemicals can cause toxicity to benthic organisms. Moreover, the relationship of measured tissue concentrations to biological responses was consistent with that expected from a narcotic mode of action and additivity among PAHs in the mixture. It should be noted that because the toxicity of the individual mixture components was predicted rather than measured (which would not be possible if they are not individually toxic at solubility), we can only conclude that these results are consistent with the additivity, or approximate additivity, hypothesis, but they are not, by themselves, proof of additivity.

Because of concerns that *Hyalella* may have avoided exposure to PAH in the flow-through test by spending more time in the overlying water which was being replaced 2x daily, an additional test was conducted using the same PAH-spiked sediments, but conducting the test with renewal of overlying water-only three times during the entire 10-day test. This reduced frequency of renewal should have increased the concentrations of PAH in the overlying water (not measured), thereby increasing exposure of *Hyalella* to the PAH mixture. While the flow-through test showed effects only on growth after 10 days of exposure, results of the second test showed a concentration-dependent response of both survival and growth (Figure 5-9). When expressed on the basis of total PAH molar concentration in the sediment

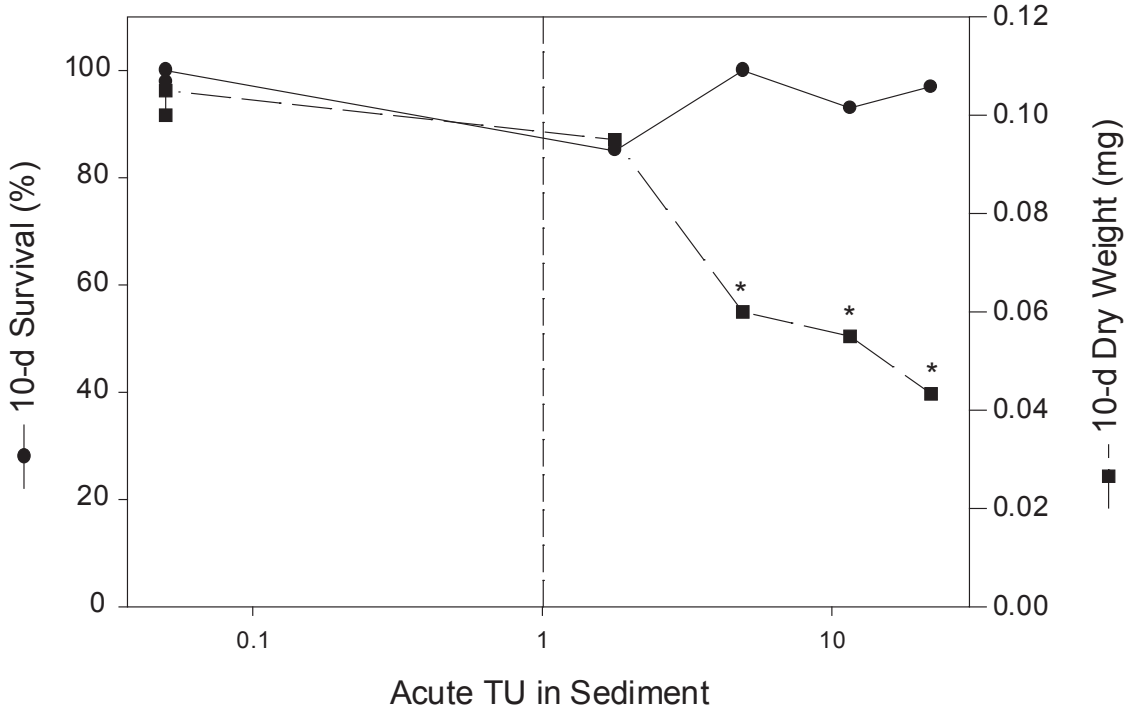


Figure 5-6. Response of *Hyalella azteca* exposed for 10 days under flow-through conditions to sediment spiked with a mixture of high K_{OW} PAH.

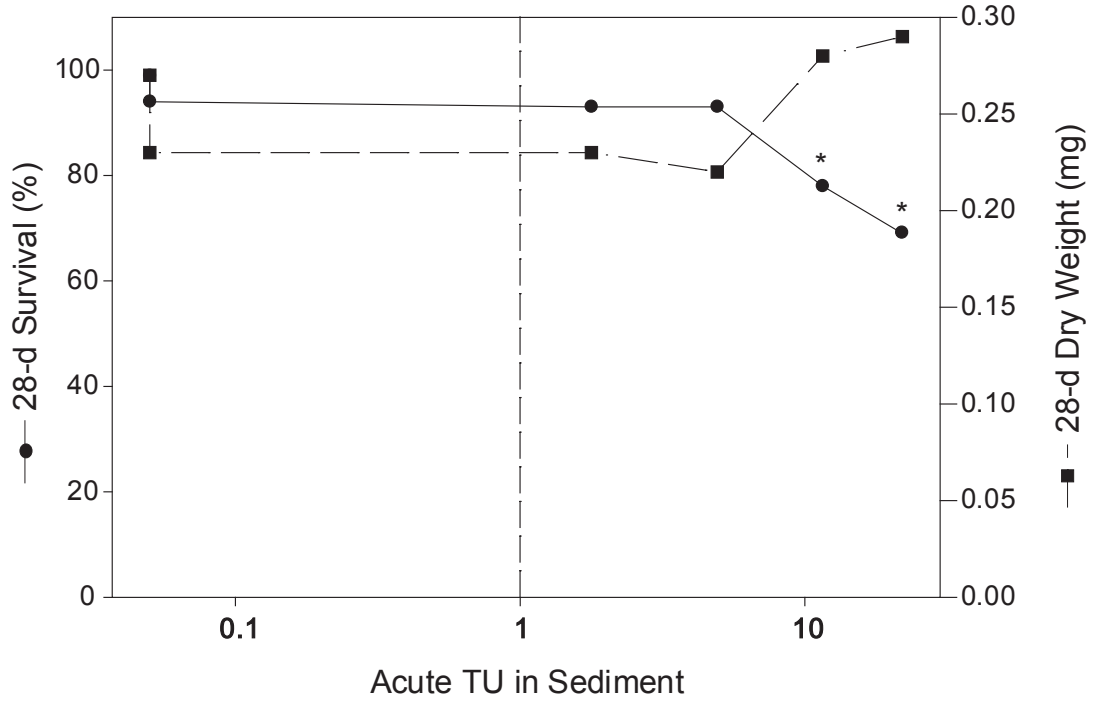


Figure 5-7. Response of *Hyalella azteca* exposed for 28 days under flow-through conditions to sediment spiked with a mixture of high K_{ow} PAH.

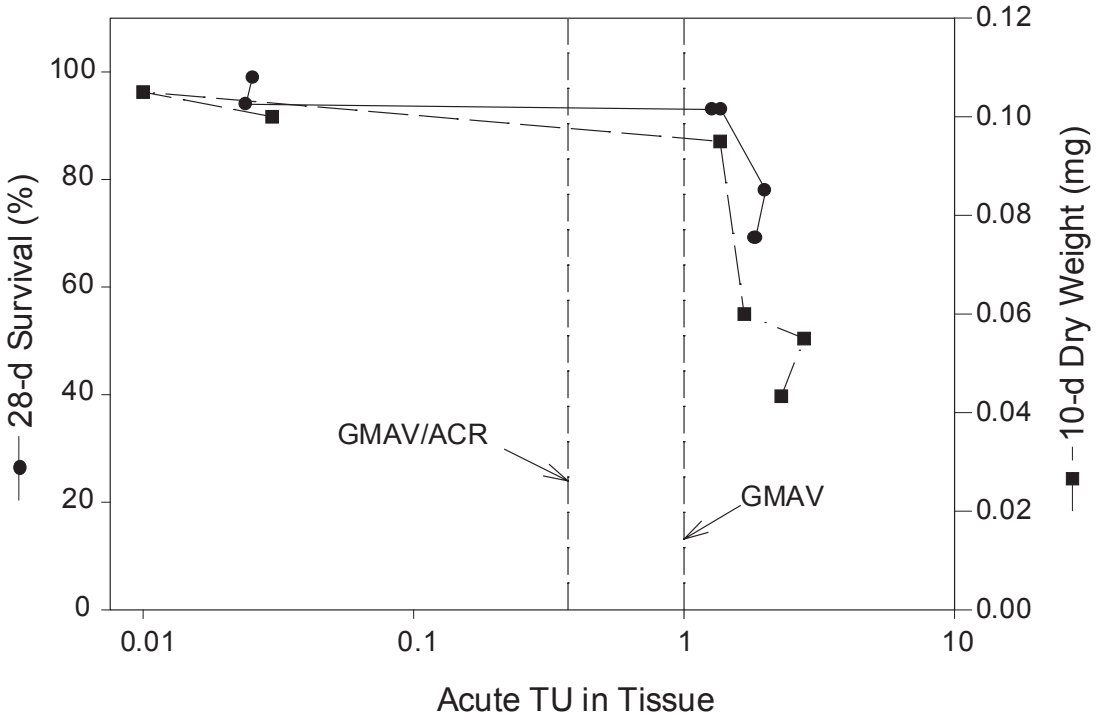


Figure 5-8. Survival (after 28 days) and growth (after 10 days) of *Hyalella azteca* expressed on the basis of measured PAH concentrations in tissues (lipid normalized).

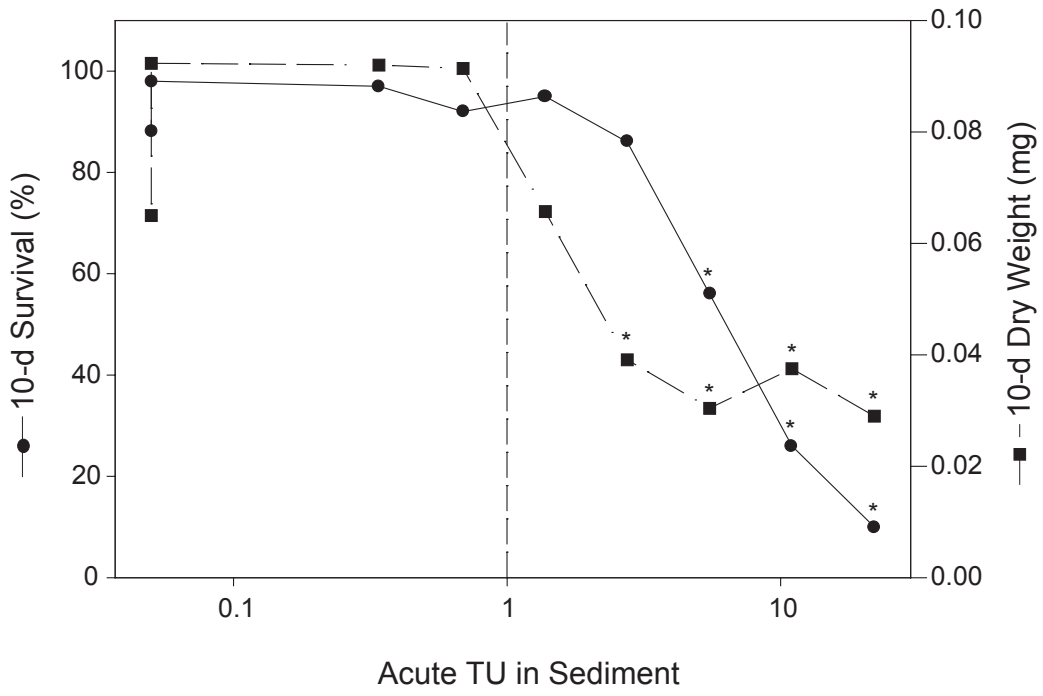


Figure 5-9. Response of *Hyalella azteca* exposed for 10 days (3 renewals) to sediment spiked with a mixture of high K_{OW} PAH.

(normalized to organic carbon), the threshold for survival and growth effects were close to the sediment concentration predicted to cause acute effects based on the narcosis model. Similarly, the tissue concentration of the mixture in the amphipods compared favorably with the critical body burden predicted to cause effects based on the narcosis model.

In addition to the freshwater experiments described above, additional experiments were conducted using marine organisms (Spehar et al., *In preparation*). Two marine organisms, a mysid (*Americamysis bahia*) and a marine amphipod (*Ampelisca abdita*), were exposed to a marine sediment spiked with the highest concentration of the PAH mixture. After 10 days of exposure to the sediment in a static system, both species showed marked mortality, with 85% mortality of mysids and 95% mortality of the amphipods. Because this sediment would be predicted to contain a large number of acute TU based on the GMAVs for these species (41 acute TU for mysids; 10 acute TU for *A. abdita*), these results cannot be used to evaluate accuracy of the narcosis model rigorously; however, they provide further support to emphasize that mixtures of high K_{OW} PAHs can cause toxicity.

In a separate test, another species of marine amphipod (*Leptocheirus plumulosus*) was exposed for 10 days to a series of concentrations of the PAH mixture spiked into the freshwater sediment used in the freshwater studies (*L. plumulosus* is tolerant of the lower salinity in the freshwater sediment, while *A. bahia* and *A. abdita* are not) (Spehar et al., In preparation). After 10 days under static conditions, *L. plumulosus* showed reduced survival in the four highest PAH concentrations (Figure 5-10). The observed toxic unit threshold for mortality was within a factor of 2 of that predicted using narcosis theory and the GMAVs in sediment from Appendix C for *L. plumulosus*.

Taken together, the results of these experiments with high K_{OW} PAHs clearly demonstrate that they can cause toxicity to benthic organisms when present in mixtures. Thresholds for toxicity in several experiments were slightly

higher than would be predicted directly from the narcosis model, though this may reflect uncertainties in the GMAV values as well as exposure-related factors (e.g., avoidance). Measured tissue concentrations in freshwater amphipods from treatments where toxicity was observed were consistent with those shown to be toxic for lower K_{OW} PAHs. Therefore, $\Sigma ESBTU_{FCV}$ for mixtures must include the partial contributions of high K_{OW} PAH in the mixture to insure that the ESB is not under protective.

5.3 Field Sediments Versus ESB_{FCV} for PAH Mixtures

The ultimate test of validity of sediment benchmarks is their predictive ability. That is, can they be used to predict effects seen in field collected samples. Unfortunately, the problem of validation using field collected samples has no straightforward solution. It is extremely difficult to separate actual cause and effect from simple correlation. The primary reason is the presence of covariation of many chemical contaminants in field collected sediments, some of which may be unmeasured. Therefore, it cannot be presumed that the response observed is due to only the chemical(s) being investigated.

However, if the PAH benchmark predicts an effect at a certain $\Sigma ESBTU_{FCV}$ for a mixture of PAHs (e.g., 50% mortality of a test organism), and the organism survives exposures significantly above the $\Sigma ESBTU_{FCV}$ value, then the benchmark may not be valid. No other comparison is more definitive. Of course, mortality at $\Sigma ESBTU_{FCV}$ values below those predicted to cause effects may be due to other causes, and provide no evidence for the validity or invalidity of the prediction.

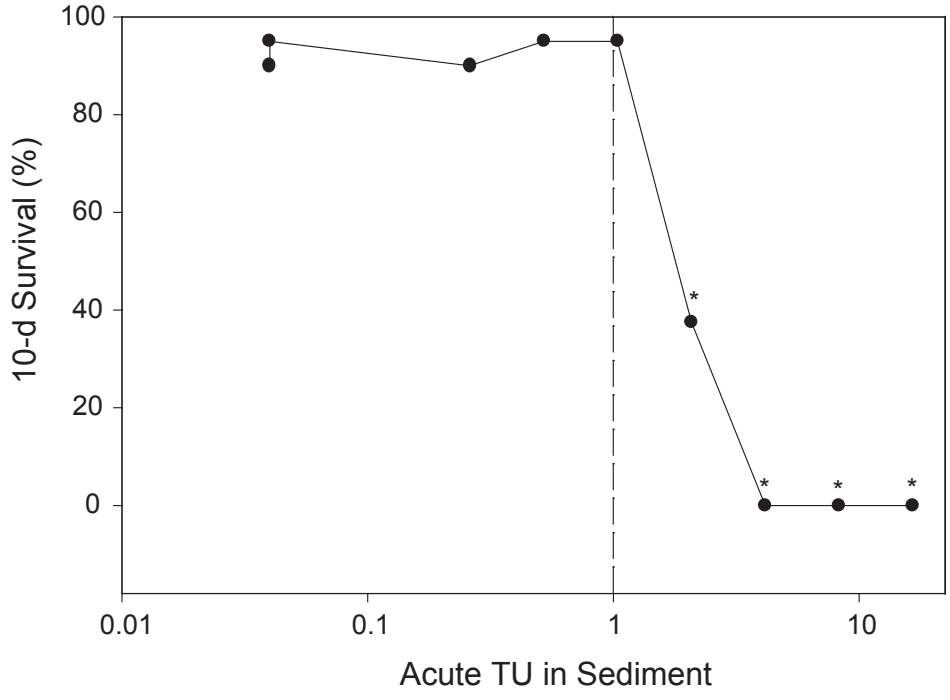


Figure 5-10. Response of *Leptocheirus plumulosus* exposed for 10 days under static conditions to sediment spiked with a mixture of high K_{OW} PAH.

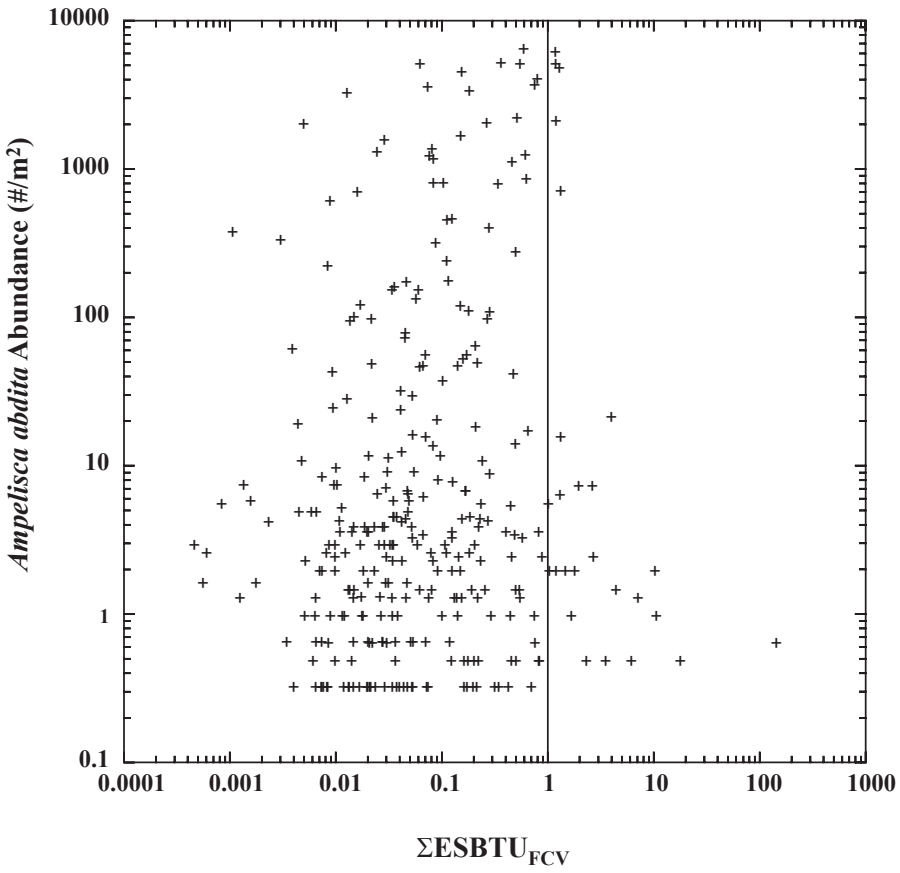


Figure 5-11. Amphipod (*Ampelisca abdita*) abundance versus $\Sigma ESBTU_{FCV}$.

5.3.1 Toxicity to R. abronius of Field Sediments Containing PAH Mixtures vs. ΣPSTUs Derived from Narcosis Theory

A set of 10-day toxicity data using *R. abronius* exposed to sediments from locations where 13 PAHs were measured and PAHs are suspected to be the primary cause of toxicity has been assembled by Swartz et al. (1995). A similar set of data from Elliott Bay where 32 PAHs (18 parent and 14 alkylated groups) were measured (Ozretich et al., 2000) is also available (See Table 3-4 for the list of PAHs). As explained in Section 5.2.7, predicted PAH-specific 10-day sediment LC50 values for *R. abronius* were derived using narcosis theory and 10-day LC50 values based on interstitial water concentrations of eight PAHs for *R. abronius*. The mortality of *R. abronius* in the standard 10-day sediment tests in each of these sediments from the field is compared to the sum of the PSTUs for that sediment (Figure 5-5B). PSTUs are the quotients of the concentration of each PAHs measured in the individual field sediments divided by the predicted PAH-specific 10-day sediment LC50 values for *R. abronius*. The sum of the PSTUs for the sediments where only 13 PAHs were analyzed were multiplied by the uncertainty factor of 2.75 (the mean ratio of the toxic contribution of the 34 PAHs analyzed by the U.S. EPA EMAP program to the 13 PAHs (see Table 6-1)). The uncertainty factor of 2.75, rather than the 95th percent uncertainty factor, was used to adjust for fewer than 34 PAHs because the goal was to use the best estimate of the sum of the toxic units to compare to the observed amphipod mortality in a specific sediment.

Consider, first, the data for which the sum of the PSTUs of the 13 PAHs (termed "predicted 34 PAH" in the figure as represented by the open circles in Figure 5-5B). There is only one

sediment where the sum of the PSTUs exceeds two where mortality was less than 50%. The important point here is that all, except one, of the sediments exceeding this concentration exhibited >50% mortality consistent with the prediction. There could be several explanations why the exception might occur in that one sediment. For example, the 13 PAHs multiplied by the mean uncertainty factor may have under-represented the true total PAH concentration.

For the remaining data, the total PAH concentrations are from field sediments where 31 of the 34 PAHs were measured (open squares). For all of these data there appears to be a concentration-response relationship with an apparent LC50 approximately at the predicted LC50 ± a factor of two, and only one sediment with less than 50% mortality had >2.0 PSTUs. This suggests that the assumption of near additivity of mixtures of PAHs is a reasonable approximation for predicting the toxicity of sediments from the field and for deriving ESBs for PAH mixtures.

5.3.2 Organism Abundance vs. ESB_{FCV} for PAH Mixtures

Another test of this sediment benchmark is the observations of the abundance of sensitive amphipods versus the total PAH concentrations in field collected sediments. Figure 5-11 presents the observed *A. abdita* abundance versus $\Sigma ESBTU_{FCV}$ when 34 PAHs were measured or estimated using the 50% uncertainty factor of 1.64 (see Table 6-1) when 23 PAHs were measured. The data are from sediments collected as part of the Virginian and Louisianian province EMAP (U.S. EPA, 1996a,b) and the New York/New Jersey Harbor REMAP (Adams et al., 1996) sediment sampling programs. The vertical line is at the ESB of 1.0 $\Sigma ESBTU_{FCV}$. The results are very encouraging. The absence of sediments having high abundances of *A. abdita* at slightly above 1.0 $\Sigma ESBTU_{FCV}$ and the decrease in amphipod abundance as the $\Sigma ESBTU_{FCV}$ increases above 1.0 is consistent with that predicted by this ESB for PAH mixtures.

It is tempting to conclude from the coincidence of $\Sigma ESBTU_{FCV}$ values >1.0 and the drop in amphipod abundance, that in fact, these data support the validity of the ESB. However, it should be pointed out again that these data can only be used to demonstrate that the ESB is not in conflict with observations. They cannot be used to validate the ESB. However, these data, and those in Figure 5-5A and B, might have cast doubt on the ESB if effects were predicted and none were observed.

The validation procedure requires sediments for which the nature of all the bioavailable chemicals are known and quantified. This is usually only satisfied with laboratory spiked sediments. This is why the experimental validity of the narcosis mixture theory as is demonstrated in Section 5.2 and illustrated in Figure 5-5A is so important.

Section 6

Implementation

6.1 Introduction

This section on implementation defines "total PAHs" for use with this ESB for PAH mixtures, presents an example ESB calculation, provides guidance on the interpretation of the ESB relative to sediment toxicity tests, describes the role of photo-activation of PAH toxicity by ultraviolet light and the relative importance of teratogenicity and carcinogenicity as a mode of toxic action for PAHs, and critically examines equilibrium of PAHs in sediments, including the presence of soot carbon, coal and similar materials as sediment binding phases other than natural organic carbon. The section ends with an approach for calculating PAH solubilities for temperatures or salinities at a specific site. This information is needed to apply this ESB and assess the risks of mixtures of sediment-associated PAHs based on the EqP methodology.

6.2 Defining Total PAH Concentration in Field Collected Sediments

"Total PAHs" required for deriving the ESB for PAH mixtures is defined in this subsection as the sum of the $ESBTU_{FCV}$ values for a minimum of the 34 PAHs (18 parents and 16 alkylated groups) measured in the U.S. EPA EMAP (U.S. EPA, 1996b, 1998) (Table 6-1). This pragmatic definition is required because databases from sediment monitoring programs that have measured a greater number of PAHs are rare, methodologies for quantification of greater than the 34 PAHs are not standard, and the use of fewer than 34 PAHs may greatly underestimate the total toxicological contribution of the PAH mixtures. We recommend that the uncertainty factors developed in this section for the 13 or 23 commonly quantified PAHs NOT be used to estimate the ESB for the 34 PAHs when important decisions are to be made based on the ESB. However, uncertainty values may be useful in specific non-ESB related decisions. The recommendation to not use the uncertainty factors for derivation of ESBs is intended to prevent the under- or over-estimation of an ESB acceptable for the protection of benthic organisms and to encourage the analysis of a minimum of the 34 PAHs using readily available analytical methodologies for new monitoring programs (NOAA, 1998)

It is expected that many sediment assessors may be in the position where available data are limited to only certain PAHs (e.g., 13 un-substituted compounds) and it is impractical to re-analyze all samples for the full suite of PAHs, but

Table 6-1. Relative distribution of $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,13}$ and $\Sigma ESBTU_{FCV,23}$ for the combined EMAP dataset (N=488).

Percentile	$\Sigma ESBTU_{FCV,TOT} / \Sigma ESBTU_{FCV,13}$	$\Sigma ESBTU_{FCV,TOT} / \Sigma ESBTU_{FCV,23}$
50	2.75	1.64
80	6.78	2.8
90	8.45	3.37
95	11.5	4.14
99	16.9	6.57

also undesirable to accept uncertainties stemming from the incomplete PAH characterization. In this instance, an intermediate approach may be to analyze a subset of sediment samples for the full suite of PAHs and use these data to develop a site-specific correction factor. This approach requires the assumption that this correction factor is consistent across the site, but it seems likely that the uncertainty with this assumption will be less than the uncertainty involved in using the generic correction factors from Table 6-1.

The following subsection presents the analysis that led to the adoption of the 34 PAHs as total PAH. Hereafter, mention of "total PAHs" in this document refers to use of a minimum of the 34 PAHs to derive the $\Sigma ESBTU_{FCV}$.

6.2.1 Introduction

PAHs are present in sediments as mixtures rather than as single compounds. It has been shown that the toxicity of sediment associated PAHs is approximately additive, and that PAHs with both low and high K_{OW} values contribute to the total toxicity (Section 5). Therefore, assessment of the toxicological contribution from the total PAH concentration present in sediments would theoretically require the measurement in every sediment of all PAHs. If the compounds formed by the alkylation of parent PAHs are included, there are more than several hundred possible structures, and quantifying all of them is impractical and costly.

As an alternative to measuring all PAHs, it may be possible to estimate the total PAH concentration in sediments using a subset of the commonly measured PAHs. This is desirable because the number of individual PAHs measured in field sediment monitoring programs varies and if too few PAHs are measured, the toxicity of sediment-associated PAHs will be underestimated. For some historical sediment monitoring data, only 13 PAHs identified by the U.S. EPA as parameters of concern were measured (Table 6-2). The National Oceanic and Atmospheric Administration (NOAA, 1991) began to quantify 10 additional PAHs in sediments, bringing the total number of PAHs measured to 23. Since then, the majority of sediment monitoring programs have measured these 23 PAHs (Table 6-2). More recently, the U.S. EPA EMAP has increased the number of PAHs measured from 23 to 34 by quantifying the C1 through C4 alkylated series for some patent PAHs where the C# indicates an alkyl group substitution (Table 6-2). The C1 represents one methyl substitution at any location on the PAH. The C2 represents either two methyl substitutions at any two locations or one ethyl substitution at any one location. The C3 represents either three methyl groups, one methyl and one ethyl group or one propyl group substitution. Similarly, the C4 represents any combination of methyl, ethyl, propyl and butyl groups so that the total number of carbons added to the parent PAH is four (Table 6-2). Although a C# PAH series by itself represents several different structures, for simplicity a C# PAH series was considered as one PAH. In total, this C# PAH alkylated series represents 16 groups of compounds as listed in Table 6-2.

In this section, the uncertainty limits are derived for estimating the total PAH toxicological contribution of the 34 PAHs from the 13 or 23 commonly measured PAHs. Data are presented using $ESBTU_{FCVi}$ to sum the contributions of the individual PAHs and determine the total PAH toxicity of the mixture as represented by the $\Sigma ESBTU_{FCV}$.

6.2.2 Data Collection

Coastal and estuarine sediment data from the Nation's water bodies were compiled from nine sources (NOAA, 1991; Adams et al., 1996; Anderson et al., 1996; Fairey et al., 1996; U.S. EPA, 1996a,b,1998; Ozretich et al., 2000; Hunt et al., 1998). With the exception of the Elliott Bay data (Ozretich et al., 2000), all of the data sources were from state and/or government funded sediment monitoring programs. In Elliott Bay, the PAHs were suspected to be causing the toxicity due to their elevated levels. Data sources that were identified had measured concentrations for at least the 23 PAHs identified by NOAA and

Table 6-2. PAH measured in various sediment monitoring programs. See Di Toro and McGrath (2000) for data sources.

Parameter	NOAA	SFEI	San Diego	Southern California	NY/NJ REMAP[A]	Virginian EMAP[B]	Elliott Bay	Carolinian EMAP	Louisianian EMAP
Acenaphthene	x	x	x	x	x	x	x	x	x
Acenaphthylene	x	x	x	x	x	x	x	x	x
Anthracene	x	x	x	x	x	x	x	x	x
Chrysene	x	x	x	x	x	x	x	x	x
Fluoranthene	x	x	x	x	x	x	x	x	x
Fluorene	x	x	x	x	x	x	x	x	x
naphthalene	x	x	x	x	x	x	x	x	x
phenanthrene	x	x	x	x	x	x	x	x	x
pyrene	x	x	x	x	x	x	x	x	x
Benzo(k)fluoranthene	x	x	x	x	x	x	x	x	x
Benzo(b)fluoranthene	x	x	x	x	x	x	x	x	x
Benzo(a)pyrene	x	x	x	x	x	x	x	x	x
Benzo(a)anthracene	x	x	x	x	x	x	x	x	x
Benzo(e)pyrene	x	x	x	x	x	x	x	x	x
Benzo(g,h,i)perylene	x	x	x	x	x	x	x	x	x
Dibenz(a,h)anthracene	x	x	x	x	x	x	x	x	x
2,6-dimethylnaphthalene	x	x	x	x	x	x	x	x	x
Indeno(1,2,3-cd)pyrene	x	x	x	x	x	x	x	x	x
1-methylnaphthalene	x	x	x	x	x	x	x	x	x
2-methylnaphthalene	x	x	x	x	x	x	x	x	x
perylene	x	x	x	x	x	x	x	x	x
1-methylphenanthrene	x	x	x	x	x	x	x	x	x
2,3,5-trimethylnaphthalene	x	x	x	x	x	x	x	x	x
2-methylanthracene							x		
2-methylphenanthrene		x					x		
3,6-dimethylphenanthrene							x		
9-methylanthracene		x					x		
9,10-dimethylanthracene							x		
C1-benzo(a)anthracenes /chrysenes							x	x	x
C2-benzo(a)anthracenes /chrysenes							x	x	x
C3-benzo(a)anthracenes /chrysenes								x	x
C4-benzo(a)anthracenes /chrysenes								x	x
C1-fluoranthenes/pyrenes							x	x	x
C2-fluoranthenes/pyrenes							x		
C1-fluorenes							x	x	x
C2-fluorenes							x	x	x
C3-fluorenes							x	x	x
C1-naphthalenes							x	x	x
C2-naphthalenes							x	x	x
C3-naphthalenes							x	x	x
C4-naphthalenes							x	x	x
C1-phenanthrenes/anthracenes							x	x	x
C2-phenanthrenes/anthracenes							x	x	x
C3-phenanthrenes/anthracenes							x	x	x
C4-phenanthrenes/anthracenes								x	x
Total Number of PAHs[B]	23	25	23	23	23	23	32	34	34
Number of data points	640	137	182	40	153	318	30	280	229

[A] Benzo(b)fluoranthene and benzo(k)flouranthene were measured together.
[B] A specific C1-PAH was not included in the total if the C1 alkylated PAH series was measured. For example, 1-methylnaphthalene was not included in the total if the C1-naphthalenes were measured.

corresponding sediment organic carbon measurements. Three sources, Elliott Bay, EMAP Louisianian Province and EMAP Carolinian Province, had measurements for some of the alkylated PAH series. The two EMAP sources analyzed for the same alkylated PAH series. The Elliott Bay dataset had some alkylated PAHs that were similar to the EMAP sources and some alkylated PAHs that were not included in the EMAP sources. A listing of the PAHs measured in each dataset is provided in Table 6-2. The first 13 PAHs in the list are the initial 13 PAHs identified by the U.S. EPA as PAHs of concern. The first 23 PAHs in the list include the additional PAHs monitored by NOAA. The total number of PAHs measured in each dataset is also provided. To prevent duplicate counting, a specific C1, C2, C3 or C4 PAH was not included in the total number of PAHs if the alkylated PAH series was measured. As an example, for Carolinian EMAP, 1-methylnaphthalene was not included in the total, because the C1-naphthalenes were measured.

To screen for insoluble PAHs, interstitial water concentrations were computed from measured solid phase concentrations using EqP theory (Di Toro et al., 1991). If the resulting interstitial water concentrations were greater than the corresponding solubilities, insoluble PAHs were assumed to be present in the sediment. For these cases, the measured solid phase concentrations were replaced by solid phase concentrations based on the aqueous solubility of each compound ($C_{OC,PAHi,Maxi}$).

The data were converted to $ESBTU_{FCVi}$ for individual PAHs by dividing the concentration of the specific PAH in the sediment (C_{OC},µg/g_{OC}) by the $C_{OC,PAHi,FCVi}$. $\Sigma ESBTU_{FCV}$ for each sediment sample were computed by summing the $ESBTU_{FCVi}$ for each PAH measured. For purposes of this section, $\Sigma ESBTU_{FCV}$ for the 34 PAHs is denoted by $\Sigma ESBTU_{FCV,TOT}$. Equation 6-1 was used to compute $\Sigma ESBTU_{FCV,TOT}$

$$ESBTU_{FCV,TOT} = \Sigma ESBTU = \sum_{i} \frac{C_{OCi}}{C_{OC,PAHi,FCVi}} \leq 1.0 \qquad (6\text{-}1)$$

6.2.3 Methodology

The objective was to determine the uncertainty of using the 13 PAHs or the 23 PAHs to predict the $\Sigma ESBTU_{FCV,TOT}$. Only the monitoring databases containing 34 PAHs were used in this analysis. The 13 PAHs were selected since the majority of the existing sediment monitoring data include these PAHs. The uncertainty values for estimating total PAHs from datasets where 13 or 23 PAHs were measured were developed from a database of ratios of $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,13}$ or $\Sigma ESBTU_{FCV,23}$. In addition, regression analyses of $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,13}$ or to $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,23}$ on a log-log linear basis were conducted to demonstrate the utility of the ratio approach across the range of $\Sigma ESBTU_{FCV}$ values.

6.2.4 Uncertainty in Predicting $\Sigma ESBTU_{FCV,TOT}$

For use in determining the uncertainty in predicting $\Sigma ESBTU_{FCV,TOT}$ from datasets consisting of the 13 or 23 PAHs, the two EMAP data sources that measured the 34 PAHs were combined and treated as a single data source. In doing this, a larger dataset that represents both alkylated and parent PAHs, and therefore, inherently has the correlative relationships of both types of PAHs, was generated (N=488). The relative distributions of the $\Sigma ESBTU_{FCV,TOT}$ to the $\Sigma ESBTU_{FCV}$ for the 13 and 23 PAHs for this dataset are provided in Table 6-1. Based on the observed ratios, the measured $\Sigma ESBTU_{FCV,13}$ for the 13 PAHs must be multiplied by 11.5 to obtain an accurate estimation of the $\Sigma ESBTU_{FCV,TOT}$ with 95 % confidence. Similarly, the measured $\Sigma ESBTU_{FCV,23}$ for the 23 PAHs must be multiplied by 4.14 to obtain an estimate of the $\Sigma ESBTU_{FCV,TOT}$ with 95% confidence. High adjustment factors needed to estimate $\Sigma ESBTU_{FCV,TOT}$, particularly from 13 PAHs, indicate the importance of having real measurements of the 34 PAHs from sediments where the PAH concentrations are of likely toxicological significance. In contrast, for

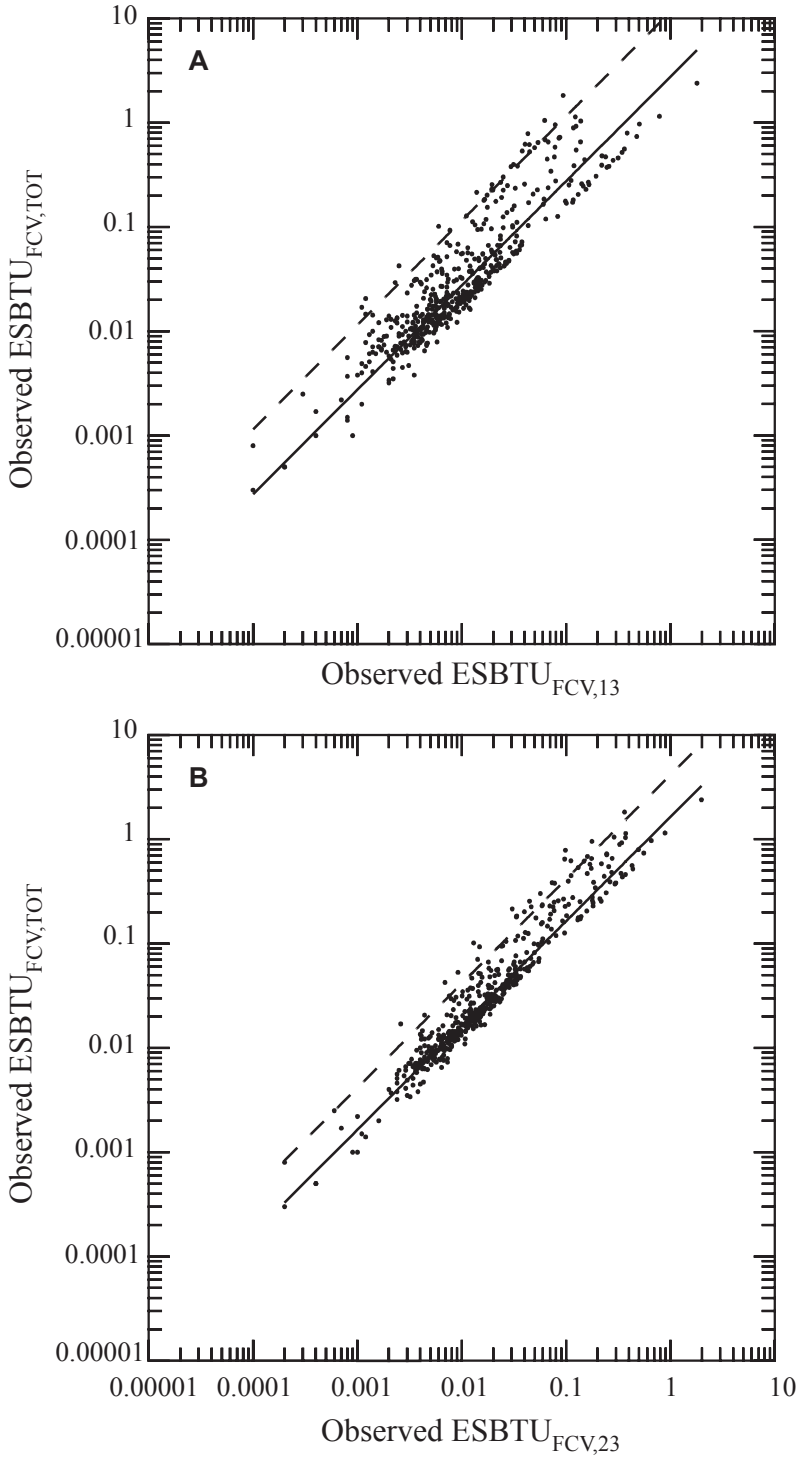


Figure 6-1. Comparison of observed ΣESBTU$_{FCV,TOT}$ to observed (A) ΣESBTU$_{FCV,13}$ from13 PAHs and (B) ΣESBTU$_{FCV,23}$ from 23 PAHs for the combined dataset including U.S. EPA EMAP Louisianian and Carolinian Provinces.

sediments where the $\Sigma ESBTU_{FCV,13}$ or $\Sigma ESBTU_{FCV,23}$ times the uncertainty factors does exceed the ESB, additional measurements including the 34 PAHs would not be warranted.

The $\Sigma ESBTU_{FCV,TOT}$ can be plotted against the $\Sigma ESBTU_{FCV,13}$ for the 13 PAHs and regression analysis conducted to show that the ratios can be used fairly well to estimate the $\Sigma ESBTU_{FCV}$ for the 34 PAHs from the sum of the 13 PAHs across a wide range of $\Sigma ESBTU_{FCV,TOT}$ because the slope is nearly 1.0 (0.9595) (Figure 6-1A). The solid line is the mean (50%) ratio of 2.75 from Table 6-1 and the dashed line is the line representing 95% of the data with a ratio of 11.5. The resulting linear regression equation from a log-log relationship is

$$\log_{10} \Sigma ESBTU_{FCV,TOT} = 0.9595 \log_{10} \Sigma ESBTU_{FCV,13} + 0.4251 \quad (R^2 = 0.8236) \qquad (6\text{-}2)$$

A similar analysis using the $\Sigma ESBTU_{FCV,TOT}$ plotted against the sum $\Sigma ESBTU_{FCV,23}$ of 23 PAHs with a regression analysis conducted to show that the slope of the regression is also nearly 1.0 (1.038) (Figure 6-1B). The solid line is the mean (50%) ratio of 1.64 from Table 6-1 and the dashed line represents 95% of the data with a ratio of 4.14. The resulting linear regression equation from a log-log relationship is

$$\log_{10} \Sigma ESBTU_{FCV,TOT} = 1.038 \log_{10} \Sigma ESBTU_{FCV,23} + 0.3576 \quad (R^2 = 0.9272) \qquad (6\text{-}3)$$

The regression approach has been used to derive uncertainty factors for estimating the $\Sigma ESBTU_{FCV,TOT}$ for the 34 PAHs using combinations of as few as three to as many as 13 or 23 PAHs (McGrath and Di Toro, 2000)

The probability distributions of the $\Sigma ESBTU_{FCV,13}$ and $\Sigma ESBTU_{FCV,23}$ values for each sediment from the databases in Table 6-2 were plotted in Figure 6-2 (A and B, respectively). The actual $\Sigma ESBTU_{FCV,13}$ values (triangles) exceeded 1.0 for 5.22% of the 1992 sediment samples (Figure 6-2A) and the $\Sigma ESBTU_{FCV,23}$ values (triangles) exceeded 1.0 for 6.55% of the 2001

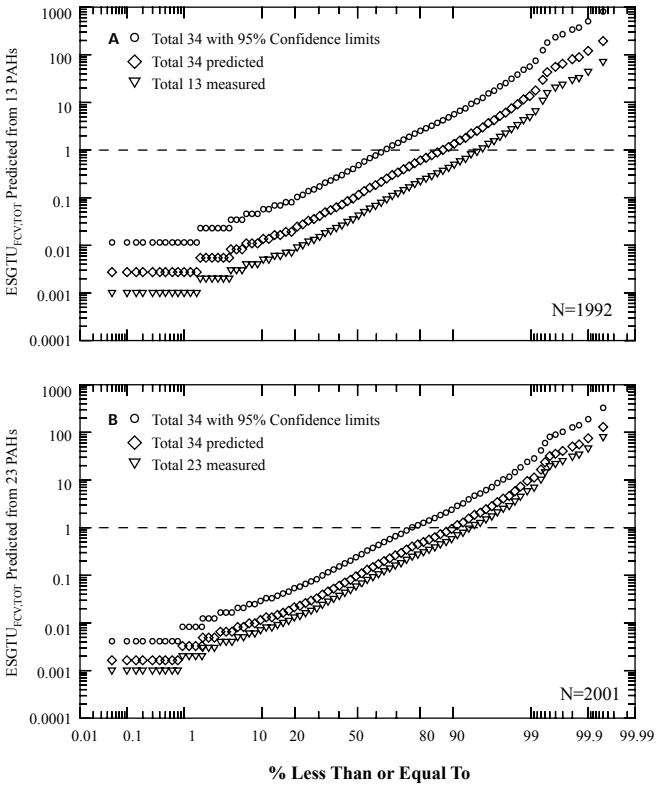


Figure 6-2. Probability distribution of the (A) $\Sigma ESBTU_{FCV,13}$ and (B) $\Sigma ESBTU_{FCV,23}$ values for each sediment from the entire database.

sediment samples (Figure 6-2B). To estimate the 50% uncertainty of $\Sigma ESBTU_{FCV,TOT}$ (plotted as diamonds in Figure 6-2), the mean ratio of the $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,13}$ (2.75; see Table 6-1) was applied to the sediment-specific $\Sigma ESBTU_{FCV,13}$ values and the mean ratio of $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,23}$ (1.64) was applied to the sediment-specific $\Sigma ESBTU_{FCV,23}$ values. The $\Sigma ESBTU_{FCV,TOT}$ estimated from the $\Sigma ESBTU_{FCV,13}$ exceeded 1.0 for 12.9% of the 1992 sediment samples and the $\Sigma ESBTU_{FCV,TOT}$ estimated from the $\Sigma ESBTU_{FCV,23}$ exceeded 1.0 for 9.85% of the 2001 sediment samples. The 95% uncertainty estimates of the $\Sigma ESBTU_{FCV,TOT}$ for each sediment (plotted as circles in Figure 6-2) was determined by multiplying the sediment-specific $\Sigma ESBTU_{FCV,13}$ values by 11.5 and by multiplying the sediment-specific $\Sigma ESBTU_{FCV,23}$ by 4.14 (Table 6-1). The 95% limits on the $\Sigma ESBTU_{FCV,TOT}$ estimated from the $\Sigma ESBTU_{FCV,13}$ exceeded 1.0 for 35.5% of the 1992 sediment samples and the 95% limits on the $\Sigma ESBTU_{FCV,TOT}$

estimated from the $\Sigma ESBTU_{FCV,23}$ exceeded 1.0 for 23.7% of the 2001 sediment samples. Therefore, if the 95% uncertainty ratios are applied to the $\Sigma ESBTU_{FCV,13}$ or the $\Sigma ESBTU_{FCV,23,}$ the predicted $\Sigma ESBTU_{FCV,TOT}$ for about one-third of the sediments are in excess of the ESB for PAH mixtures of 1.0 $\Sigma ESBTU_{FCV}$. This strongly suggests that new monitoring programs should quantify a minimum of the 34 PAHs monitored by the U.S. EPA EMAP program. In addition, field sediments containing PAHs of principally petrogenic origin will contain a greater proportion of alkalyated PAHs and PAHs not quantified in the 34 "total PAHs"(Bence et al., 1996; Means, 1998; Ho et al., 1999; Page et al., 2002). Therefore, the uncertainty factors derived above from sediments containing mostly pyrogenic PAHs, will underestimate the total PAH toxic unit contribution of the PAH mixture in sediments contaminated with mostly petrogenic PAHs. It is important to repeat that at present, the uncertainty of using the 34 PAHs to estimate the total toxicological contributions of the unmeasured PAHs is unknown and needs additional research.

For existing databases, individuals may wish to utilize uncertainty factors for sediment assessment applications other than the derivation of an ESB for PAH mixtures. An example, the use of the 50% uncertainty factors from Table 6-1 to provide the "best estimate" of the $\Sigma ESBTU_{FCV,TOT}$ for the 34 "total PAHs" in field sediments to compare with amphipod abundance (Section 5.3.1, Figure 5-11). If the number and kinds of PAHs are of similar proportions from a database from a specific site, uncertainty factors for adjusting the concentrations of the PAHs at that site may be derived using the approach detailed above. Research to determine the toxicological contributions of PAHs in sediments that are not included in the 34 PAHs is encouraged so that the uncertainty of this definition of "total PAHs" can be estimated.

6.3 Example Calculation of ESB_{FCV} for PAHs and EqP-based Interpretation

To assist the users of this ESB for mixtures of PAHs, example calculations for deriving the ESB are provided in Table 6-3. For each of three sediments, the calculations began with measured concentrations (in bold) of individual PAHs (µg/g dry wt.) and TOC (%) in each sediment. All other values were calculated. The specific concentrations in each sediment were selected to provide examples of how the chemical measurements are used with the ESB to determine the acceptability of the mixture of PAHs in a specific sediment and how the risks of sediment-associated PAHs can be evaluated within the technical framework of the EqP and narcosis approaches. The 34 PAHs constituting what is defined as "total PAH" in Section 6 are listed. Also listed are the critical concentrations in sediment of each of the 34 individual PAHs ($C_{OC,PAHi,FCVi}$) from Table 3-4.

Sediment A is provided as an example to demonstrate how to calculate the ΣESBTU when less than the required 34 PAHs have been chemically analyzed. It is important to remember that because of the uncertainty in such calculations the resultant ΣESBTU must not be considered as an ESB nor used in important sediment management decisions. Uncertainty factors applied to the ΣESBTU have value, for example, in determining if additional chemical analyses are required and prioritizing which sediments require the additional analyses.

Sediment A is from a historical monitoring database, it contains concentrations of 13 PAHs measured as µg PAH/g dry sediment and has 0.81% TOC. First, the dry weight concentrations for each PAH were converted to µg PAH/g organic carbon (C_{OC}, $\mu g/g_{OC}$) by dividing by the fraction organic carbon (f_{OC} = 0.0081), where f_{OC} = %TOC/100. Second, the organic carbon-normalized PAH concentrations in the sediment were divided by the PAH-specific sediment concentration of concern ($C_{OC,PAHi,FCVi}$) to derive the toxic unit-like $ESBTU_{FCVi}$ for each individual PAH. In this sediment, none of the measured C_{OCi} exceed the corresponding $C_{OC,PAHi,Maxi}$ so solubility constraints do not affect the calculation of $ESBTU_{FCVi}$ for this sediment. The $ESBTU_{FCVi}$ for the 13 PAHs were added to derive the ΣESBTU for the 13 PAHs ($\Sigma ESBTU_{FCV,13}$) which is 0.348

Table 6-3A. Example calculations of ESBs for PAH mixtures: three sediments.

			Sediment A (TOC=0.81%; f_{OC}=0.0081)		
PAH[A]	$C_{OC, PAHi, FCVi}$ (µg/g_{OC})	$C_{OC, PAHi, Maxi}$ (µg/g_{OC})	Conc. (µg/g dry wt.)	C_{OC} (µg/g_{OC})	$ESBTU_{FCVi}$
naphthalene	385	61700	**0.0894**	11	0.0287
C1 naphthalenes	444	-			
acenaphthylene	452	24000	**0.0348**	4.29	0.0095
acenaphthene	491	33400	**0**	0	0
C2 naphthalenes	510	-			
fluorene	538	26000	**0.0722**	8.91	0.0166
C3 naphthalenes	581	-			
anthracene	594	1300	**0.628**	77.6	0.1306
phenanthrene	596	34300	**0.139**	17.1	0.0287
C1 flourenes	611	-			
C4 naphthalenes	657	-			
C1 phenanthrenes	670	-			
C2 flourenes	686	-			
pyrene	697	9090	**0.171**	21.1	0.0303
flouranthene	707	23870	**0.0806**	9.96	0.0141
C2 phenanthrenes	746	-			
C3 flourenes	769	-			
C1 fluoranthenes	770	-			
C3 phenanthrenes	829	-			
benz(a)anthracene	841	4153	**0.0709**	8.75	0.0104
chrysene	844	826	**0.157**	19.4	0.023
C4 phenanthrenes	913	-			
C1 chrysenes	929	-			
benzo(a)pyrene	965	3840	**0.164**	20.3	0.021
perylene	967	431			
benzo(e)pyrene	967	4300			
benzo(b)fluoranthene	979	2169	**0.139**	17.2	0.0175
benzo(k)fluoranthene	981	1220	**0.139**	17.2	0.0175
C2 chrysenes	1008	-			
benzo(g,h,i)perylene	1095	648			
C3 chrysenes	1112	-			
indeno(1,2,3-cd)pyrene	1115	-			
dibenzo(a,h)anthracene	1123	2389			
C4 chrysenes	1214	-			
Sum total of $ESBTU_{FCVi}$					$\Sigma ESBTU_{FCV,13} = 0.3479$

[A] PAHs and corresponding $C_{OC,PAHi,FCVi}$ and $C_{OC,PAHi,Maxi}$ values are from Table 3-4 (bold).

Table 6-3B. Continued.

			Sediment B (TOC=0.886%; f_{OC}=0.00886)		
PAH[A]	$C_{OC, PAHi, FCVi}$ (µg/g$_{OC}$)	$C_{OC, PAHi, Maxi}$ (µg/g$_{OC}$)	Conc. (µg/g dry wt.)	C_{OC} (µg/g$_{OC}$)	$ESBTU_{FCVi}$
naphthalene	385	61700	**0.2703**	30.51	0.07925
C1 naphthalenes	444	-	**1.2084**	136.39	0.30719
acenaphthylene	452	24000	**0.0165**	1.86	0.00412
acenaphthene	491	33400	**0.0401**	4.53	0.00922
C2 naphthalenes	510	-	**3.2691**	368.98	0.72348
fluorene	538	26000	**0.3702**	41.78	0.07766
C3 naphthalenes	581	-	**5.1079**	576.51	0.99227
anthracene	594	1300	**0.0507**	5.72	0.00962
phenanthrene	596	34300	**0.5679**	64.09	0.1075
C1 flourenes	611	-	**0.9362**	105.67	0.17294
C4 naphthalenes	657	-	**3.3088**	373.46	0.56843
C1 phenanthrenes	670	-	**0.9267**	104.6	0.15611
C2 flourenes	686	-	**1.2384**	139.77	0.20375
pyrene	697	9090	**0.408**	46.05	0.06606
flouranthene	707	23870	**0.3244**	36.62	0.0518
C2 phenanthrenes	746	-	**1.0645**	120.15	0.16106
C3 flourenes	769	-	**1.2664**	142.94	0.18587
C1 fluoranthenes	770	-	**0.3824**	43.16	0.05605
C3 phenanthrenes	829	-	**0.81**	91.43	0.11028
benz(a)anthracene	841	4153	**0.2011**	22.69	0.02698
chrysene	844	826	**0.2574**	29.05	0.03442
C4 phenanthrenes	913	-	**0.5644**	63.71	0.06978
C1 chrysenes	929	-	**0.2987**	33.72	0.03629
benzo(a)pyrene	965	3840	**0.1817**	20.51	0.02125
perylene	967	431	**0.3511**	39.63	0.04098
benzo(e)pyrene	967	4300	**0.1673**	18.89	0.01953
benzo(b)fluoranthene	979	2169	**0.1708**	19.28	0.01969
benzo(k)fluoranthene	981	1220	**0.1962**	22.15	0.02258
C2 chrysenes	1008	-	**0.2242**	25.3	0.0251
benzo(g,h,i)perylene	1095	648	**0.1504**	16.97	0.0155
C3 chrysenes	1112	-	**0.0279**	3.15	0.00283
indeno(1,2,3-cd)pyrene	1115	-	**0.1473**	16.63	0.01491
dibenzo(a,h)anthracene	1123	2389	**0.0423**	4.77	0.00425
C4 chrysenes	1214	-	**0.1196**	13.5	0.01112
Sum total of $ESBTU_{FCVi}$					$\Sigma ESBTU_{FCV,TOT}$ = 4.408

[A] PAHs and corresponding $C_{OC,PAHi,FCVi}$ and $C_{OC,PAHi,Maxi}$ values are from Table 3-4 (bold).

Table 6-3C. Continued.

			Sediment C (TOC=6.384%; f_{OC}=0.06384)		
PAH[A]	$C_{OC, PAHi, FCVi}$ (µg/g_{OC})	$C_{OC, PAHi, Maxi}$ (µg/g_{OC})	Conc. (µg/g dry wt.)	C_{OC} (µg/g_{OC})	$ESBTU_{FCVi}$
naphthalene	385	61700	**2.193**	34.4	0.0892
C1 naphthalenes	444	-	**1.37**	21.9	0.0493
acenaphthylene	452	24000	**2.04**	32	0.0707
acenaphthene	491	33400	**0.806**	12.6	0.0257
C2 naphthalenes	510	-	**1.448**	22.7	0.0445
fluorene	538	26000	**1.387**	21.7	0.0404
C3 naphthalenes	581	-	**1.979**	31	0.0533
anthracene	594	1300	**3.695**	57.9	0.0974
phenanthrene	596	34300	**4.208**	65.9	0.1106
C1 flourenes	611	-	**1.03**	16.1	0.0264
C4 naphthalenes	657	-	**2.009**	31.5	0.0479
C1 phenanthrenes	670	-	**4.559**	71.4	0.1066
C2 flourenes	686	-	**1.928**	30.2	0.0440
pyrene	697	9090	**20.14**	315.5	0.4526
flouranthene	707	23870	**2.519**	39.5	0.0558
C2 phenanthrenes	746	-	**4.789**	75	0.1006
C3 flourenes	769	-	**3.419**	53.6	0.0696
C1 fluoranthenes	770	-	**11.73**	183.7	0.2386
C3 phenanthrenes	829	-	**5.378**	84.2	0.1016
benz(a)anthracene	841	4153	**8.293**	129.9	0.1545
chrysene	844	826	**9.197**	144.1	0.1707
C4 phenanthrenes	913	-	**4.674**	73.2	0.0802
C1 chrysenes	929	-	**5.24**	82.1	0.0884
benzo(a)pyrene	965	3840	**10.97**	171.8	0.1781
perylene	967	431	**28.23**	442.2	0.4457[B]
benzo(e)pyrene	967	4300	**8.92**	139.7	0.1445
benzo(b)fluoranthene	979	2169	**18.14**	284.1	0.2902
benzo(k)fluoranthene	981	1220	**5.5**	86.2	0.0878
C2 chrysenes	1008	-	**4.753**	74.5	0.0739
benzo(g,h,i)perylene	1095	648	**5.583**	87.5	0.0799
C3 chrysenes	1112	-	**0.398**	6.2	0.0056
indeno(1,2,3-cd)pyrene	1115	-	**10.8**	169.2	0.1517
dibenzo(a,h)anthracene	1123	2389	**2.499**	39.1	0.0349
C4 chrysenes	1214	-	**1.581**	24.8	0.0204
Sum total of $ESBTU_{FCVi}$					$\Sigma ESBTU_{FCV,TOT}$ = 3.831

[A] PAHs and corresponding $C_{OC,PAHi,FCVi}$ and $C_{OC,PAHi,Maxi}$ values are from Table 3-4 (bold).

[B] Because C_{OC} exceeds $C_{OC,PAHi,Maxi}$, $C_{OC,PAHi,Maxi}$ is substituted for C_{OC} to calculate $ESBTU_{FCVi}$ (see text).

(Table 6-3). Importantly, only 13 of the 34 individual PAHs defined as total PAH were measured. Because the toxicological contributions of all 34 PAHs must be considered if the ESB is to be protective of benthic organisms, some assumption must be made regarding the contribution of the unmeasured PAHs. For a confidence level of 95%, the uncertainty factor from Table 6-1 is 11.5, which is then multiplied by the calculated $\Sigma ESBTU_{FCV,13}$ of 0.348 for an estimated value of $\Sigma ESBTU_{FCV,34}$ of 4.00. Since this value is greater than one, it suggests the potential for adverse effects from PAHs. However, one must realize that this finding is, in part, a function of the correction factor selected to relate the data for 13 PAHs to an estimated ΣESBTU for 34 PAHs. If the value for 50% confidence was selected from Table 6-1 (2.75), the estimated $\Sigma ESBTU_{FCV,34}$ drops to 0.957, which is much lower than the value predicted for the 95% confidence interval. This difference illustrates the importance of measuring all 34 PAH compounds in order to eliminate unnecessary uncertainty in applying the PAH ESB.

Sediment B is a PAH-contaminated sediment from one of the U.S. EPA EMAP monitoring programs where all 34 of the PAHs in the sediment and TOC (0.886%) were measured. The concentrations of each PAH on a μg PAH/g organic carbon (C_{OC}, $\mu g/g_{OC}$) basis were derived by dividing the dry weight concentrations by the fraction organic carbon (f_{OC} = 0.00886), where f_{OC} = %TOC/100. The organic carbon-normalized PAH concentrations in sediment were divided by the PAH-specific sediment concentration of concern ($C_{OC,PAHi,FCVi}$) to derive the $ESBTU_{FCVi}$ for each individual PAH. As was the case for Sediment A, none of the measured C_{OC} exceeded $C_{OC,PAHi,Maxi.}$, so solubility constraints did not factor into the calculation of $ESBTU_{FCVi}$. The $ESBTU_{FCVi}$ values for the 34 PAHs were summed to determine the $\Sigma ESBTU_{FCV}$ which was 4.41, which exceeds the ESB ($\Sigma ESBTU_{FCV}$ >1.0) for PAH mixtures. Further examination of this sediment suggested that it is contaminated with primarily petrogenic PAHs; i.e., the ratio of $\Sigma ESBTU_{FCV,13}$ (which contains no alkylated PAHs) to $\Sigma ESBTU_{FCV}$ for the 34 PAHs is low (approximately 0.1). Chemical analysis of the PAHs in interstitial water indicated that this sediment may be unacceptably contaminated by the mixture of PAHs because it contained 5.6 interstitial water toxic units ($IWTU_{FCV}$). Ten day toxicity tests, which were part of the monitoring project, showed 64% mortality of *R. abronius* which is consistent with the $IWTU_{FCV}$ and the 10-day spiked sediment LC50 for *R. abronius* at 3.68 $\Sigma ESBTU_{FCV}$ values (Appendix D). This suggests the EqP- and narcosis-based ESB is appropriate to the sediment. The sediment is unacceptable for the protection of benthic organisms due to the PAH mixture present and additional studies to quantify the spatial extent of contamination are desirable.

Sediment C is also a PAH-contaminated sediment from an U.S. EPA EMAP monitoring program where the 34 PAHs and TOC of 6.38% were measured. The concentrations of each PAH on a µg PAH/g organic carbon (C_{OC}, $\mu g/g_{OC}$) basis were derived by dividing the dry weight concentrations by the fraction organic carbon (f_{OC} = 0.0638), where f_{OC} = %TOC/100. Except for perylene, the organic carbon-normalized PAH concentrations in sediment were divided by the PAH-specific sediment concentration of concern ($C_{OC,PAHi,FCVi}$) to derive the $ESBTU_{FCVi}$ for each individual PAH. The concentration of perylene 442.2 $\mu g/g_{OC}$ exceeded the solubility-constrained solid phase concentration ($C_{OC,PAHi,Maxi}$). Thus, the $ESBTU_{FCVi}$ for perylene was calculated as the quotient of the solubility-constrained solid phase concentration over the perylene-specific solid phase concentration equivalent to the FCV ($ESBTU_{FCV,Perylene} = C_{OC,Perylene,Maxi} / C_{OC,Perylene,FCV}$). The $ESBTU_{FCVi}$ values for the 34 PAHs were summed to determine the $\Sigma ESBTU_{FCV}$ which was 3.83, a similar value as in sediment B. The PAH mixture in sediment C exceeds the ESB ($\Sigma ESBTU_{FCV}$ >1.0) for PAH mixtures. In contrast to sediment B, sediment C was not toxic to *R. abronius* in 10-day sediment toxicity tests. This sediment is contaminated with primarily pyrogenic PAHs; i.e., the ratio of $\Sigma ESBTU_{FCV,13}$ (which contains no alkylated PAHs) to $\Sigma ESBTU_{FCV}$ for the 34 PAHs is high (approximately 0.5).

Because this PAH mixture appears to be combustion related, it suggests the potential for the presence of soot carbon, coal, or other carbon forms that show unusual partitioning behavior relative to normal diagenetic carbon (see Section 6.8). Indeed, chemical analysis of interstitial water from this sediment showed <0.12 $IWTU_{FCV}$ of PAHs. If normal partitioning behavior was occuring, one would expect the $IWTU_{FCV}$ to be very close to the calculated $\Sigma ESBTU_{FCV}$ (in this case, 3.89) is indicative of this unusual partitioning behavior. Physical examination of the sediment showed the presents of soot-like particles. The presence of soot and associated differences in chemical partitioning make the directly calculated $\Sigma ESBTU_{FCV}$ overly protective for this sediment. However, one could apply the general PAH ESB approach to the interstitial water using $IWTU_{FCV}$, or develop site-specific partition coefficients and recalculate $\Sigma ESBTU_{FCV}$ using site-specific $C_{OC,PAHi,FCVi}$ values calculated from the site-specific partition coefficients, as described in U.S. EPA (2003b).

6.4 Interpreting ESBs in Combination with Toxicity Tests

Sediment toxicity tests provide an important complement to ESBs in interpreting overall risk from contaminated sediments. Toxicity tests have different strengths and weaknesses compared to chemical-specific benchmarks, and the most powerful inferences can be drawn when both are used together.

Unlike chemical-specific benchmarks, toxicity tests are capable of detecting any toxic chemical, if it is present in toxic amounts; one does not need to know what the chemicals of concern are to monitor the sediment. Toxicity tests are also useful for detecting the combined effect of chemical mixtures, if those effects are not considered in the formulation of the applicable chemical-specific benchmark. However, if the sediment requirements of the test species are not met, observed mortality may not be due to chemical contaminants in the sediment.

On the other hand, toxicity tests have weaknesses also; they provide information only for the species tested, and also only for the endpoints measured. This is particularly critical given that most sediment toxicity tests conducted at the time of this writing measure primarily short-term lethality; chronic test procedures have been developed and published for some species, but these procedures are more resource-intensive and have not yet seen widespread use. In contrast, chemical-specific benchmarks are intended to protect most species against both acute and chronic effects.

Many assessments may involve comparison of sediment chemistry (e.g., using ESB values) and toxicity test results. In cases where results using these two methods agree (either both positive or both negative), the interpretation is clear. In cases where the two disagree, the interpretation is more complex; some investigators may go so far as to conclude that one or the other is "wrong," which is not necessarily the case.

Individual ESBs consider only the effects of the chemical or group of chemicals for which they are derived. For this reason, if a sediment shows toxicity but does not exceed the ESB for a chemical of interest, it is likely that the cause of toxicity is a different chemical or group of chemicals.

In other instances, it may be that an ESB is exceeded but the sediment is not toxic. As explained above, these findings are not mutually exclusive, because the inherent sensitivity of the two measures is different. ESBs are intended to protect relatively sensitive species against both acute and chronic effects, whereas toxicity tests are performed with specific species that may or may not be sensitive to chemicals of concern, and often do not encompass the most sensitive endpoints (e.g., chronic survival, growth or reproduction). It is also possible for a sediment above the ESB to be non-toxic if there are site-specific partitioning conditions that run counter to the equilibrium partitioning model and its assumptions (see Section 7.2).

The first step in interpreting this situation is to consider the magnitude of the ESB exceedance

and the sensitivity of the test organism and endpoint to the suspect chemical. For example, the acute-chronic ratio used for the PAH mixtures ESB is 4.16 (Section 3.3.7); as such, if $\Sigma ESBTU_{FCV} = 4$, one would anticipate lethal effects only for highly sensitive species. Between $\Sigma ESBTU_{FCV}$ of 1 and 4, one would expect only chronic effects, unless the test species was unusually sensitive. If $\Sigma ESBTU_{FCV}$ for PAHs was 2, for example, one would not generally expect to see lethality from PAHs in short term sediment lethality tests.

A more precise method for evaluating the results of toxicity tests is to calculate effect concentrations in sediment that are species specific. For species contained in the toxicity data for the PAH mixtures ESB (Section 3.2.1), effect concentrations in sediment can be calculated that are specific for that organism (using procedures in Section 4). These values could then be used to directly judge whether the absence of toxicity in the toxicity test would be expected from the corresponding level of sediment contamination.

If the exceedance of the PAH ESB is sufficient that one would expect effects in a toxicity test but they were not observed, it is prudent to initially evaluate the partitioning behavior of the chemical in the sediment based on sediment organic carbon content. Later evaluations may require evaluating partitioning based on other partitioning phases as described in Section 6.8. This is performed by isolation of interstitial water from the sediment and analyzing it for the same PAHs measured in the solid phase. Predicted concentrations of chemicals in the interstitial water can be calculated from the measured concentrations in the solid phase (normalized to organic carbon)

$$C_d = C_{OC} / K_{OC} \qquad (6\text{-}4)$$

For chemicals with $\log_{10}K_{OW}$ greater than 5.5, corrections for DOC binding in the interstitial water will be necessary

$$C_d = C_{OC}/K_{DOC} \qquad (6\text{-}5)$$

If the measured chemical in the interstitial water is substantially less (e.g., 2-3 fold lower or more), it suggests that the organic carbon in that sediment may not partition similarly to more typical organic carbon, and derivation of site-specific ESBs based on interstitial water may be warranted (U.S.EPA, 2003b).

6.5 Photo-Activation

6.5.1 Overview

Research over the last decade has shown that the presence of ultraviolet (UV) light can greatly enhance the toxicity of many PAHs. This "photo-activated" toxicity has been shown to cause rapid, acute toxicity to several freshwater and marine species including fish, amphibians, invertebrates, plants and phytoplankton (Bowling et al., 1983; Cody et al., 1984; Kagan et al., 1984; Landrum et al., 1984a,b; Oris et al., 1984; Allred and Giesy, 1985; Kagan et al., 1985; Oris and Giesy, 1985, 1986, 1987; Gala and Giesy, 1992; Huang et al., 1993; Gala and Giesy, 1994; Ren et al., 1994; Arfsten et al., 1996; Boese et al., 1997; Huang et al., 1997; McConkey et al., 1997; Pelletier et al., 1997; Diamond and Mount, 1998; Hatch and Burton, 1998; Boese et al.,1999; Monson et al., 1999; Spehar et al., 1999; Pelletier et al., 2000; Barron et al., 2003). Depending on the organism and exposure regime, photo-activation can increase toxicity of certain PAHs by one to four orders of magnitude over that caused by narcosis.

The mechanism for phototoxicity has been related to the absorption of ultraviolet radiation (UV) by the conjugated bonds of selected PAH molecules

$$PAH + UV \rightarrow PAH^* + O_2 \rightarrow PAH + O_2^* \qquad (6\text{-}6)$$

This excites the PAH molecules to a triplet state (PAH*) which rapidly transfers the absorbed energy to ground state molecular oxygen (O_2) forming excited singlet oxygen intermediaries (O_2*) (Newsted and Giesy, 1987). Although extremely short-lived (2 to 700 μs), oxygen intermediaries are highly oxidizing and can cause severe tissue damage upon contact. Despite the many different parent PAHs and related alkylated

forms, not all PAHs induce photo-activated toxicity. Those PAHs that are photo-activated can be predicted using various molecular physical-chemical variables (Newsted and Giesy, 1987; Oris and Giesy, 1987); however, the Highest Occupied Molecular Orbital - Lowest Unoccupied Molecular Orbital gap model (HOMO-LUMO) has been the most successful (Mekenyan et al., 1994a,b; Veith et al., 1995a,b; Ankley et al., 1996; Ankley et al., 1997). As research on the nature of photo-activated toxicity has evolved, certain key elements of this phenomena have been better defined including interactions of UV and PAH dose, effects of temperature, humic substances, organism behavior, turbidity, dissolved oxygen, mixtures, photoperiod and additivity (Oris et al., 1990; McCloskey and Oris, 1991; Ankley et al., 1995, 1997; Ireland et al., 1996; Hatch and Burton,1998, 1999; Erickson et al., 1999; Nikkilä et al., 1999; Weinstein and Oris 1999, Weinstein 2002).

Several studies have been performed with sediments contaminated with PAHs to assess the importance of photo-activated toxicity in the benthos (Davenport and Spacie, 1991; Ankley et al., 1994; Monson et al., 1995; Sibley et al., 1997; Swartz et al., 1997; Boese et al., 1998; Kosian et al., 1998; Boese et al., 1999; Spehar et al., 1999; Wernersson et al., 1999; Pelletier et al., 2000). These studies conclude that photo-activated toxicity may occur in shallow water environments; however, the magnitude of these effects are not as well characterized as in water-only exposures and are probably not as dramatic as those observed in the water column. Comparisons by Swartz et al. (1995) suggest that responses of benthic communities in PAH-contaminated sites correlate well with the toxicity that is predicted based on narcosis, suggesting that photo-activation was not a major confounding factor for those environments. However, Boese et al., (1997) and Pelletier et al. (In prepartion) show that life history of benthic organisms is critical to assessing whether or not photo-activated toxicity will occur. For example, several marine species which frequently encounter ultraviolet radiation during low tide are not vulnerable to photo-activated toxicity due to light protective adaptation (e.g., shells, pigments, borrowing). Additionally, there is evidence that maternal transfer of PAHs from benthic adult bivalves to pelagic embryos does occur (Pelletier et al., 2000).

6.5.2 Implications to Derivation of ESB

Because the PAH mixture ESB derived here is based on narcosis, if there is additional toxicity caused by photo-activation it may cause the ESB to be underprotective. At present, the magnitude of potential errors can not be specifically quantified, and are the subject of scientific debate (Swartz et al., 1997; Boese et al., 1999; Diamond and Mount, 1998; McDonald and Chapman, 2002). If photoactivation of PAHs is ecologically relevant, it is probably most significant primarily for organisms that inhabit very shallow or very clear water. This is because of the rapid attenuation of ultraviolet radiation in the water column (Pickard and Emery 1982; Wetzel, 1983). For example, <25% of incident UV penetrates below the first meter of water in productive aquatic systems. In areas where PAH-contaminated sediments are present in shallow environments the risk of photo-activated toxicity is greater and a site-specific ESB may need to be generated that considers this potential risk (U.S. EPA, 2003b).

6.6 Teratogenicity and Carcinogenicity

This subsection presents an analysis intended to determine if the ESB for PAH mixtures of <1.0 $\Sigma ESBTU_{FCV}$ is protective for non-narcosis modes of toxic action of individual PAHs. Published articles were screened for applicable data on teratogenic (Appendix G) and carcinogenic (Appendix H) effects of individual PAHs and their mixtures. Five laboratory studies with benzo(a)pyrene (BaP), predominantly water exposures, and one with anthracene were selected for analysis of teratogenic effects; two laboratory studies with BaP were selected for analysis of carcinogenic effects (Table 6-4). In the teratogen studies, typically radio-labeled BaP was used to quantify the accumulation of the PAH and its metabolites in fish ranging in lifestage from

Table 6-4. Teratogenic and carcinogenic effects of benzo(a)pyrene (BaP) and anthracene on freshwater and saltwater fishes. Measured concentrations of exposure are converted to sediment concentrations (C_{OC}) likely to result in the equivalent effect using EqP and SAR methodology.

Organism/ Chemical	Measured C_d[A] (µg/L)	C_d-derived C_{OC} (µg/g$_{OC}$)	Measured C_{ORG}[B] (µg/g)	f_{Lipid}	C_L[B] (µg/g Lipid)	C_L-derived C_{OC} (µg/g$_{OC}$)	References
TERATOGENIC EFFECTS							
FRESHWATER							
Fathead minnow eggs Anthracene	-	-	8.8	0.06	147	219	Hall and Oris, 1991
Topminnows BaP	>3.81[C] (1,000)	>3810	9	0.06	150	256	Goddard et al., 1987
Rainbow trout eggs BaP	0.21	210	1.9	0.05	38.6	66	Hannah et al., 1982 Hose et al., 1984
SALTWATER							
English sole eggs BaP	-	-	157	0.03	5233[D]	8,937[D]	Hose et al., 1981
Sand sole eggs BaP	0.1	100	2.1	0.03	70	120	Hose et al., 1982
Calif. grunion eggs	>3.81 (5)	>3810	1	0.03	33.3	57	Winkler et al., 1983
Calif. grunion eggs	>3.81 (24)	>3810	10.5	0.03	350	598	Winkler et al., 1983
Calif. grunion eggs	>3.81[C] (869)	>3810	20	0.03	666	1137	Winkler et al., 1983
CARCINOGENIC EFFECTS							
FRESHWATER							
Japanese medaka	>3.81[C] (261)	>3840	-	-	-	-	Hawkins et al., 1988, 1990
Guppy	>3.81[C] (209)	>3840	-	-	-	-	Hawkins et al., 1988, 1990

[A] If the concentration of BaP exceeded its solubility of 3.81 µg/L, the published concentration in water is listed in parenthesis with the solubility of 3.81 µg/L listed above as the concentration of exposure. Therefore the maximum C_{OC} value for these exposures is 3840µg BaP/g$_{OC}$.

[B] Concentrations in eggs on a wet weight basis are converted to concentrations on a lipid basis using lipid concentrations (f_{Lipid}) from Table 1 in Kamler (1992).

[C] Water concentrations of BaP were not stable throughout the duration of the experiment.

[D] The solubility of BaP in water theoretically limits the maximum concentration in eggs to ~3,840 µg/g lipid and in sediments to ~3,840 µg/g$_{OC}$, but metabolites of BaP will likely be included in radio-labeled quantification of total BaP equivalents.

embryo to adults. The water PAH concentrations associated with teratogenic and carcinogenic effects were generally high and steady-state was not always achieved. The solubility limit in water for BaP of 3.81 µg/L was exceeded in 6 of 8 experiments (Table 6-4). In contrast, for seven of the experiments, the BaP concentration in eggs or fish tissue was also listed as an observed effect concentration. The theoretical solubility-limited maximum of 3840 µg BaP/g lipid was exceeded only in one of the experiments. For these reasons, when the concentration of BaP plus metabolites was measured in the eggs or tissue of the organism, this concentration was considered the most valid representation of the true observed exposure concentration and the water concentration was not used in further analysis. Elutriates from crude oil contained non-PAH compounds and the relationship of total PAH concentrations in the study vs total PAH as defined in this document were difficult to determine in the Carls et al. (1999) study; therefore, these data were also excluded from this analysis. Although metabolism of PAHs is known to occur in invertebrates such as polychaetes, mollusks and crustaceans (McElroy et al., 2000), data on the potential carcinogenic effects of the metabolites is unknown.

As indicated in Table 6-4 and Appendix H, the database for carcinogenic effects of PAHs on aquatic (fish) species from laboratory studies is limited. Most of the available data are from studies of epizootic outbreaks of neoplasia (tumors) from highly contaminated field sites such as the Black River, Ohio (see Baumann and Harshbarger 1998 for a review) or Puget Sound, WA (Malins et al., 1987, Myers et al., 1990), to mention only a notable few. The applicability of these field studies to a causal relationship between carcinogenic effects observed and PAH concentrations is limited by the possible interactive effects of the PAHs with PCBs and other simultaneously occurring chemicals. The bulk of laboratory experimental evidence for carcinogenic effects of PAHs is based on the distribution of neoplasms in fish species exposed to PAH-enriched sediment extracts (Black, 1983; Metcalfe et al., 1988; Fabacher et al., 1991), dietary exposures or inter-peritoneal injection (Hendricks et al., 1985), or intermittent water exposures of 7,12-dimethylbenzanthracene (Schultz and Schultz, 1982). These studies are listed in Appendix H for completeness, but were not included in Table 6-4 for further analysis. This is because the exposure regime or concentrations of individual or mixtures of PAHs were not provided in sufficient detail to permit critical measured sediment concentrations, or sediment concentrations derived from concentrations in water or tissue, to be compared to the observed carcinogenic effects. The study with 7,12-dimethylbenzanthracene (Schultz and Schultz, 1982) was not considered for analysis because this PAH is not commonly measured as part of environmental monitoring programs (see Table 6-2).

A far more extensive database exists on the influence of PAHs on various aspects of tumor biology, such as PAH-DNA adduct formation and phase I (oxidation, reduction, and hydrolysis reactions) and phase II (glucuronidation and glutathione conjugation) metabolism of individual compounds. However, as indicative of cytotoxicity as these biomarkers may or may not be, they have been excluded from the analysis for the explicit purposes of this subsection. The methods of PAH exposure that were useful for this analysis were aqueous (Hannah et al., 1982; Hose et al., 1982, 1984; Winkler et al., 1983; Goddard et al., 1987; Hawkins et al., 1988, 1990), maternal (Hall and Oris, 1991), or inter-peritoneal injection of adult English sole (Parophrys vetulus) followed by measurement of concentrations in embryos (Hose et al., 1981).

6.6.1 Calculations

When the measured concentration of the PAH dissolved in water (C_d; µg/L) associated with a teratogenic or carcinogenic effect was available it was multiplied by its K_{OC} (L/kg_{OC}) x 10^{-3} to derive an equivalent effect concentration in sediment (C_d-derived C_{OC}; µg/g_{OC}), as per the EqP methodology (Table 6-4; Appendix G and H). When the measured concentration of the PAH in eggs or tissue (C_L; μg PAH/g lipid) associated with an

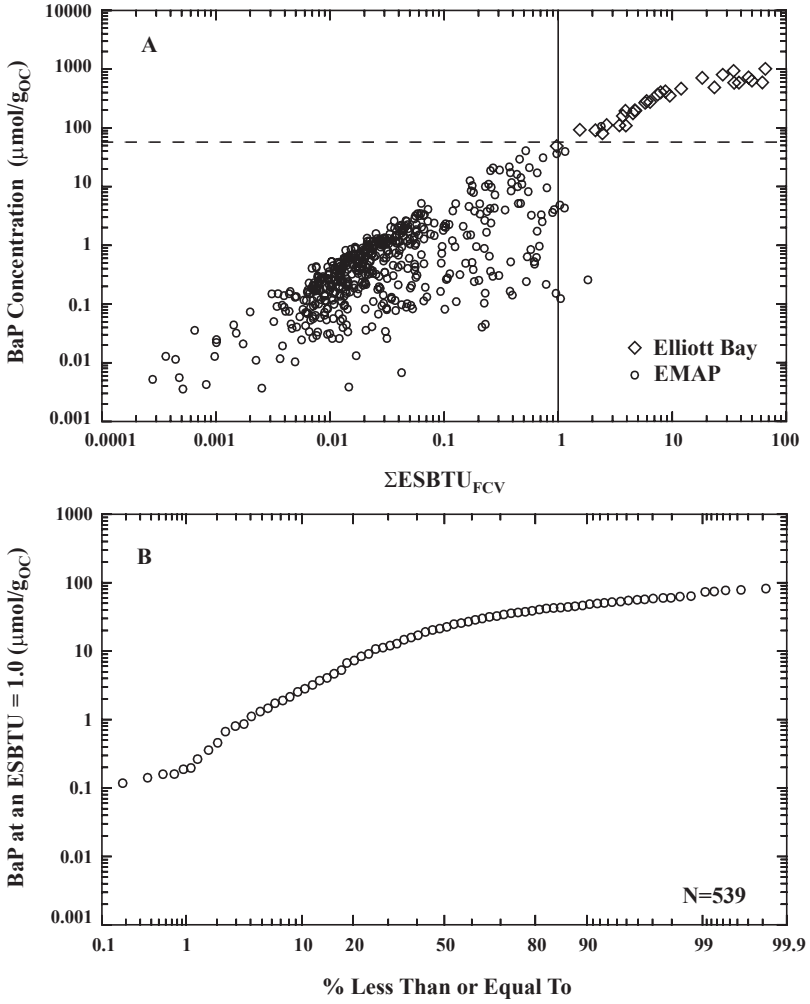


Figure 6-3. BaP concentration of 539 sediment samples from the EMAP and Elliott Bay datasets versus (A) the ΣESBTU$_{FCV}$ values of 34 PAHs and (B) a probability plot of these BaP concentrations at an ΣESBTU$_{FCV}$ = 1.0.

effect was available, its equivalent effect concentration in sediment (C_L-derived C_{OC}; μg/g_{OC}) was calculated using the equation.

$$\log_{10}C_{OC} = 0.00028 + \log_{10}C_L + 0.038\log_{10}K_{OW} \qquad (6\text{-}7)$$

6.6.2 Critical Sediment Concentrations for Teratogenic and Carcinogenic Effects versus ESBs for PAH Mixtures

The critical sediment concentrations (i.e., C_d- or C_L-derived C_{OC}) that would be expected to cause teratogenic or carcinogenic effects on the five freshwater and three saltwater fishes exposed to BaP ranged from 57 to 8,937 µg/g_{OC}; the only C_{OC} for anthracene was 219 µg/g_{OC} (Table 6-4). The majority of C_{OC} values were derived using concentrations measured in fish eggs. Six of the nine C_{OC} concentrations for BaP were less than the solubility-limited maximum concentration of 3,840 µg/g_{OC}. The C_{OC} value of 8,937 µg /g_{OC} is retained because the concentrations in the eggs probably included metabolites of BaP that are quantified as total BaP equivalents in the radio-label analysis. The C_{OC} values for individual PAHs in sediments were then compared to PAH concentrations in monitored field sediments to determine if teratogenic or carcinogenic effects might occur in sediments having <1.0 ΣESBTU$_{FCV}$. This analysis was used to determine if the ESB derived from the narcosis mode of action was protective of teratogenic or carcinogenic effects.

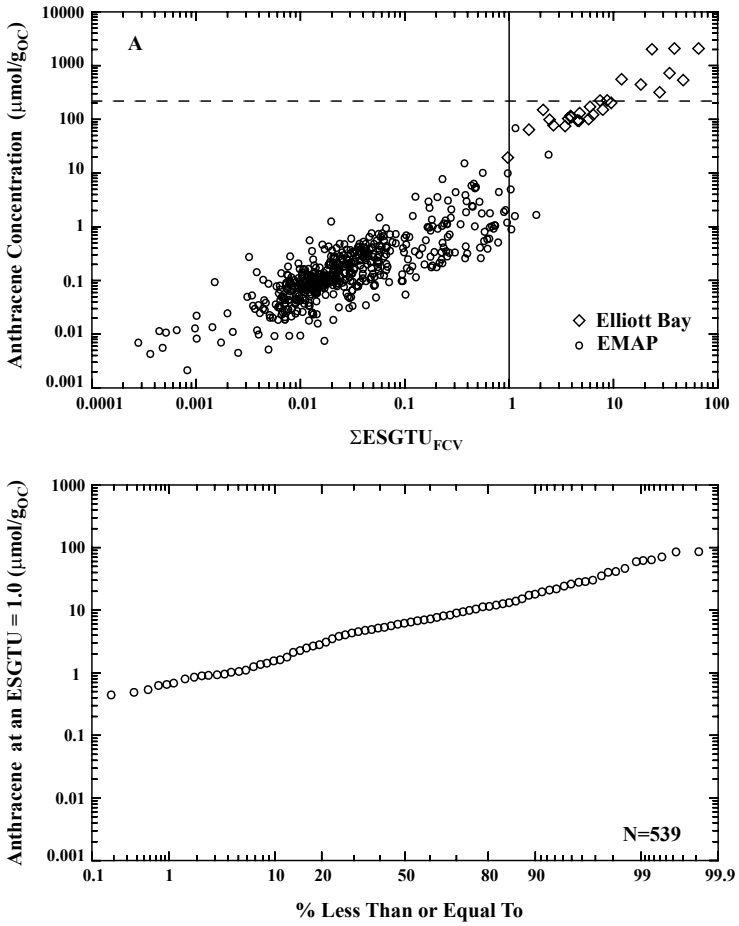


Figure 6-4. Anthracene concentration of 539 sediment samples from the EMAP and Elliott Bay datasets versus (A) the ΣESBTU$_{FCV}$ values of 34 PAHs and (B) a probability plot of these anthracene concentrations at an ΣESBTU$_{FCV}$ 1.0.

The database from the U.S. EPA EMAP (U.S. EPA 1996b, 1998) and Elliot Bay (Ozretich et al., 2000) sediment monitoring programs were used to compare the BaP (Figure 6-3A) or anthracene (Figure 6-4A) concentration of 539 sediment samples where 34 PAHs, or 33 of 34 PAHs for Elliott Bay, were measured versus the ΣESBTU$_{FCV}$ for all PAHs measured in those sediments. The lowest critical sediment concentration for teratogentic or carcinogenic effects is indicated with a solid line at 57 µg/g$_{OC}$ for BaP and at 219 μg/g$_{OC}$ for anthracene. None of the sediments having <1.0 ΣESBTU$_{FCV}$ contained BaP or anthracene at concentrations likely to cause the teratogenetic or carcinogenic effects reported in Table 6-4. The same database of PAH concentrations in field sediments was used to calculate the sediment-specific BaP:ΣESBTU$_{FCV}$ ratio and the sediment-specific anthracene: ΣESBTU$_{FCV}$ ratio. The total PAH concentration in each of the 539 sediments was multiplied by its sediment-specific ratio to determine the BaP or anthracene concentration for the sediment if the ΣESBTU$_{FCV}$ was equal to 1.0. Probability plots of the calculated concentrations for BaP and anthracene at 1.0 ΣESBTU$_{FCV}$ are in Figures 6-3B and 6-4B, respectively. The dashed lines represent the critical sediment concentration of 57 µg/g$_{OC}$ for BaP and 219 µg/g$_{OC}$ for anthracene. Based on this analysis, none of the sediments for anthracene and only 3.53% of the sediments for BaP would be expected to produce teratogenic or carcinogenic effects if the proportions of BaP or anthracene in these sediments were maintained and the concentrations of each of the other PAHs were increased so that all sediments contained 1.0

$\Sigma ESBTU_{FCV}$. The approach of examining these relationships individually with BaP or anthracene may be flawed because it may under-represent the teratogenic of carcinogenic contributions of other PAHs with the same mode of action in the PAH mixture. However, at present insufficient data are available to appropriately sum the contributions of multiple teratogenic or carcinogenic PAHs.

6.7 Equilibrium and ESBs

Care must be used in application of ESBs in disequilibrium conditions. In some instances site-specific ESBs may be required to address this condition (U.S. EPA, 2003b). Benchmarks based on EqP theory assume that nonionic organic chemicals are in equilibrium with the sediment and interstitial water, and that they are associated with the sediment primarily through absorption into sediment organic carbon. In order for these assumptions to be valid, the chemical must be dissolved in interstitial water and partitioned into sediment organic carbon. The chemical must, therefore, be associated with the sediment for a sufficient length of time for equilibrium to be reached. With PAHs, the absence of toxicity when the ESB is exceeded may be because of the presence of less available PAHs associated with soot, coal or similar materials in sediments (see discussion in Section 6.8). Alternatively, disequilibrium exists, and ESB may be over-protective, when PAHs occur in sediments as undissolved liquids or solids; although the use of solubility limited acceptable sediment concentrations should adequately account for this.

In very dynamic locations, with highly erosional or depositional sediments, the partitioning of nonionic organic chemicals between sediment organic carbon and interstitial water may only attain a state of near equilibrium. Likewise, nonionic organic chemicals with high $\log_{10}K_{OW}$ values may come to equilibrium in clean sediment only after a period of weeks or months. Equilibrium times are shorter for chemicals with low $\log_{10}K_{OW}$ values and for mixtures of two sediments with similar organic carbon-normalized concentrations, each previously at equilibrium. This is particularly relevant in tidal situations where large volumes of sediments are continually eroded and deposited, yet near equilibrium conditions between sediment and interstitial water may predominate over large spatial areas. For locations where times are sufficient for equilibrium to occur, near equilibrium is likely the rule and disequilibrium uncommon. In many environments, disequilibrium may occur intermittently, but in those cases ESBs would be expected to apply when the disturbance abates. In instances where long-term disequilibrium is suspected, application of site-specific methodologies may be desirable (U.S. EPA, 2003b).

6.8 Other Partioning Phases

6.8.1 Overview

In general, laboratory studies with PAHs have shown the same partitioning behavior demonstrated by many classes of nonpolar organic contaminants (Chiou et al., 1979,1983; Karickhoff et al., 1979; Means et al., 1980; Di Toro et al., 1991). However, there are some data indicating that PAHs do not always follow equilibrium partitioning behavior in the environment. Specifically, some studies have reported larger partitioning coefficients for PAHs in field-collected sediments than is predicted based on laboratory or theoretically-generated $\log_{10}K_{OW}/K_{OC}$ values (Prahl and Carpenter, 1983; Socha and Carpenter, 1987; Broman et al., 1990; McGroddy and Farrington, 1995; Maruya et al., 1996; McGroddy et al., 1996). The observed differences in partitioning of PAHs may relate to differences in PAH sources with the speculation that PAHs from pyrogenic sources (e.g., soot carbon, coal or similar materials) may be more strongly associated with the particulate phase than PAHs from some petrogenic sources (Readman et al., 1984; Socha and Carpenter, 1987; McGroddy and Farrington, 1995; Meador et al., 1995; Naes et al., 1995; Chapman et al., 1996; Maruya et al., 1996; McGroddy et al., 1996; Gustafsson and Gschwend, 1997; Gustafsson et al., 1997; Naes and Oug, 1997; de Maagd et al. 1998; Naes and Oug 1998;

Naes et al., 1998; Bucheli and Gschwend 2000; Jonker and Smedes 2000; Ozretich et al., 2000; Jonker and Koelmans 2001, 2002a,b; Accardi-Dey and Gschwend 2002, 2003). The result is that PAH concentrations in interstitial water are significantly lower compared to the organic carbon-based sediment concentration from laboratory or theoretically-predicted K_{OC} values and, presumably, exhibit correspondingly lower bioavailability. Several studies have proposed that the lack of observable biological effects from sediments (and other samples) containing high concentrations of presumably bioavailable PAHs is related to this phenomena (Farrington et al., 1983; Varanasi et al., 1985; Bender et al., 1987; Hickey et al., 1995; Knutzen, 1995; Chapman et al., 1996; Paine et al., 1996; Maruya et al., 1997; Oug et al., 1998; Lamoureux and Brownawell 1999; Naes et al., 1999; West et al., 2001; Talley et al., 2002).

The mechanisms causing these field observations of unusual PAH partitioning are not well understood. One explanation proposes that PAHs condense into the soot matrix during particle formation, and are thereby sterically inhibited from partitioning to interstitial water as would be expected under equilibrium conditions. A second perspective assumes that the soot fraction represents a second partitioning phase in addition to normal organic carbon. The partitioning of PAHs from this phase approximates the equilibrium behavior assumed for normal organic carbon, but have a much higher partition coefficient than biologically-derived organic carbon (represented by K_{OC}) (Gustafsson and Gschwend, 1997, 1999). Methods for measuring the soot carbon fraction in sediments (f_{sc}) continue to be developed and evaluated (Verardo 1997; Gustafsson et al., 1997; Karapanagioti et al., 2000; Gelinas et al., 2001; Currie et al., 2002; Gustafsson et al., 2001; Song et al., 2002) but no one method is recognized as most accurate, although those based on Gustaffson et al. (1997) are probably used most frequently.

Once partition coeffifients are available and f_{SC} can be measured, the soot phase can then be incorporated into an expanded partitioning equation with two partitioning terms

$$K_p = f_{OC} K_{OC} + f_{SC} K_{SC} \qquad (6\text{-}8)$$

where, K_p is the partition coefficient for the expanded partitioning equation, f_{OC} and f_{SC} are the fraction organic carbon and fraction soot carbon, respectively, and K_{OC} and K_{SC} are the organic carbon and soot carbon partition coefficients. Recently, Bucheli and Gustaffson (2000) and Accardi-Dey and Gschwend (2002; 2003) proposed a new version of Equation 6-8 which includes a non-linear term for the soot carbon contribution to partitioning

$$K_p = f_{OC} K_{OC} + f_{SC} K_{SC} C_d^{n-1} \qquad (6\text{-}9)$$

where, the exponential 'n' is the Freundlich term used to fit the non-linear relationship between particulate and dissolved PAH. This description of the interaction of PAHs and soot carbon is more accurate but is currently limited in practicality by the lack of values for K_{SC} and n for many PAHs.

Another phase for which there is less data available as compared to soot carbon but which may also alter the partitioning and bioavailability of PAHs is non-aqueous phase liquids (NAPLs) like coal tar found at manufactured gas plant sites (Lane and Loehr 1992; Luthy et al., 1994; Mahjoub et al., 2000). The significance of these liquids relative to the benthic toxicity of PAHs is not yet understood fully.

6.8.2 Implications to Derivation of ESB

Irrespective of the mechanisms, these issues have the potential to affect the predictive power and accuracy of the PAH mixtures ESB. For soot, coal and similar materials, their presence are associated with reduced concentrations of PAH in interstitial water, one would presume that this results in decreased bioavailability of PAHs, a phenomenon demonstrated by West et al. (2001). This, in turn, would make the PAH mixtures ESB derived here overprotective, because the K_{OC}-based partitioning model would overpredict chemical activity and, therefore, concentrations of PAH in interstitial water and in organisms.

Importantly, most sediments are expected to

contain insufficient concentrations of PAHs to exceed the ESB. Therefore, even if partitioning to soot, coal and similar materials reduces the interstitial water concentration and biological availability of the PAHs, the partitioning effect is not important because PAH concentrations in the sediment are judged by the ESB as acceptable without invoking complex measurements of partitioning to soot, coal and similar materials. Further, most sediments where empirical data on partitioning that demonstrates soot, coal and similar materials are important are sediments that relative to the ESB are uncontaminated. Also, for sediments that have concentrations of PAHs in excess of the ESB, data suggest minimal error in ignoring partitioning to soot, coal and similar materials and ascribing partitioning to only organic carbon. Most applications of the PAH mixture narcosis model to toxicity data for field-collected sediments show good predictive ability for the ESB (see Section 5.3). This may be because most sediments that are sufficiently contaminated to cause narcosis are contaminated by PAH sources that exhibit normal partitioning behavior, such as creosote and other petrogenic sources. In their study of PAH-contaminated sediments, Ozretich et al. (2000) found that discrepancies between measured and predicted partitioning behavior predominated in sediments with lower PAH concentrations, while those with higher PAH concentrations showed partitioning behavior closer to that predicted from published K_{OW}/K_{OC} relationships. This differential behavior was attributed to the presence of two PAH sources, with creosote being the source causing the highest levels of contamination and toxicity.

In cases where it is suspected that soot, coal, or other materials including coal tars and other NAPLs may be causing unusual partitioning, direct measurement of PAH concentrations in interstitial water may be used to evaluate this possibility and, where necessary, derive site-specific sediment benchmarks which account for local differences in partitioning behavior (see U.S. EPA 2003b).

6.9 Aqueous Solubility Under Non-Standard Conditions

It has been long established that organic compounds are generally less soluble in aqueous solutions at colder temperatures than at warmer, and in salt solutions such as seawater, than in freshwater, a phenomenon termed the salting-out effect (May, 1980; Schwarzenbach et al., 1993; Xie et al., 1997). Setschenow (1889) derived an empirical relationship for the magnitude of the salting-out effect

$$\log_{10}({}^{t}S_0 / {}^{t}S_{‰}) = K_S\, C_{salt} \qquad (6\text{-}9)$$

where ${}^{t}S_0$ and ${}^{t}S_{‰}$ are the aqueous solubilities of the solute in fresh and saltwater (mol/L) at a given temperature (t in the units °C), respectively, K_S is

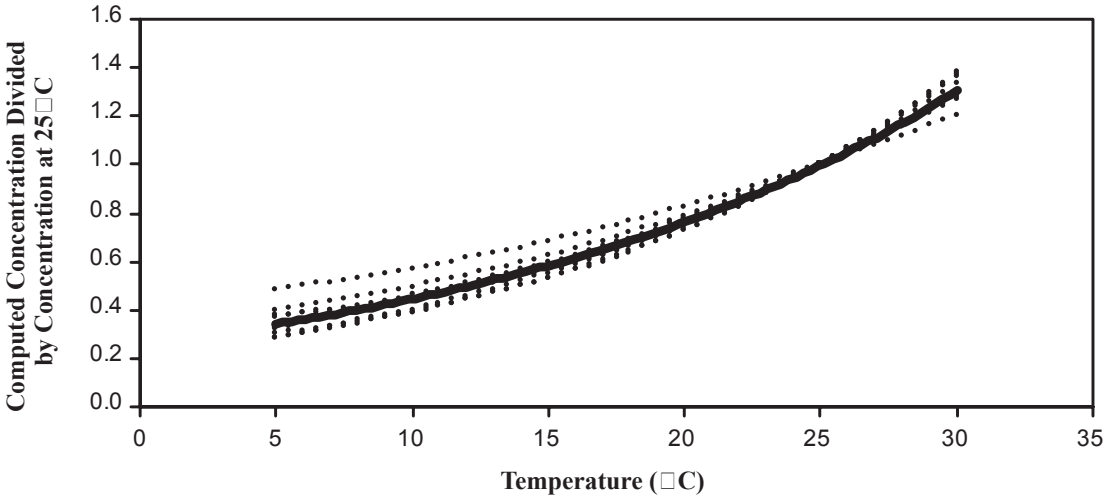


Figure 6-5. Computed solubilities of nine PAHs relative to their 25°C solubilities as a function of temperature.

the Setschenow constant (L/mol) for the salt solution and the solute of interest, and C_{salt} is the molar salt concentration. A one molar salt solution (NaCl) is approximately equivalent to 48‰ sea water (Owen and Brinkley, 1941), and K_S was found to be essentially invariant with temperatures from 1 to 30°C, averaging 0.28 ± 0.02 (mean ± SE) (May, 1980) for 9 PAHs. Temperature has been shown to have a non-linear effect on PAHs solubilities (May, 1980). Concentrations of nine PAHS (naphthalene, fluorene, phenanthrene, 1-methylphenanthrene, anthracene, fluoranthene, pyrene, benz(a)anthracene, and chrysene) were computed for distilled water at temperatures between 5 and 30°C using the relationships of May (1980) and are compared with the compound's concentrations at 25°C (Figure 6-5). The least-squares exponential representation of the data is as follows

$$({}^{t}S_0 = {}^{25}S_0) = 0.261\ e^{0.0536t},\ r^2 = 0.959 \qquad (6\text{-}10)$$

where ${}^{25}S_0$ is the commonly reported solubility of a compound at 25°C in freshwater. Although naphthalene's solubility has the least response to temperature of PAHs, estimates from Equation 6-10 are only +8% and -30% inaccurate for naphthalene at the temperature extremes (Figure 6-5).

The solubility of PAHs under environmental conditions can be estimated from the following relationship that is a combination of Equations 6-9 and 6-10 using the average Setschenow constant

$${}^{t}S_{‰} = {}^{t}S_0\ 10^{-0.000583‰} \qquad (6\text{-}11)$$

where ‰ is the salinity of the sea water. This correction for solubility can be used as part of the procedures to modify this ESB for site-specific conditions.

Section 7

Sediment Benchmark Values: Application and Interpretation

7.1 Benchmark Value

The procedures described in this document and in the "Technical Basis for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Nonionic Organics" (U.S. EPA, 2003a) indicate that, except possibly where a locally important species is very sensitive or benthic organisms are exposed to both significant amounts of PAHs and UV light, benthic organisms should be acceptably protected from the effects of PAH mixtures in freshwater and saltwater sediments if the $EESBTU_{FCV}$ is less than or equal to 1.0:

$$ESB = \Sigma ESBTU_{FCV} = \sum_{i} \frac{C_{OCi}}{C_{OC,PAHi,FCVi}} \leq 1.0 \qquad (7\text{-}1)$$

Freshwater or saltwater sediments containing ≤ 1.0 $\Sigma ESBTU_{FCV}$ of the mixture of the 34 PAHs or more PAHs are acceptable for the protection of benthic organisms, and if the $\Sigma ESBTU_{FCV}$ is greater than 1.0, sensitive benthic organisms may be unacceptably affected. PAHs.

As indicated, this sediment-specific benchmark is the sum of the quotients of the concentrations of individual PAHs in a sediment, on an organic carbon basis, each divided by its respective $C_{OC,PAHi,FCVi}$. At a minimum, the definition of total PAHs for this ESB requires quantification of the 34 PAHs analyzed by the U.S. EPA as part of the EMAP and REMAP programs (PAHs are identified in bold in Table 3-4).

The ESB is intended to protect benthic organisms from direct toxicity associated with exposure to PAH-contaminated sediments. The ESB does not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with PAHs or the potential for bioaccumulation and trophic transfer of PAHs to aquatic life, wildlife or humans.

7.2 Special Considerations

To establish a national benchmark that is widely applicable, certain issues must be considered. It is possible that site-specific conditions may affect the broad applicability of such a benchmark. These include:

1. Fewer than 34 PAHs have been measured. Particularly in cases where historical data are being examined, chemistry data may be available for fewer than the 34 PAHs recommended for this benchmark. Calculating $\Sigma ESBTU_{FCV}$ directly using fewer PAHs will cause the benchmark to be underprotective because PAH mixtures found in the environment typically contain substantial concentrations of PAHs outside the suites of 13 or 23 PAHs commonly measured in monitoring programs. The analysis of PAH distributions across many geographic regions has been used to develop uncertainty factors that can be used to adjust $\Sigma ESBTU_{FCV}$ based on subsets of 13 or 23 PAHs with varying degrees of certainty (see Section 6.2). In some applications using these uncertainty factors, it may be important to minimize the frequency of false negatives (sediments judged to be acceptable when they are not). For these cases, the $\Sigma ESBTU_{FCV}$ calculated from a subset of 13 PAHs (see Table 6-1 for listing) can be multiplied by 11.5, or the $\Sigma ESBTU_{FCV}$ calculated from a subset of 23 PAHs

(see Table 6-1 for listing) can be multiplied by 4.14 to achieve 95% confidence that the actual $\Sigma ESBTU_{FCV}$ for all 34 PAHs would not be higher than the calculated value. In this case, the uncertainty for the 95% confidence level is applied. This means that most of the sediments may actually contain fewer $\Sigma ESBTU_{FCV}$ than indicated by the calculation. In cases where less conservative assumptions are appropriate, factors with lower confidence can be applied, as detailed in Section 6.2.

Use of the uncertainty factors from Section 6.2 assumes that the relative frequency distributions of PAHs in sediments used to calculate the factors are similar to those of the sediments to which the uncertainty factors are applied. This assumption is likely significantly violated for sediments containing predominately petrogenic PAHs. While the uncertainty factors can be used to derive the $\Sigma ESBTU_{FCV}$, this value should not be considered as an ESB. In principal, ESBs based on the $\Sigma ESBTU_{FCV}$ calculated using a minimum of the 34 specified PAHs and can be used to make important sediment decisions. In contrast, important sediment decisions should not be made using $\Sigma ESBTU_{FCV}$ values when fewer PAHs, such as the 13 or 23 PAHs commonly quantified, and uncertainty factors. To avoid errors introduced by the use of uncertainty factors, wherever possible, a more complete PAH chemical analysis should be undertaken with concentrations for a minimum of the 34 specified PAHs analyzed.

2. Interaction of PAHs with UV light. Benchmarks calculated in this document are based on narcotic toxicity only and do not consider enhanced toxicity that can occur if PAH-exposed organisms are simultaneously exposed to UV light. In environments where significant sunlight penetrates to the sediment and benthic organisms are exposed to UV light, the ESB may be underprotective. Consult Section 6.5 for additional details.

3. Influence of soot carbon and coal on PAH partitioning. PAHs may partition less to interstitial water in sediments that contain soot and/or coal particles or similar materials that expected with typical organic carbon partitioning. This could cause the benchmark to be overprotective. The influence of these phases can be assessed by measuring concentrations of PAHs directly in interstitial water and comparing these measures with concentrations predicted by EqP or through quantification of partitioning to these other sediment phases. See Section 6.8 and the site-specific ESBs (U.S. EPA, 2003b) for further discussion. NAPLs are not directly addressed by this document, but may be expected to result in reduced interstitial water concentrations of PAHs.

4. Unusual composition of organic carbon. Partition coefficients used for calculating the national PAH mixture ESB are based on measured partitioning from natural organic carbon in typical field sediments. Some sediments influenced heavily by industrial activities may contain sources of organic carbon whose partitioning properties are not similar, such as rubber, animal processing wastes (e.g., hair or hide fragments), or wood processing wastes (bark, wood fiber or chips). Relatively undegraded woody debris or plant matter (e.g., roots, leaves) may also contribute organic carbon that results in partitioning different from that of typical organic carbon. Sediments with large amounts of these materials may show higher concentrations of chemicals in interstitial water than would be predicted using generic K_{OC} values, making the ESB underprotective. Direct analysis of interstitial water can be used to evaluate this possibility (see U.S. EPA, 2003a,b).

5. Presence of additional narcotic compounds. The PAH mixture ESB is based on the additivity of the narcotic toxicity of PAHs. However, some sediments may contain additional nonionic narcotic chemicals that would contribute to narcotic toxicity, such as chlorobenzenes or PCBs (note: PCBs may also cause adverse effects through bioaccumulation and transfer to higher trophic levels; these bioaccumulative effects are not addressed by this narcosis-based ESB and should be evaluated separately). The presence of additional nonionic narcotic chemicals may make the PAH mixture ESB underprotective, because the ESB itself only addresses that part of the narcotic potency caused by PAHs. Di Toro et al.

(2000) and Di Toro and McGrath (2000) describe methods by which the contributions of other narcotic chemicals can be incorporated into an ESB-type assessment.

6. Site-specific temperature and salinity corrections. Temperature and salinity both affect solubility of PAHs and can therefore affect the solubility-constrained maximum contribution of individual PAHs to the overall ESB. Solubilities used in this document are calculated for 25°C and salinities less than 1‰. Solubilities can be recalculated to meet site specific conditions using procedures described in Section 6.9. Within a temperature range of 0 to 35°C and salinities from 0 to 35‰, solubility can be expected to decrease by a factor of about 30 to 40% with decrease in temperature or increase in salinity. Site-specific recalculation of solubilities will only affect $\Sigma ESBTU_{FCV}$ in cases where the contribution of one or more PAHs are solubility constrained (see Section 6.9).

7.3 Summary

Benthic organisms should be acceptably protected from the narcotic effects of PAH mixtures in freshwater and saltwater sediments if the $\Sigma ESBTU_{FCV}$ is less than or equal to 1.0, and if the $\Sigma ESBTU_{FCV}$ is greater than 1.0, sensitive benthic organisms may be adversely affected**.** This ESB is intended to protect benthic organisms from direct toxicity associated with exposure to PAH-contaminated sediments. This ESB does not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with PAH mixtures or the potential for bioaccumulation and trophic transfer of PAH mixtures to aquatic life, wildlife or humans.

Section 8

References

Abernethy S, Bobra AM, Sliiu WY, Wells PG , Mackay D. 1986. Acute lethal toxicity of hydrocarbons and chlorinated hydrocarbons to two planktonic crustaceans: the key role of organism-water partitioning. *Aquat Toxicol* 8:163-174.

Abernethy S, Mackay D, McCarty L. 1988. Volume fraction correlation for narcosis in aquatic organisms: the key role of partitioning. *Environ Toxicol Chem* 7:469-481.

Academy of Natural Sciences. 1981. Early life stage studies using the fathead minnow (*Pimephales promelas*) to assess the effects of isophorone and acenaphthene. Final report to U.S. EPA, Cincinnati, OH. Academy of Natural Sciences, Philadelphia, PA. 26 pp.

Accardi-Dey A, Gschwend PM. 2002. Assessing the combined roles of natural organic matter and black carbon as sorbents in sediments. *Environ Sci Technol* 36:21-29.

Accardi-Dey A. Gschwend PM. 2003. Reinterpreting literature sorption data considering both absorption into OC and adsorption onto black carbon. *Environ Sci Technol* 37:99-106.

Adams D, O'Conner J, Weisberg SB. 1996. Sediment quality of the NY/NJ harbor system. An investigation under the Regional Environmental Monitoring and Assessment Program (R-EMAP). Draft Final Report. U.S. Environmental Protection Agency, Washington, DC.

Adams WJ, Kimerle RA, Mosher RG. 1985. Aquatic safety assessment of chemicals sorbed to sediments. In Cardwell RD, Purdy R, Bahner RC (eds.), *Aquatic Toxicology and Hazard Assessment: Seventh Symposium.* STP 854. American Society for Testing and Materials, Philadelphia, PA, pp. 429-453.

Adema, DMM. 1978. *Daphnia magna* as test organism in acute and chronic toxicity experiments. *Hydrobiol* 59:125-134.

Ahmad N, Benoit D, Brooke L, Call D, Carlson A, DeFoe D, Huot J, Moriarity A, Richter J, Shubat P, Veith G, Wallbridge C. 1984. Aquatic toxicity tests to characterize the hazard of volatile organic chemicals in water: A toxicity data summary - Parts I and II. EPA-600-3-84-009. U.S. Environmental Protection Agency, Washington, DC.

Aitchison J, Brown J. 1957. *The Lognormal Distribution*. Cambridge University Press, Cambridge, The United Kingdom.

Alexander HC, McCarty WM, Bartlett EA. 1978. Toxicity of perchloroethylene, trichloroethylene, 1,1,1-trichloroethane and methylene chloride to fathead minnows. *Bull Environ Contam Toxicol* 20:344-352.

Allred PM, Giesy JP. 1985. Solar radiation-induced toxicity of anthracene to Daphnia pulex. *Environ Toxicol Chem* 4:219-226.

American Society for Testing and Materials. 1993. Guide for conducting 10-day static sediment toxicity tests with marine and estuarine amphipods. E 1367-92. In: *Annual Book of ASTM Standards*, Vol. 11.04, Philadelphia, PA, pp 1138-1163.

American Society for Testing and Materials. 1998. Standard guide for conducting early life stage toxicity tests with fishes. E1241-92. In: *Annual Book ASTM Standards*. Philadelphia, PA. pp. 550-577.

Anderson B, Hunt J, Tudor S, Newman J, Tjeerdema R, Fairey R, Oakden J, Bretz C, Wilson C, LaCaro F, Stephenson M, Puckett M, Long E, Fleming T, Summers K. 1996. Chemistry, toxicity and benthic community conditions in sediments of the southern California bays and estuaries. Draft Report. California State Water Resources Control Board. Sacramento, CA.

Ankley GT, Collyard SA, Monson PD, Kosian PA. 1994. Influence of ultraviolet light on the toxicity of sediments contaminated with polycyclic aromatic hydrocarbons. *Environ Toxicol Chem* 13:1791-1796.

Ankley GT, Erickson RJ, Phipps GL, Mattson VR, Kosian PA, Sheedy BR, Cox JS. 1995. Effects of light intensity on the phototoxicity of fluoranthene to a benthic macroinvertebrate. *Environ Sci Technol* 29:2828-2833.

References

Ankley GT, Mekenyan OG, Kosian PA, Makynen EA, Mount DR, Monson PD, Call CJ. 1996. Identification of phototoxic polycyclic aromatic hydrocarbons in sediments through sample fractionation and QSAR analysis. *SAR QSAR Environ Res* 5:177-183.

Ankley GT, Erickson RJ, Sheedy BR, Kosian PA, Mattson VR, Cox JS. 1997. Evaluation of models for predicting the phototoxic potency of polycyclic aromatic hydrocarbons. *Aquat Toxicol* 37:37-50.

Arfsten DP, Schaeffer DJ, Mulveny DC. 1996. The effects of near ultraviolet radiation on the toxic effects of polycyclic aromatic hydrocarbons in animals and plants: a review. *Ecotoxicol Environ Safety* 33:1-24.

Barrick R, Becker S, Pastorok R, Brown L, Beller H. 1988. Sediment quality values refinement: 1988 update and evaluation of Puget Sound AET. Prepared by PTI Environmental Services for the Environmental Protection Agency, Bellevue, Washington.

Barron MG, Carls MG, Short JW, Rice SD. 2003. Photoenhanced toxicity of aqueous phase and chemically dispersed weathered Alaska north slope crude oil to pacific herring eggs and larvae. *Environ Toxicol Chem* 22: 650-660.

Battelle Ocean Sciences. 1987. Acute toxicity of phenanthrene to saltwater animals. Report to U.S. EPA Criteria and Standards Division. Battelle Ocean Sciences, Duxbury, MA.

Baumann PC and Harshburger JC. 1998. Long-term trends in liver neoplasm epizootics of brown bullhead in the Black River, Ohio. *Environ Monit Assess* 53:213:223.

Bence AE, Kvenvolden KA and Kennicutt II MC. 1996. Organic geochemistry applied to environmental assessments of Prince William Sound, Alaska, after the Exxon Valdez oil spill—a review. *Org Geochem* 24:7-42.

Bender ME, Roberts MH, deFur PO. 1987. Unavailability of polynuclear aromatic hydrocarbons from coal particles to the eastern oyster. *Environ Pollut* 44:243-260.

Bengtsson B, Renberg L, Tarkpea M. 1984. Molecular structure and aquatic toxicity - an example with C1 - C13 aliphatic alcohols. *Chemosphere* 13:613-622.

Bently RE, Heitmuller T III, Sleight BH, Parrish PR. 1975. Acute toxicity of chloroform to bluegill (*Lepomis macrochirus*), rainbow trout (*Salmo gaidneri*) and pink shrimp (*Penaeus duorarum*). Contract no. WA-6-99-1414-b. U.S. Environmental Protection Agency, Washington, DC.

Birge WJ, Black JA, Ballard ST, McDonnell WE. 1982. Acute toxicity testing with freshwater fish. Contract No. 68-01-6201. Final Report Task 11 and 111. U.S. EPA.

Black JJ. 1983. Field and laboratory studies of environmental carcinogenesis in Niagara River fish. *J. Great Lakes Res* 9(2): 326-334.

Blumer M .1976. Polycyclic aromatic compounds in nature. *Scientif Amer* 234:35-45.

Boehm PD, Page DS, Burns WA, Bence AE, Mankiewicz PJ, Brown JS. 2001. Resolving the origin of the petrogenic hydrocarbon background in Prince William Sound, Alaska. *Environ Sci Technol* 35:471-479.

Boese BL, Lamberson JO, Swartz RC, Ozretich RJ. 1997. Photoinduced toxicity of fluoranthene to seven marine benthic crustaceans. *Arch Environ Contamin Toxicol* 32:389-393.

Boese BL, Lamberson JO, Swartz RC, Ozretich RJ, Cole FA. 1998. Photoinduced toxicity of PAHs and alkylated PAHs to a marine infaunal amphipod (*Rhepoxynius abronius*). *Arch Environ Contamin Toxicol* 34:235-240.

Boese BL, Ozretich RJ, Lamberson JO, Swartz RC, Cole FA, Pelletier J, Jones J. 1999. Toxicity and phototoxicity of mixtures of highly lipophilic PAH compounds in marine sediments: can the ΣPAH model be extrapolated? *Arch Environ Contamin Toxicol* 36:270-280.

Bowling JW, Leversee GJ, Landrum PF, Giesy JP. 1983. Acute mortality of anthracene-contaminated fish exposed to sunlight. *Aquat Toxicol* 3:79-90.

Bradbury S, Carlson R, Henry T. 1989. Polar narcosis in aquatic organisms. *Aquat Toxicol Haz Assess* 12:59-73.

Brenniman G, Hartung R, Weber WJ, Jr. 1976. A continuous flow bioassay method to evaluate the effects of outboard motor exhausts and selected aromatic toxicants on fish. *Water Res* 10:165-169.

Brezonik PL. 1994. *Chemical Kinetics and Process Dynamics in Aquatic Systems.* CRC Press, Inc., Boca Raton, Florida.

Bridie AL, Wolff CJM, Winter M. 1979. The acute toxicity of some petrochemicals to goldfish. *Water Res* 13:623-626.

Bringman G, Kuhn R. 1959. Vergleichende wasser - toxikologische untersuchungen an bakterien, algen and kleinkrebsen. *Gesundheits Ingenieur* 80:115.

Broderius S, Kahl M. 1985. Acute toxicity of organic chemical mixtures to the fathead minnow. *Aquat Toxicol* 6:307-322.

Broman D, Naf C, Wik M, Renberg I. 1990. The importance of spheroidal carbonaceous oarticles (SCPs) for the distribution of particulate polycyclic aromatic hydrocarbons (PAHs) in an estuarine-like urban coastal water area. *Chemosphere* 21:69-77.

Brooke LT. 1991. Summary of results of acute and chronic exposures of fluoranthene with and without ultraviolet (UV) light to various organisms. Memorandum to Walter J. Berry, U.S. Environmental Protection Agency, NHEERL, Naragansett, RI. December 3, 1991.

Brooke LT. 1993. Conducting toxicity tests with freshwater organisms exposed to dieldrin, fluoranthene and phenanthrene. Report to Robert L. Spehar, Project Officer, U.S. EPA. Environmental Research Laboratory-Duluth, Duluth, MN. l8pp.

Brooke LT. 1994. Acute phenanthrene toxicity to *Daphnia magna*. Contract No. 68-C 1-0034 Report to Robert L. Spehar, WA No. 2-14. U.S. Environmental Protection Agency, Washington, DC.

Buccafusco RJ, Ells SJ, LeBlanc GA. 1981. Acute toxicity of priority pollutants to bluegill (*Lepomis macrochirus*). *Bull Environ Contam Toxicol* 26:446-452.

Bucheli TD, Gustafsson O. 2000. Quantification of the soot-water distribution coefficient of PAHs provides mechanistic basis for enhanced sorption observations. *Environ Sci Technol* 34:5144-5151.

Burgess RM, Scott KJ. 1992. The significance of in-place contaminated marine sediments on the water column: processes and effects. In G.A. Burton, Jr. (ed.) *Sediment Toxicity Assessment*. Lewis Publishing, Boca Raton, FL. pp. 129-154.

Burgess RM, Ryba SA, Cantwell MG. 2000a. Importance of organic carbon quality on the variation of K_{OC} in marine sediments. *Toxicol Environ Chem* 77:9-29.

Burgess RM, Serbst JR, Champlin DM, Kuhn A, Ryba SR. 2000b. Toxicity of high molecular weight PAH mixtures in sediment to *Ampelisca abdita* and *Americamysis bahia*. Memorandum to David Mount. November 6, 2002.

Burgess RM, Ahrens MJ, Hickey, CW. 2003. Geochemistry of PAHs in aquatic environments: source, persistence and distribution. In P.E.T. Douben (ed.) *PAHs: An Ecotoxicological Perspective*. John Wiley & Sons Ltd., Sussex, The United Kingdom.

Cairns MA, Nebeker AV. 1982. Toxicity of acenaphthene and isophorone to early life stages of fathead minnows. *Arch Environ Contam Toxicol* 11:703-707.

Call DJ, Brooke LT, Ahmad A, Richter JE. 1983. Toxicity and metabolism studies with EPA priority pollutants and related chemicals in freshwater organisms. Report no. EPA-600/3-83-095. U.S. Environmental Protection Agency, Washington, DC.

Call DJ, Brooke LT, Knuth ML, Poirier SH, Hoglund MD. 1985. Fish sub-chronic toxicity prediction model for industrial organic chemicals that produce narcosis. *Environ Toxicol Chem* 4:335-341.

Call DJ, Brooke LT, Harting SL, Poirer SH, McCauley DJ. 1986. Toxicity of phenanthrene to several freshwater species. Final report to Battelle Memorial Research Institute, Columbus, OH. Center for Lake Superior Environmental Studies, University of Wisconsin -Superior, Superior, Wis. 18 pp. Contact no. F-4114(8834)-411. U.S. Environmental Protection Agency, Washington, DC.

Canton JH, Adema DMM. 1978. Reproducibility of short-term reproduction toxicity experiments with *Daphnia magna* and comparison of the sensitivity of *Daphnia magna* with *Daphnia pulex* and *Daphnia cucullata* in short-term experiments. *Hydrobiol* 59:135-140.

Carls MG, Rice SD and Hose JE. 1999. Sensitivity of fish embryos to weathered crude oil: Part I. low-level exposure during incubation causes malformations, genetic damage, and mortality in larval pacific herring (Clupea pallasi). *Environ Toxicol Chem* 18(3): 481-493.

Chaisuksant, QY, Connell, D. 1997. Internal lethal concentrations of halobenzenes with fish (*Gambusia affinis*). *Ecotoxicol Environ Safety* 37:66-75.

Champlin DM, Poucher SL. 1992a. Acute toxicity of pyrene to saltwater animals. Memorandum to Suzanne Lussier and Dave Hansen, September 15, 1992.

Champlin DM, Poucher SL. 1992b. Chronic toxicity of pyrene to *Americamysis bahia* in a 28-day flow-through test. Memorandum to Suzanne Lussier and Dave Hansen, September 15, 1992.

Chapman GA. 1987. Establishing sediment criteria for chemicals-Regulatory perspectives. In Dickson KL, Maki AW, Brungs WA (eds.), *Fate and Effects of Sediment-Bound Chemicals in Aquatic Systems.* Pergamon Press, Elmsford, NY, pp. 355-377.

Chapman PM, Downie J, Maynard A, Taylor LA. 1996. Coal and deoderizer residues in marine sediments - contaminants or pollutants? *Environ Toxicol Chem* 15:638-642.

Chiou CT, Peters LJ, Freed VH. 1979. A physical concept of soil-water equilibria for nonionic organic compounds. *Science* 206:831-832.

Chiou CT, Porter PE, Schmedding DW. 1983. Partition equilibria of nonionic organic compounds between soil organic matter and water. *Environ Sci Technol* 17:227-231.

CLSES. 1984. Acute toxicities of organic chemicals to fathead minnows (*Pimephales promelas*). Center for Lake Superior Environmental Studies, University of Wisconsin- Superior, Duluth, MN.

CLSES. 1985. Acute toxicities of organic chemicals to fathead minnows (*Pimephales promelas*). Center for Lake Superior Environmental Studies, University of Wisconsin- Superior, Duluth, MN.

CLSES. 1986. Acute toxicities of organic chemicals to fathead minnows (*Pimephales promelas*). Center for Lake Superior Environmental Studies, University of Wisconsin- Superior, Duluth, MN.

CLSES. 1988. Acute toxicities of organic chemicals to fathead minnows (*Pimephales promelas*). Center for Lake Superior Environmental Studies, University of Wisconsin- Superior, Duluth, MN.

CLSES. 1990. Acute toxicities of organic chemicals to fathead minnows (*Pimephales promelas*). Center for Lake Superior Environmental Studies, University of Wisconsin- Superior, Duluth, MN.

Cody TE, Radike M, Warshawsky D. 1984. The phototoxicity of benzo[a]pyrene in the green alga *Selenastrum capricornutum*. *Environ Res* 35:122-132.

Cubbage J, Batts D, Briedenbach S. 1997. Creation and analysis of freshwater sediment quality values in Washington State. Environmental Investigations and Laboratory Services Program, Washington Department of Ecology, Olympia, WA.

Currie et al. 2002. A critical evaluation of interlaboratory data on total, elemental, and isotopic carbon in the carbonaceous particle reference material, NIST SRM 1649a. *J Res Nation Instit Stand Technol* 107:279-298.

Darville RG, Wilhm JL. 1984. The effect of naphthalene on oxygen consumption and hemoglobin concentration in *Chironomus attenuatus* and on oxygen consumption and lifecycle of Tanytarsus dissimilis. *Environ Toxicol Chem* 3:135-41.

Davenport R, Spacie A. 1991. Acute phototoxicity of harbor and tributary sediments from lower Lake Michigan. *J Great Lakes Res* 17:51-56.

Dawson GW, Jennings AL, Drozdowski D, Rider E. 1977. The acute toxicity of 47 industrial chemical to fresh and saltwater fishes. *J Hazardous Materials* 1:303-318.

de Bruijn J, Busser F, Seinen W, Hermens J. 1989. Determination of octanol/water partition coefficients for hydrophobic organic chemicals with the "slow-stirring" method. *Environ Toxicol Chem* 8:499-512.

DeGraeve GM et al., 1980. Effects of naphthalene and benzene on fathead minnows and rainbow trout. Unpublished manuscript.

DeGraeve GM, Elder RG, Woods DC, Bergman HL. 1982. Effects of naphthalene and benzene on fathead minnows and rainbow trout. *Arch Environ Contam Toxicol* 11:487-90.

de Maagd, PG, et al. 1996. Lipid content and time-to-death explain most of the intraspecies variation in lethal body burdens of napthalene and 1,2,4-trichlorobenzene in fathead minnow (*Pimephales promelas*). Chapter 8, Part IV. PhD thesis. Univ. of Ultrech, In *Polycyclic Aromatic Hydrocarbons: Fate and Effects in the Aquatic Environment.* Ultrech, The Netherlands.

de Maagd PGJ, Sinnige TL, Schrap SM, Opperhuizen A, Sijm DTHM. 1998. Sorption coefficients of polycyclic aromatic hydrocarbons for two lake sediments: influences of the bactericide sodium azide. *Environ Toxicol Chem* 17:1899-1907.

DeWitt TH, Swartz RC, Lamberson JO. 1989. Measuring the acute toxicity of estuarine sediments. *Environ Toxicol Chem* 8:1035-1048.

DeWitt TH, Ozretich RJ, Swartz RC, Lamberson JO, Schults DW, Ditsworth GR, Jones JKP, Hoselton L, Smith LM. 1992. The influence of organic matter quality on the toxicity and partitioning of sediment-associated fluoranthene. *Environ Toxicol Chem* 11:197-208.

Diamond S, Mount D. 1998. Evaluating the role of photo-activated PAH toxicity in risk assessment. *SETAC News: Learned Discourses* 18:17-18.

Dill DC. 1980. Toxicity of 1,1-dichloroethylene (vinylidene chloride) to aquatic organisms. Technical report. EPA/600/3-80/057. U.S. Environmental Protection Agency, Washington, DC.

Di Toro DM. 1985. A particle interaction model of reversible organic chemical sorption. *Chemosphere* 14:1503-1538.

Di Toro DM, Zarba CS, Hansen DJ, Berry WJ, Swartz RC, Cowan CE, Pavlou SP, Allen HE, Thomas NA, Paquin PR. 1991. Technical basis for the equilibrium partitioning method for establishing sediment quality criteria. *Environ Toxicol Chem* 11:1541-1583.

Di Toro DM, McGrath JA, Hansen DJ. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. I. Water and tissue. *Environ Toxicol Chem*. 19:1971-1982.

Di Toro DM, JA McGrath. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. II. Mixtures and sediments. *Environ Toxicol Chem.* 19:1983-1991.

Driscoll SK, Harkey GA, Landrum PF. 1997a. Accumulation and toxicokinetics of fluoranthene in sediment bioassays with freshwater amphipods. *Environ Toxicol Chem* 16:742-753.

Driscoll SK, Landrum PF, Tigue E. 1997b. Accumulation and toxicokinetics of fluoranthene in water-only exposures with freshwater amphipods. *Environ Toxicol Chem* 16:754-761.

Driscoll SK, Schaffner LC, Dickhut RM. 1998. Toxicokinetics of fluoranthene to the amphipod, *Leptocheirus plumulosus*, in water-only and sediment exposures. *Mar Environ Res* 45:269-284.

Eastmond DA, Booth GM, Lee ML. 1984. Polycyclic aromatic hydrocarbons: accumulation and elimination of polycyclic aromatic sulfur heterocycles in *Daphnia magna*. *Arch Environ Contam Toxicol* 13:105-111.

Edmisten GE, Bantle JA. 1982. Use of *Xenopus ldevis* larvae in 96-hour, flow-through toxicity tests with naphthalene. *Bull Environ Contam Toxicol* 29:392-9.

Edsall CC. 1991. Acute toxicities to larval rainbow trout of representative compounds detected in Great Lakes fish. *Bull Environ Contam Toxicol* 46:173-8.

EG&G Bionomics. 1978. Acute and chronic toxicity of fluoranthene to mysid shrimp (*Americamysis bahia*). Contract no. 68-01-4646. Preliminary research report.

EG&G Bionomics, 1982. Acute toxicity of selected chemicals to fathead minnow, water flea and mysid shrimp under static and flow-through test conditions. Contract no, 807479-01-0. U.S. Environmental Protection Agency, Washington, DC.

ERCO. 1981. Toxicity testing inter-laboratory comparison early life stage test with fathead minnow. Final report to U.S. EPA, Cincinnati, OH and U.S. EPA, Duluth MN. ERCO/Energy Resources Co., Inc., 185 Alewife Brook Parkway, Cambridge, MA, 47 pp.

Erickson RJ, Ankley GT, DeFoe DL, Kosian PA, Makynen EA. 1999. Additive toxicity of binary mixtures of phototoxic polycyclic aromatic hydrocarbons to the oligochaete *Lumbriculus variegatus*. *Toxicol Appl Pharmacol* 154:97-105

Fabacher DL, Besser JM, Schmitt CJ, Harshbarger JC, Peterman PH and Lebo JA. 1991. Contaminated sediments from tributaries of the Great Lakes: Chemical characterization and carcinogenic effects in medaka (Oryzias latipes). *Arch Environ Contam Toxicol* 20:17-34

Fairey R, Bretz C, Lamerdin S, Hunt J, Anderson B, Tudor S, Wilson C, LaCaro F, Stephenson M, Puckett M, Long E. 1996. Chemistry, toxicity and benthic community conditions in sediments of the San Diego Bay Region. Final Report. California State Water Resources Control Board. Sacremento, CA.

Farrington FW, Goldberg ED, Risebrough RW, Martin JH, Bowen VT. 1983. U.S. "Mussel Watch" 1976-1978: An overview of the trace-metal, DDE, PCB, hydrocarbon, and artificial radionuclide data. *Environ Sci Technol* 17:490-496.

Finger SE, Little EF, Henry MG, Fairchild JF, Boyle TP. 1985. Comparison of laboratory and field assessment of fluorene - part 1: effects of fluorene on the survival, growth, reproduction, and behavior of aquatic organisms in laboratory tests. In *Validation Predict. Lab. Methods Assess. Fate Eff. Contam. Aquat. Ecosyst.*, ASTM Spec. Tech. Publ. 865, American Society of Testing and Materials, Philadelphia, PA., pp. 120-33.

Franks and Lieb, 1990. Mechanisms of general anesthesia. *Environ Health Perspect* 87:199-205.

Gala WT, Giesy JP. 1992. Photo-induced toxicity of anthracene to the green alga, *Selenastrum capricornutum*. *Arch Environ Contam Toxicol* 23:316-23.

Gala WR, Giesy JP. 1994. Flow cytometric determination of the photoinduced toxicity of anthracene to the green alga *Selenastrum capricornutum*. *Environ Toxicol Chem* 13:831-840.

Geiger JG, Buikema AL, Jr. 1981. Oxygen consumption and filtering rate of *Daphnia pulex* after exposure in water-soluble fractions of naphthalene, phenanthrene, No. 2 fuel oil, and coal-tar creosote. *Bull Environ Contam Toxicol* 27:783-789.

Geiger JG, Buikema AL, Jr. 1982. Hydrocarbons depress growth and reproduction of *Daphnia pulex* (Cladocera). *Can J Fish Aquat Sci* 39:830-6.

Geiger DL, Northcott CE, Call DJ, Brooke LT. 1985. Acute toxicities of organic chemicals to fathead minnows (*Pimephales promelas*). Vol. 2. Center for Lake Superior Environmental Studies, University of Wisconsin, Superior WI, 326 pp.

Gelinas Y, Prentice KM, Baldock JA, Hedges JI. 2001. An improved thermal oxidation method for the quantification of soot/graphitic black carbon in sediments and soils. *Environ Sci Technol* 35:3519-3525.

Gendussa AC 1990. Toxicity of chromium and fluoranthene from aqueous and sediment sources to selected freshwater fish. Ph.D. Thesis. University of North Texas, U.M.I., Ann Arbor, MI, 138 pp.

Goddard KA, Schultz JR and Stegeman JJ. 1987. Uptake, Toxicity, and distribution of benzo[a]pyrene and monooxygenase induction in the topminnows *Poeciliopsis monacha* and *Poeciliopsis lucida*. *Drug Metabol Dispos* 15: 449-455.

Gschwend PM, Hites RA. 1981. Fluxes of polycyclic aromatic hydrocarbons to marine and lacustrine sediments in the northeastern United States. *Geochim Cosmo Acta* 45:2359-2367.

Gschwend PM, Chen PH, Hites RA. 1983. On the formation of perylene in recent sediments: kinetic models. *Geochim Cosmo Acta* 47:2115-2119.

Gustafsson O, Gschwend PM. 1997. Soot as a strong partition medium for polycyclic aromatic hydrocarbons in aquatic systems. In Eganhouse, RP (ed.) *Molecular Markers in Environmental Geochemistry: Source Indicators and Process Probes*. American Chemical Society, Washington D.C.

Gustafsson O, Gschwend PM. 1999. Phase distribution of hydrophobic chemicals in the aquatic environment. In Baveye et al (eds.), *Bioavailability of Organic Xenobiotics in the Environment.* Kluwer Academic Publishers, Amsterdam, The Netherlands, pp. 327-348.

Gustafsson O, Haghseta F, Chan C, MacFarlane J, Gschwend PM. 1997. Quantification of the dilute sedimentary soot phase: Implications for PAH speciation and bioavailability. *Environ Sci Tech* 31:203-209.

Gustafsson O, Bucheli TD, Kukulska Z, Andersson M, Largeau C, Rouzaud J-N, Reddy CM, Eglinton TI. 2001. Evaluation of a protocol for the quantification of black carbon in sediments. *Global Biogeochem Cycle* 15:881-890.

Hall L, Maynard E, Kier L. 1989. QSAR investigation of benzene toxicity to fathead minnow using molecular connectivity. *Environ Toxicol Chem* 8:783-788.

Hall LH, Kier LB, Phipps G. 1984. Structure-activity relationships on the toxicity of benzene derivatives: I. An additivity model. *Environ Toxicol Chem* 3:355-365.

Hall TA and Oris JT. 1991. Anthracene reduces reproductive potential and is maternally transferred during long-term exposure in fathead minnows. *Aquat Toxicol* 19: 249-264.

Hannah, JB, Hose JE, Landolt ML, Miller BS, Felton, SP, and Iwaoka WT. 1982. Benzo[a]pyrene- induced morphologic and developmental abnormalities in rainbow trout. *Arch Environ Contam Toxicol* 11: 727-734.

Hansch C, Leo A. 1995. *Exploring QSAR Fundamentals and Applications in Chemistry and Biology.* American Chemical Society, Washington DC.

Hargreaves BR, RL Smith, CQ Thompson, SS Herman. 1982. Toxicity and accumulation of naphthalene in the mysid *Neomysis americana* (Smith) and effects of environmental temperature. In Vernberg, WB (ed.) *Physiol Mech Mar Pollut Toxic, Proc. Symp. Pollut. Mar. Org.*, Academic Press, New York, NY, pp 391-412.

Harkey GA, Driscoll SK, Landrum PF. 1997. Effect of feeding in 30-d bioaccumulation assays using *Hyalella azteca* in fluoranthene-dosed sediment. *Environ Toxicol Chem* 16:762-769.

Hase A, Hites RA. 1976. On the origin of polycyclic aromatic hydrocarbons in recent sediments: biosynthesis by anaerobic bacteria. *Geochim Cosmo Acta* 40: 1141-1143.

Hatch AC, Burton GA. 1998. Effects of photoinduced toxicity of fluoranthene on amphibian embryos and larvae. *Environ Toxicol Chem* 17:1777-1785.

Hatch AC, Burton GA. 1999. Photo-induced toxicity of PAHs to *Hyalella azteca* and *Chironomus tentans*: effects of mixtures and behavior. *Environ Pollut* 106:157-167.

Hawkins WE, Walker WW, Overstreet RM, Lytle TF and Lytle JS. 1988. Dose-related carcinogenic effects of water-borne benzo[a]pyrene on livers of two small fish species. *Ecotoxicol Environ Safety* 16: 219-231.

Hawkins, WE, Walker WW, Overstreet RM, Lytle JS and Lytle TF. 1990. Carcinogenic effects of some polycyclic aromatic hydrocarbons on the Japanese medaka and guppy in waterborne exposures. *Sci Total Environ* 94: 155-167.

Heitmuller PT, Hollister TA, Parrish PR. 1981. Acute toxicity of 54 industrial chemicals to sheepshead minnows (*Cyprinodon variegatus*). *Bull Environ Contam Toxicol* 27:596-404.

Hendricks JD, Meyers TR, Shelton DW, Casteel JL and Bailey GS. 1985. Hepatocarcinogenicity of benzo[a]pyrene to rainbow trout by dietary exposure and intraperitoneal injection. *J. Nat. Cancer Inst.* 74(4): 839-851.

Hermens JLM. 1989. Quantitative structure-activity relationships of environmental pollutants. In Hutzinger O, ed, Handbook of Environmental Chemistry. Vol. 2E-Reactions and processes. Springer Verlag, Berlin, pp 111-162.

Hermens J, Leeuwangh P, Musch A. 1984. Quantitative structure-activity relationships and mixture toxicity studies of chloro- and alkylanilines at an acute lethal toxicity level to the guppy (*Poecilia reticulate*). *Ecotoxicol Environ Safety* 8:388-394.

Hickey CW, Roper DS, Holland PT, Trower TM. 1995. Accumulation of organic contaminants in two sediment-dwelling shellfish with contrasting feeding modes: deposit- (*Macomona liliana*) and filter-feeding (*Austrovenus stutchburyi*). *Arch Environ Contamin Toxicol* 29: 221-231.

Hilal SH, Carreira L, Karickhoff SW. 1994. Estimation of chemical reactivity and parameters and physical properties of organic molecules using SPARC. In: Politzer P, Murry JS, (eds.), *Quantitative Treatments of Solute/Solvent Interactions,* volume 1. Elsevier, Amsterdam, pp. 291-348.

Hites RA, LaFlamme RE, Farrington JW .1977. Sedimentary polycyclic aromatic hydrocarbons: the historical record. *Science* 198: 829-831.

Hites RA, LaFlamme RE, Windsor J.G . 1980. Polycyclic aromatic hydrocarbons in the marine/aquatic sediments: their ubiquity. In *Petroleum in the Marine Environment*. Petrakis L, Weiss FT, (eds.), American Chemical Society, Advances in Chemistry Series. Washington DC., pp 289 - 311.

Hodson PV, Dixon DG, Kaiser K. 1988. Estimating the acute toxicity of water-borne chemicals in trout from measurements of median lethal dose and the octanol-water partition coefficient. *Environ Toxicol Chem* 7:443-454.

Ho, K., L. Patton, J.S. Latimer, R.J. Pruell, M. Pelletier, R. McKinney and S. Jayaraman. 1999. The Chemistry and Toxicity of Sediment Affected by Oil from the North Cape Spilled into Rhode Island Sound. *Mar Pollut Bull* 38:314 - 323.

Hoke R. 1992. Results of the third dieldrin sediment-spiking study. Memorandum to D. Hansen, D. Di Toro and G. Ankley. December 2, 5 pp.

Holcombe GH, Phipps GL, Fiandt JT. 1983. Toxicity of selected priority pollutants to various aquatic organisms. *Ecotoxicol Environ Safety* 7:400-409.

Holcombe GW, Phipps GL, Knuth ML, Felnaber T. 1984. The acute toxicity of selected substituted phenols, benzenes and benzoic acid esters to fathead minnows (*Pimephales promelas*). *Environ Pollut* 35:367-81.

Holcombe G, Phipps G, Sulaiman A, Hoffman A. 1987. Simultaneous multiple species testing: Acute toxicity of 13 chemicals to 12 diverse freshwater amphibian, fish, and invertebrate. *Arch Environ Contam Toxicol* 16:697-710.

Holst LL, Giesy JP. 1989. Chronic effects of the photoenhanced toxicity of anthracene on *Daphnia magna* reproduction. *Environ Toxicol Chem* 8:933-42.

Horne JD, Oblad BR. 1983. Aquatic toxicity studies of six priority pollutants. Contract No. 68-01-6201. Final Report Task II. U.S. Environmental Protection Agency, Washington, DC.

Horne JD, Swirsky MA, Hollister TA, Oblad BR, Kennedy JH. 1983. Aquatic toxicity studies of five pollutants. Report no. 4398, U.S. EPA contract no. 68-01-6201. NUS Corporation, Houston, TX, 93 pp.

Hose JE, Hannah JB, Landolt ML, Miller BS, Felton SP and Iwaoka WT. 1981. Uptake of benzo[a]pyrene by gonadal tissue of flatfish (family pleuronectidae) and its effects on subsequent egg development. *J. Toxicol. Environ. Health.* 7: 991-1000.

Hose JE, Hannah, JB, DiJulio D, Landolt ML, Miller BS, Iwaoka WT and Felton SP. 1982. Effects of benzo[a]pyrene on early development of flatfish. *Arch Environ Contam Toxicol* 11: 167-171.

Hose JE, Hannah JB, Puffer HW and Landolt ML. 1984. Histologic and skeletal abnormalities in benzo[a]pyrene-treated rainbow trout alevins. *Arch Environ Contam Toxicol* 13: 675-684.

Huang X, Dixon DG, Greenberg BM. 1993. Impacts of UV radiation and photomodification on the toxicity of PAHs to the higher plant, *Lemna gibba* (duckweed). *Environ Toxicol Chem* 12:1067-1077.

Huang X-D, McConkey BJ, Babu TS, Greenberg BM. 1997. Mechanisms of photoinduced toxicity of photomodified anthracene to plants: Inhibition of photosynthesis in the aquatic higher plant *Lemna gibba* (Duckweed). *Environ Toxicol Chem* 16:1707-1715.

Hunt GJ, Anderson B, Phillips B, Newman J, Tjeerdema R, Taberski K, Wilson C, Stephenson M, Puckett HM, Fairey R, Oakden J. 1998. Sediment quality and biological effects in San Francisco Bay. Final Technical Report. California State Water Resources Control Board. Sacremento, CA.

Ingersoll CG, Haverland PS, Brunson EL, Canfield TJ, Dwyer FJ, Henke CE, Kemble NE, Mount DR, Fox RG. 1996. Calculation and evaluation of sediment effect concentrations for the amphipod Hyalella azteca and the Midge Chironomus riparius. *J Great Lakes Research* 22:602-623.

Ireland DS, Burton GA, Hess GG. 1996. In situ toxicity evaluations of turbidity and photoinduction of polycyclic aromatic hydrocarbons. *Environ Toxicol Chem* 15:574-581.

Jonker MTO, Smedes F. 2000. Preferential sorption of planar contaminants in sediments from Lake Ketelmeer, The Netherlands. *Environ Sci Technol* 34:1620-1626.

Jonker MTO, Koelmans AA. 2001. Polyoxymethylene solid phase extraction as a partitioning method for hydrophobic organic chemicals in sediment and soot. *Environ Sci Technol* 35:3742-3748.

Jonker MTO, Koelmans AA. 2002a. Sorption of polycyclic aromatic hydrocarbons and polychlorinated biphenyls to soot and soot-like materials in the aqueous environment: mechanistic considerations. *Environ Sci Technol* 36:3725-3734.

Jonker MTO, Koelmans AA. 2002b. Extraction of polycyclic aromatic hydrocarbons from soot and sediment: solvent evaluation and implications for sorption mechanism. *Environ Sci Technol* 36:4107-4113.

Juhnke VI, Ludemann D. 1978. Ergebnisse der untersuchung von 200 chemischen verbindungen auf akute fischtoxizitat mit dem goldorfentest. *Z Wasser und Abivasser- Forschung* 11I:161-164.

Kagan J, Kagan PA, Buhse HE, Jr. 1984. Light-dependent toxicity of "-terthienyl and anthracene to late embryonic stages of *Rana pipiens*. *J Chem Ecol* 10:1115-1122.

Kagan J, Kagan ED, Kagan IS, Kagan PA, Quigley S. 1985. The phototoxicity of non-carcinogenic polycyclic aromatic hydrocarbons in aquatic organisms. *Chemosphere* 14:1829-1834.

Kamler, EWA. 1992. *Early Life History of Fish: an Energetics Approach*. *Fish and Fisheries.* Ser. 4. Chapman and Hall, New York.

Karapanagioti HK, Kleineidam S, Sabatini DA, Grathwohl P, Ligouis B. 2000. Impacts of heterogeneous organic matter on phenanthrene sorption: equilibrium and kinetics studies with aquifer material. *Environ Sci Technol* 34: 406-414.

Karickhoff SW, Brown DS, Scott TA. 1979. Sorption of hydrophobic pollutants on natural sediments. *Water Res* 13:241-248.

Karickhoff SW, McDaniel VK, Melton C, Vellino AN, Nute DE, Carreira LA. 1991. Predicting chemical reactivity by computer. *Environ Toxicol Chem* 10:1405-1416.

Kimball G. 1978. The effects of lesser known metals and one organic to fathead minnows (*Pimephales promelas*) and *Daphnia magna*. Manuscript. Dept. of Entomology, Fisheries and Wildlife, Univ. of Minnesota. Minneapolis, MN, 88 pages.

Knutsen J. 1995. Effects of marine organisms from polycyclic aromatic hydrocarbons (PAH) and other constituents of waste water from aluminum smelters with examples from Norway. *Sci Total Environ* 163:107-122.

Kolok AS, Huckins JN, Petty JD and Oris JT. 1996. The role of water ventilation and sediment ingestion in the uptake of benzo[a]pyrene in gizzard shad (*Dorosoma cepedianum*). *Environ Toxicol Chem* 15(10): 1752-1759.

Konemann H. 1980. Structure-activity relationships and additivity in fish toxicities of environmental pollutants. *Ecotoxicol Environ Safety* 4:415-421.

Konemann H. 1981. Quantitative structure-toxicity relationships in fish toxicity studies. Part 1: Relationship for 50 industrial pollutants. *Toxicology* 19:209-221.

Korn S., Rice S. 1981. Sensitivity to, and accumulation and depuration of, aromatic petroleum components by early life stages of coho salmon (*Oncorhynchus kisutch*). *Rapp P-V Reun, Cons Int Explor Mer* 178:87-92.

Kosian PA, Makynen EA, Monson PD, Mount DR, Spacie A, Mekenyan OG, Ankley GT. 1998. Application of toxicity-based fractionation techniques and structure-activity relationship models for the identification of phototoxic polycyclic aromtaic hydrocarbons in sediment pore water. *Environ Toxicol Chem* 17:1021-1033.

Kreyszig E. 1972. Advanced Engineering Mathematics. J. Wiley and Sons, New York.

Kuhn A, Lussier S. 1987. Results from acute and life-cycle tests with *Americamysis bahia* exposed to phenanthrene. Memorandum to David J. Hansen, U.S. EPA, Narragansett, RI. August 3, 1987. 3 pp.

Kukkonen J, Landrum PF. 1994. Toxicokinetics and toxicity of sediment-associated pyrene to *Lumbriculus variegatus* (Oligochaeta). *Environ Toxicol Chem* 13:1457-1468.

LaFlamme RE, Hites RA. 1978. The global distribution of polycyclic aromatic hydrocarbons in recent sediments. *Geochim Cosmo Acta* 42:289-303.

Lake JL, Norwood C, Dimock C, Bowen R. 1979. Origins of polycyclic aromatic hydrocarbons in estuarine sediments. *Geochim Cosmo Acta* 43:1847-1854.

Lamoureux EM, Brownawell BJ. 1999. Chemical and biological availability of sediment-sorbed hydrophobic organic contaminants. *Environ Toxicol Chem* 18:1733-1741.

Landrum PF, Bartell SM, Giesy JP, Leversee GJ, Bowling JW, Haddock J, LaGory K, Gerould S, Bruno M. 1984a. Fate of anthracene in an artificial stream: a case study. *Ecotoxicol Environ Safety* 8:183-201.

Landrum PF, Giesy JP, Oris JT, Allred PM. 1984b. Photoinduced toxicity of polycyclic aromatic hydrocarbons to aquatic organisms. In Vandermeulen JH and Hrudly S (eds.), *Oil in Freshwater: Chemistry, Biology, Countermeasure Technology,* Pergamon Press, New York, pp 304 - 318.

Landrum PF, Eadie BJ, Faust WR. 1991. Toxicokinetics and toxicity of a mixture of sediment-associated polycyclic aromatic hydrocarbons to the amphipod *Diporeia* sp. *Environ Toxicol Chem* 10:35-46.

Landrum PF, Dupuis WS, Kukkonen J. 1994. Toxicokinetics and toxicity of sediment associated pyrene and phenanthrene in *Diporeia* spp.: Examination of equilibrium partitioning theory and residue-based effects for assessing hazard. *Environ Toxicol Chem* 13:1769-1780.

Lane WF, Loehr RC. 1992. Estimating the equilibrium aqueous concentrations of polynuclear aromatic hydrocarbons in complex mixtures. *Environ Sci Technol* 26:983-990.

Larsson P. 1985. Contaminated sediments of lakes and oceans act as sources of chlorinated hydrocarbons for release to water and atmosphere. *Nature* 317:347-349.

LeBlanc GA. 1980a. Acute toxicity of priority pollutants to water flea (*Daphnia magna*). *Bull Environ Contam Toxicol* 24:684-691.

LeBlanc GA. 1980b. Acute toxicity of fluoranthene to various marine organisms. Personal communication.

Lemke AE 1984. Inter-laboratory comparison of continuous flow, early life stage testing with fathead minnows. EPA-600/3-84-005 or PB84-129493. National Technical Information Service, Springfield, VA, 26 pp.

Lemke AE, Anderson RL. 1984. Insect interlaboratory toxicity test comparison study for the chironomid (*Paratanytarsus* sp.) procedure. EPA-600/3-84-054 or P1384-180025. National Technical Information Service. Springfield, VA, 15 pp.

Lemke AE, Durhan E, Felhaber T. 1983. Evaluation of a fathead minnow *Pimephales promelas* embryo-larval test benchmark using acenaphthene and isophorone. EPA-600/3-83-062 or P1383-243436. National Technical Information Service, Springfield, VA, 26 pp.

Leo A, Hansch C, Elkins D. 1971. Partition coefficients and their uses. *Chem. Rev.* 71:525-553.

Leo AJ. 1972. Relationships between partitioning solvent systems. In Biological Correlations - The Hansch Approach Advances in Chemistry Series 114, American Chemical Society, Washington, DC, pp 51-60.

Long ER, LG Morgan. 1990. The potential for biological effects of sediment-sorbed contaminants tested in the National Status and Trends Program. NOAA Tech. Memo. NOS OMA 52, National Oceanic and Atmospheric Agency, Rockville, MD, 175 pp.

Long ER, Mac Donald DD, Smith SL, Calder FD. 1995. Incidence of adverse effects within ranges of chemical concentrations in marine and estuarine sediments. *Environ Manag* 19:81-97.

Lotufo GR, Fleeger JW. 1996. Toxicity of sediment-associated pyrene and phenanthrene to *Limnodrilus hoffmeisteri* (Oligochaeta: Tubificidae). *Environ Toxicol Chem* 15:1508-1516.

Luthy RG, Dzombak DA, Peters CA, Roy SB, Ramaswami A, Nakles DV, Nott BR. 1994. Remediating tar-contaminated soils at manufactured gas plant sites. *Environ Sci Technol* 28:266A-276A.

MacDonald DD, Carr RS, Calder, Long ER, Ingersoll CG. 1996. Development and evaluation of sediment quality guidelines for Florida coastal waters. *Ecotoxicol* 5:253-278.

MacDonald DD, Ingersoll CG, Berger TA. 2000. Development and evaluation of consensus-based sediment quality guidelines for freshwater ecosystems. *Archives Environ Contam Toxicol.* 39:20-31.

Mackay D, Shiu WY, Ma KC. 1992. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals. Vol II Polynuclear Aromatic Hydrocarbons,Polychlorinated Dioxins and Dibenzofurans.* Lewis Publishers, Chelsea, MI.

Mahjoub B, Jayr E, Bayard R and Gourdon R (2000) Phase partition of organic pollutants between coal tar and water under variable experimental conditions. *Wat Res* 34:3551-3560.

Malins DC, McCain BB, Myers MS, Brown DW, Krahn MM, Roubal WT, Schiewe MH, Landahl JT, Chan SL. 1987. Field and laboratory studies of the etiology of liver neoplasms in marine fish from Puget Sound. *Environ Health Persp.* 71:5-16.

Marine Bioassay Laboratories. 1981. Flow-through early-life stage toxicity tests with fathead minnows (*Pimephales promelas*). Final report to United States EPA, Duluth, MN. Marine Bioassay Laboratories, 1234 Highway One, Watsonville, CA, 71 pp.

Maruya KA, Risebrough RW, Horne AJ. 1996. Partitioning of polynuclear aromatic hydrocarbons between sediments from San Francisco Bay and their porewaters. *Environ Sci Technol* 30:2942-2947.

Maruya KA, Risenbrough RW, Horne AJ. 1997. The bioaccumulation of polynuclear aromatic hydrocarbons by benthic invertebrates in an intertidal marsh. *Environ Toxicol Chem* 16:1087-1097.

Mattson VR, Arthur JW, Walbridge CT. 1976. Acute toxicity of selected organic compounds to fathead minnows. EPA-600/3-76-097, United States EPA, Office of Research and Development, 13 pp.

May WE. 1980. The solubility behavior of polycyclic aromatic hydrocarbons in aqueous systems. In *Petroleum in the Marine Environment.* Petrakis L, Weiss FT (eds.), American Chemical Society, Advances in Chemistry Series. Washington, DC, pp 143-192.

McCarty LS, Mackay D, Smith AD, Ozburn GW, Dixon DG. 1991. Interpreting aquatic toxicity QSARs: The significance of toxicant body residues at the pharmacologic end point. *In QSAR In Environmental Toxicology.* IV. Elsevier, Amsterdam.

McCloskey JT, Oris JT. 1991. Effect of water temperature and dissolved oxygen concentration on the photo-induced toxicity of anthracene to juvenile bluegill sunfish (*Lepomis macrochirus*). *Aquat Toxicol* 21:145-156.

McConkey BJ, Duxbury CL, Dixon DG, Greenberg BM. 1997. Toxicity of a PAH photooxidation product to the bacteria Photobacterium phosphoreum and the duckweed *Lemna gibba*: Effects of phenethrene and its primary photoproduct, phenanthrenequinone. *Environ Toxicol Chem* 16:892-899.

McDonald BG, Chapman, PM. 2002. PAH phototoxicity-an ecologically irrelevant phenomenon? *Marine Pollut Bull* 44:1321-1326.

McElroy A, Leitch K, Fay A. 2000. A survey of in vivo benz[*a*]pyrene metabolism in small benthic marine invertebrates. *Mar Environ Res* 50:33-38.

McGroddy SE, Farrington JW. 1995. Sediment porewater partitioning of polycyclic aromatic

hydrocarbons in three cores from Boston Harbor, Massachusetts. *Environ Sci Technol* 29:1542-1550.

McGroddy SE, Farrington JW, Gschwend PM. 1996. Comparison of the in situ and desorption sediment-water partitioning of polycyclic aromatic hydrocarbons and polychlorinated biphenyls. *Environ Sci Technol* 30:172-177.

Meador JP, Casillas E, Sloan CA, Varanasi U .1995. Comparative bioaccumulation of polycyclic aromatic hydrocarbons from sediment by two infaunal invertebrates. *Mar Ecol Prog Ser* 123:107-124.

Means JC. 1998. Compound-specific gas chromatographic/mass spectrometric analysis of alkylated and parent polycyclic aromatic hydrocarbons in waters, sediments, and aquatic organisms. J. *AOAC International* 81:657-672.

Means JC, Wood SG, Hassett JJ, Banwart WL. 1980. Sorption of polynuclear aromatic hydrocarbons by sediments and soils. *Environ Sci Technol* 14:1524-1528.

Mekenyan OG, Ankley GT, Veith GD, Call DJ. 1994a. QSAR estimates of excited states and photoinduced acute toxicity of polycyclic aromatic hydrocarbons. *SAR QSAR Environ Res* 2:237-247.

Mekenyan OG, Ankley GT, Veith GD, Call DJ. 1994b. QSARs for photoinduced toxicity: 1. Acute lethality of polycyclic aromatic hydrocarbons. *Chemosphere* 28:567-582.

Metcalfe CD, Cairns VW and Fitzsimons JD. 1988. Experimental induction of liver tumors in rainbow trout (*Salmo gairdneri*) by contaminated sediment from Hamilton Harbour, Ontario. *Can J Fish Aquat Sci* 45: 2161-2167.

Meyers PA, Ishiwatari R .1993. Lacustrine organic geochemistry - an overview of indicators of organic matter sources and diagenesis in lake sediments. *Org Geochem* 20:867-900.

Millemann RE, Birge WJ, Black JA, Cushman RM, Daniels KL, Franco PJ, Giddings JM, McCarthy JF, Stewart AJ. 1984. Comparative acute toxicity to aquatic organisms of components of coal-derived synthetic fuels. *Trans Am Fish Soc* 113:74-85.

Monson PD, Ankley GT, Kosian PA. 1995. Phototoxic response of *Lumbriculus variegatus* to sediments contaminated by polycyclic aromatic hydrocarbons. *Environ Toxicol Chem* 14:891-894.

Monson PD, Call DJ, Cox DA, Liber K, Ankley GT. 1999. Photoinduced toxicity of fluoranthene to northern leopard frogs (*Rana pipiens*). *Environ Toxicol Chem* 18:308-312.

Mortimer MR, Connell DW. 1994. Critical internal and aqueous lethal concentrations of chlorobenzenes with the crab *Portunus pelagicus* (1). Ecotoxicol Environ Safety 28:298-312.

Mount DR, Ingersoll CG, McGrath JA. 2003. Approaches for developing sediment quality guidelines for PAHs. In PET Douben (ed.) *PAHs: An Ecotoxicological Perspective*. John Wiley & Sons Ltd. Sussex, The United Kingdom.

Munkittrick KR, Power EA, Sergy GA. 1991. The relative sensitivity of micro-tox, daphnid, rainbow trout, and fathead minnow acute leth ality tests. *Environ Toxicol Water Quality* 6:35-62.

Myers MS, Landahl JT, Krahn MM, Johnson LL, McBain BB. 1990. Overview of studies on liver carcinogenesis in English sole from Puget Sound: evidene for a xenobiotic chemical etiology I: pathology and epizootiology. *Sci Total Environ* 94:33-50.

Naes K, Knutzen J, Berglind L. 1995. Occurence of PAH in marine organisms and sediments from smelter discharge in Norway. *Sci Total Environ* 163:93-106.

Naes K, Oug E. 1997. Multivariate approach to distribution patterns and fate of polycyclic aromatic hydrocarbons in sediments from smelter-affected Norwegian fjords and coastal waters. *Environ Sci Technol* 31:1253-1258.

Naes K, Oug E. 1998. The distribution and environmental relationships of polycyclic aromatic hydrocarbons (PAHs) in sediments from Norwegian smelter-affected fjords. *Chemosphere* 36:561 - 576.

Naes K, Axelman J, Naf C, Broman D. 1998. Role of soot carbon and other carbon matrices in the distribution of PAHs among particles, DOC, and the dissolved phase in the effluent and recipient waters of an aluminum reduction plant. *Environ Sci Technol* 32:1786-1792.

Naes K, Hylland K, Oug E, Forlin L, Ericson G. 1999. Accumulation and effects of aluminium smelter-generated polycyclic aromatic hydrocarbons on soft-bottom invertebrates and fish. *Environ Toxicol Chem* 18: 2205-2216.

National Research Council. 1985. *Oil in the Sea: Inputs, Fates, and Effects*. National Academy Press, Washington, DC, 601 pp.

Nebeker AV, Schuytema GS, Griffis WL, Barbitta JA, Carey LA. 1989. Effect of sediment organic carbon on survival of *Hyalella azteca* exposed to DDT and endrin. *Environ Toxicol Chem* 8:705-718.

Neff JM .1979. *Polycyclic Aromatic Hydrocarbons in the Aquatic Environment.* Applied Science Publishers, Ltd., London, 262 pp.

Neff JM and Anderson JW. 1975. Accumulation, release, and distribution of benzo[a]pyrene-C^{14} in the clam *Rangia cuneata*. 1975 Conference on Prevention and Control of Oil Pollution proceedings. March 25-27, 1975. San Francisco, California.

Newsted JL, Giesy JP. 1987. Predictive models for photoinduced acute toxicity of polycyclic aromatic hydrocarbons to *Daphnia magna*, Strauss (Cladocera, Crustacea). *Environ Toxicol Chem* 6:445-461.

Nikkilä A, Penttinen S, Kukkonen JVK. 1999. UV-B-induced acute toxicity of pyrene to the waterflea *Daphnia magna* in natural freshwaters. *Ecotoxicol Environ Safe* 44:271-279.

NOAA. 1991. National Status and Trends Program - Second summary of data on chemical contaminants in sediments from the National Status and Trends Program. NOAA Technical Memorandum NOS OMA 59. NOAA Office of Oceanography and Marine Assessment, Rockville, MD, 29 pp + appendices.

NOAA. 1998. Sampling and analytical methods of the National Status and Trends Program Mussel Watch Project: 1993-1996 update. Lauenstein GG and Cantillo AY (eds), NOAA Technical Memorandum NOS ORCA 130. Silver Spring, Maryland. p. 233.

Noreen EW. 1989. *Computer Intensive Methods for Testing Hypotheses: An Introduction.* John Wiley and Sons Inc., New York, N.Y.

Northwestern Aquatic Sciences, Inc. 1982. Round robin testing of the midge (*Tanytarsus*): Acute and chronic toxicity tests of 2,4,6-trichlorophenol and acenaphthene. Contract No. 68-03-3081. Report to United States EPA, ERL-Duluth, MN, Northwestern Aquatic Sciences, Inc., Newport, OR, 66 pp.

Oris JT, Giesy JP, Jr. 1985. The photoenhanced toxicity of anthracene to juvenile sunfish (*Lepomis* spp.). *Aquat Toxicol* 6:133-46.

Oris JT, Giesy JP, Jr. 1986. Photoinduced toxicity of anthracene to juvenile bluegill sunfish (*Lepomis macrochirus* Rafinesque): Photoperiod effects and predictive hazard evaluation. *Environ Toxicol Chem* 5:761-768.

Oris JT, Giesy JP, Jr. 1987. The photo-induced toxicity of polycyclic aromatic hydrocarbons to larvae of the fathead minnow (*Pimephales promelas*). *Chemosphere* 16:1395-404.

Oris JT, Giesy JP, Allred PM, Grant DF, Landrum PF. 1984. Photoinduced toxicity of anthracene in aquatic organisms: an environmental perspective. In Veziroglu TN (ed.) *The Biosphere: Problems and Solutions*, Elsevier Science Publishers B.V., Amsterdam, The Netherlands, pp 639 - 658.

Oris JT, Hall AT, Tylka JD. 1990. Humic acids reduce the photo-induced toxicity of anthracene to fish and Daphnia. *Environ Toxicol Chem* 9:575-583.

Oris JT, Winner RW, Moore MV. 1991. A four-day survival and reproduction toxicity test for *Ceriodaphilia dubia*. *Environ Toxicol Chem* 10:217-224.

Ostrander GK, Landolt ML and Kocan RM. 1989. Whole life history studies of coho salmon (*Oncorhynchus kisutch*) following embryonic exposure to benzo[a]pyrene. *Aquatic Toxicol* 15: 109-126.

Ostrander GK, Landolt ML and Kocan RM. 1988. The ontogeny of coho salmon (*Oncorhynchus kisutch*) behavior following embryonic exposure to benzo[a]pyrene. *Aquat Toxicol* 13: 325-346.

Ott FS, Harris RP, O'Hara SCM. 1978. Acute and sublethal toxicity of naphthalene and three methylated derivatives to the estuarine copepod, *Eurytemora affinis*. *Mar Environ Res* l:49-58.

Oug E, Naes K, Rygg B. 1998. Relationship between soft bottom macrofauna and polycyclic aromatic hydrocarbons (PAH) from smelter discharge in Norwegian fjords and coastal waters. *Mar Ecol Prog Ser* 173:39 - 52.

Owen, B.B. and S.R. Brinkley. 1941. Calculation of the effect of pressure upon ionic equilibrium in pure water and in salt solutions. *Chem Rev* 29:461-472.

Ozretich RJ, Swartz RC, Lamberson JO, Ferraro SP. 1997. An extension of the ∑PAH model to alkylated and other polynuclear aromatic hydrocarbons. Poster presentation at the Annual Meeting of the Society of Environmental Toxicology and Chemistry, San Francisco, CA.

Ozretich RJ, Ferraro SP, Lamberson JO, Cole FA. 2000. A test of the Σ polycyclic aromatic hydrocarbon model at a creosote-contaminated site in Elliott Bay, Washington, USA. *Environ Toxicol Chem* 19:2378-2389.

Page DS, Boehm PD, Stubblefield WA, Parker KR, Gilfillan ES, Neff JM and Maki AW. 2002. Hydrocarbon composition and toxicity of sediments following the Exon Valdeze oil spill in Prince William Sound, Alaska, USA. *Environ Toxicol Chem*. 21:1438-1450.

Paine MD, Chapman PM, Allard PJ, Murdoch MH, Minifie D. 1996. Limited bioavailability of sediment PAH near an aluminum smelter: Contamination does no equal effects. *Environ Toxicol Chem* 15:2003-2018.

Passino DRM, Smith SB. 1987. Acute bioassays and hazard evaluation of representative contaminants detected in Great Lakes fish. *Environ Toxicol Chem* 6:901-907.

Patterson AM, Capell LT, Walker DF. 1960. *The Ring Index, A List of Ring Systems Used in Organic Chemistry* (2nd Edition). American Chemical Society, Washington, DC, 1425 pp.1980

Pelletier MC, Burgess RM, Ho KT, Kuhn A, McKinney RA, Ryba SA. 1997. Phototoxicity of individual PAHs and petroleum to marine invertebrate larvae and juveniles. *Environ Toxicol Chem* 16:2190-2199.

Pelletier MC, Burgess RM, Cantwell MG, Serbst JR, Ho KT, Ryba SA. 2000. Importance of maternal transfer of the photo-reactive polycyclic aromatic hydrocarbon fluoranthene from benthic adult bivalves to their pelagic larvae. *Environ Toxicol Chem*. 19:2691-2698.

Pelletier MC, Burgess RM, Cantwell MG, May AJ, Serbst JR, Ho KT. Resistance of intertidal and subtidal organisms to photo-enhanced PAH toxicity. (In preparation).

Pickard GL, Emery WJ. 1982. *Descriptive Physical Oceanography* (4th Edition). Pergamon Press, Oxford, U.K.

Pickering QH, Henderson C. 1966. Acute toxicity of some important petrochemicals to fish. *J Water Pollut Control Fed* 38:1419-1429.

Prahl FG, Carpenter R. 1983. Polynuclear aromatic hydrocarbon (PAH) - phase association in Washington coastal sediment. *Geochim Cosmo Acta* 47:1013-1023.

PTI Environmental Services. 1991. Pollutants of concern in Puget Sound. EPA 910/9-91-003. United States EPA, Region 10, Seattle, WA.

Randall TL, Knopp PV. 1980. Detoxification of specific organic substances by wet oxidation. *J Water Pollut Control Fed* 52:2117-2130.

Readman JW, Mantoura RFC, Rhead MM. 1984. The physico-chemical speciation of polycyclic aromatic hydrocarbons (PAH) in aquatic systems. *Fresenius Z Anal Chem* 319:126-131.

Readman JW, Fowler SW, Villeneuve J-P, Cattini C, Oregioni B, Mee LD .1992. Oil and combustion-product contamination of the Gulf marine environment following the war. *Nature* 358:662-665.

Ren L, Huang XD, McConkey BJ, Dixon DG, Greenberg BM. 1994. Photoinduced toxicity of three polycyclic aromatic hydrocarbons (fluoranthene, pyrene and naphthalene) to the duckweed *Lemma gibba*. *Ecotoxicol Environ Safety* 28:160-171.

Rice SD, Thomas RE. 1989. Effect of pre-treatment exposures of toluene or naphthalene on the tolerance of pink salmon (*Oncorhynchus gorbuscha*) and kelp shrimp (*Eualis suckleyi*). *Comp Biochem Physiol* 94C:289-293.

Rogerson A, Sliiu W, Huang G, Mackay D, Berger J. 1983. Determination and interpretation of hydrocarbon toxicity to ciliate protozoan. *Aquat Toxicol* 3:215- 228.

Rossi SS, Neff JM. 1978. Toxicity of polynuclear aromatic hydrocarbons to the polychaete *Neanthes arenaceodentata*. *Mar Pollut Bull* 9:220-223.

Salomons W, de Rooij NM, Kerdijk H, Bril J. 1987. Sediments as sources of contaminants? *Hydrobiol* 149:13-30.

Schultz ME and Schultz RJ. 1982. Induction of hepatic tumors with 7,12-dimethylbenz[a]anthracene in two species of viviparous fishes (genus *Poepciliopsis*). *Environ Research* 27: 337-351.

Schuytema GS, Nebeker AV, Griffis WL, Miller CE. 1989. Effect of freezing on toxicity of sediments contaminated with DDT and endrin. *Environ Toxicol Chem* 8:883-891.

Schwarzenbach RP, Gschwend PM, and Imboden DM. 1993. *Environmental Organic Chemistry,* Wiley Interscience. John Wiley & Sons Inc., New York.

Setschenow JZ. 1889. Uber di Konstitution der Salzlosungen aur Grund ihres Verhaltens zu Kohlensaure. *Z. Physik. Chem* 4:117

Sibley PK, Ankley GT, Mount DR. 1997. Comparison of sensitivity of several aquatic invertebrates to UV light following exposure to fluoranthene-spiked sediment. Society of Environmental Toxicology and Chemistry, 18th Annual Meeting, San Francisco, CA.

Sijm D, Schipper M, Opperhuizen A. 1993. Toxicokinetics of halogenated benzenes in fish: Lethal body burden as a toxicological end point. *Environ Sci Technol* 12:1117-1127.

Silliman JE, Meyers PA, Eadie BJ .1998. Perylene: an indicator of alteration processes or precursor materials? *Org Geochem* 29: 1737-1744.

Site AD. 2001. Factors affecting sorption of organic compounds in natural sorbent/water systems and sorption coefficients for selected pollutants. A review. *J Phys Chem Ref Data* 30:187-439.

Slooff NV, Baerselman R. 1980. Comparison of the usefulness of the Mexican axolotl (*Ambystoma mexicanum*) and the clawed toad (Xellopus laevis) in toxicological bioassays. *Bull Environ Contam Toxicol* 24:439-443.

Slooff W, Canton JH, Hermens JLM. 1983. Comparison of the susceptibility of 22 freshwater species to 15 chemical compounds. I. (sub)acute toxicity tests. *Aquat Toxicol* 4:113-128.

Smith AD, Bharath A, Mallard C, Orr D, Smith K, Sutton JA, Vukmanich J, McCarty LS, Ozbum GW. 1991. The acute and chronic toxicity of ten chlorinated organic compounds to the American flagfish (*Jordanella floridae*). *Arch Environ Contam Toxicol* 20:94-102.

Smith SB, Savino JF, Blouin MA. 1988. Acute toxicity to *Daphnia pulex* of six classes of chemical compounds potentially hazardous to great lakes aquatic biota. *J Great Lakes Res* 14: 394-404.

Socha SB, Carpenter R. 1987. Factors affecting pore water hydrocarbon concentrations in Puget Sound sediments. *Geochim Cosmo Acta* 51:1273-1284.

Song J, Peng P, Huang W. 2002. Black carbon and kerogen in soils and sediments. 1. quantification and characterization. *Environ Sci Technol* 36:3960-3967.

Spehar RL, Poucher S, Brooke LT, Hansen DJ, Champlin D, Cox DA. 1999. Comparative toxicity of fluoranthene to freshwater and saltwater species under fluorescent and ultraviolet light. *Arch Environ Contam Toxicol.* 37:496-502.

Spehar RL, Mount DR, Lukasewycz MT, Leonard EN, Burgess RM, Serbst JR, Heinis LJ, Berry WJ, Mattson VR, Burkhard LP, Kuhn A. Response of freshwater and saltwater invertebrates to a mixture of high K_{OW} PAHs in sediment. (*In preparation*)

Stephan CE, Mount DI, Hansen DJ, Gentile JH, Chapman GA, Brungs WA. 1985. Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses. PB85-227049. National Technical Information Service, Springfield, VA, 98 pp.

Suedel BC, Rodgers JH, Clifford PA. 1993. Bioavailability of fluoranthene in freshwater sediment toxicity tests. *Environ Toxicol Chem* 12:155-165.

Suedel BG, Rodgers JH, Jr. 1996. Toxicity of fluoranthene to *Daphnia magna*, *Hyalella azteca*, *Chironomus tentans*, and *Stylaria lacustris* in water-only and whole sediment exposures. *Bull Environ Contam Toxicol* 57:132-138.

Suess MJ . 1976. The environmental load and cycle of polycyclic aromatic hydrocarbons. *Sci Tot Environ* 6: 239-250.

Suter, S.W. II, and Mabrey, J.M. 1994. Toxicological benchmarks for screening potential contaminants of concern for effects on aquatic biota: 1994 revision. ES/ER/TM-96/RI. Oak Ridge National Laboratory. Environmental Sciences Division, Oak Ridge, TN.

Swartz RC. 1991a. Acenaphthene and phenanthrene files. Memorandum to David J. Hansen, June 26, 1991. 160 pp.

Swartz RC. 1991b. Fluoranthene experimental design: Final. Unpublished manuscript. 10pp. December 31, 1991.

Swartz RC. 1999. Consensus sediment quality guidelines forpolycyclic aromatic hydrocarbon mixtures. *Environ Toxicol Chem* 18:280-287.

Swartz RC, Kemp PF, Schults DW, Lamberson JO. 1988. Effects of mixtures of sediment contaminants on the marine infaunal amphipod, *Rhepoxynius abronius*. *Environ Toxicol Chem* 7:1013-1020.

Swartz RC, Schults DW, DeWitt TH, Ditsworth GR, Lamberson JO. 1990. Toxicity of fluoranthene in

sediment to marine amphipods: A test of the equilibrium partitioning approach to sediment quality criteria. *Environ Toxicol Chem* 9:1071-1080.

Swartz RC, Schults DW, Ozretich RJ, Lamberson JO, Cole FA, DeWitt TH, Redmond MS, Ferraro SP. 1995. ΣPAH: A model to predict the toxicity of field-collected marine sediment contaminated with polynuclear aromatic hydrocarbons. *Environ Toxicol Chem* 14:1977-1987.

Swartz RC, Ferraro SP, Lamberson JO, Cole FA, Ozretich RJ, Boese BL, Schults DW, Behrenfeld M, Ankley GT. 1997. Photoactivation and toxicity of mixtures of PAH compounds in marine sediment. *Environ Toxicol Chem* 16:2151-2157.

Talley JW, Ghosh U, Tucker SG, Furey JS, Luthy RG. 2002. Particle-scale understanding of the bioavailability of PAHs in sediment. *Environ Sci Technol* 36:477-483.

Tatem HE. 1977. Accumulation of naphthalenes by grass shrimp: effects on respiration, hatching and larval growth. In Wolfe OA (ed.), *Fate and Effects of Petroleum Hydrocarbons in Marine Organisms and Ecosystems*, Pages 201-209. Pergamon Press, New York, NY.

Tatem HE, Cox BA, Anderson JW. 1978. The toxicity of oils and petroleum hydrocarbons to estuarine crustaceans. *Estuar Coast Mar Sci* 8:365- 373.

Thursby GB. 1991a. Review of freshwater round-robin data for acenaphthene. Memorandum to David J. Hansen, September 18, 1991. 2 pp.

Thursby GB. 1991b. Re-analyses of data from Horne et al., 1983. Memorandum to David J. Hansen, September 24, 1991, 2pp.

Thursby GB, Berry WJ, Champlin D. 1989a. Acute toxicity of acenaphthene to saltwater animals. Memorandum to David J. Hansen, February 7, 1989, 9 pp.

Thursby GB, Berry WJ, Champlin D. 1989b. Flow-through acute and chronic tests with acenaphthene using *Americamysis bahia*. Memorandum to David J. Hansen, September 19, 1989, 5 pp.

Thurston R, Giffoil T, Meyn E, Zajdel R, Aoki T, Veith G. 1985. Comparative toxicity of ten organic chemicals to ten common aquatic species. *Water Res* 9:1145-1155.

Trucco RG, Engelhardt FR, Stacey B. 1983. Toxicity, accumulation and clearance of aromatic hydrocarbons in *Daphnia pulex*. *Environ Pollut* (Series A) 31:191-202.

United States Environmental Protection Agency. 1978. In-depth studies on health and environmental impacts of selected water pollutants. Contract no. 68-01-4646. (Table of data available from Charles E. Stephan U.S. EPA, Duluth, MN) Duluth, MN.

United States Environmental Protection Agency. 1980. Ambient water quality criteria for naphthalene. EPA 440/5-80-059. Office of Water Regulations and Standards, Washington, DC.

United States Environmental Protection Agency. 1989. Handbook: Water Quality Control Information System, STORET. Washington, DC.

United States Environmental Protection Agency. 1992. Sediment classification methods compendium. EPA 823-R-92-006, Office of Water, Washington, DC. 280p.

United States Environmental Protection Agency. 1993a. Sediment quality criteria for the protection of benthic organisms: Acenaphthene. EPA 822-R-93-013. Office of Water, Washington, D.C.

United States Environmental Protection Agency. 1993b. Sediment quality criteria for the protection of benthic organisms: Fluoranthene. EPA 822-R-93-012. Office of Water, Washington, DC.

United States Environmental Protection Agency. 1993c. Sediment quality criteria for the protection of benthic organisms: Phenanthrene. EPA 822-R-93-014. Office of Water, Washington, DC.

United States Environmental Protection Agency. 1994. Methods for assessing the toxicity of sediment-associated contaminants with estuarine and marine amphipods. EPA/600/R-94/025. Office of Research and Development, Washington DC.

United States Environmental Protection Agency. 1996a. EMAP - Estuaries Virginian Province Data 1990-1993. Available from: EMAP Home Page WWW site. http://www. epa.gov/emap. Accessed Nov. 1998.

United States Environmental Protection Agency. 1996b. EMAP - Estuaries Louisianian Province Data 1991-1993. Available from: EMAP Home Page WWW site. http://www.epa.gov/emap. Accessed Nov. 1998.

United States Environmental Protection Agency. 1997a. The incidence and severity of sediment contamination in surface waters of the United States. U.S. EPA, Office of Science and Technology. Vol. 1: National sediment quality survey (EPA 823-R-97-006), Washington, DC.

United States Environmental Protection Agency. 1997b. The incidence and severity of sediment contamination in surface waters of the United States. Vol. 2: Data summaries for areas of probable concern. EPA 823-R-97-007. U.S. EPA, Office of Science and Technology, Washington, DC.

United States Environmental Protection Agency. 1997c. The incidence and severity of sediment contamination in surface waters of the United States. Vol. 3: National sediment contaminant point source inventory EPA 823-R-97-008. U.S. EPA, Office of Science and Technology, Washington, DC.

United States Environmental Protection Agency. 1998. EMAP-Estuaries Carolinian Province Data 1994-1997. Available from EMAP Home Page WWW site. Http://www.epa.gov/emap.

United States Environmental Protection Agency. 2003a. Technical basis for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: Nonionic organics. EPA-600-R-02-014. Office of Research and Development. Washington, DC. (draft)

United States Environmental Protection Agency. 2003b. Procedures for the derivation of site-specific equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: Nonionic organics. EPA-600-R-02-012. Office of Research and Development. Washington, DC. (draft)

United States Environmental Protection Agency. 2003c. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: Endrin. EPA-600-R-02-009. Office of Research and Development, Washington, DC.

United States Environmental Protection Agency. 2003d. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: Dieldrin. EPA-600-R-02-010. Office of Research and Development, Washington, DC.

United States Environmental Protection Agency. 2003e. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: Metals mixtures (cadmium, copper, lead, nickel, silver and zinc). EPA-600-R-02-011. Office of Research and Development, Washington, DC. (draft)

United States Environmental Protection Agency. 2003f. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: Nonionics compendium. EPA-600-R-02-016. Office of Research and Development. Washington, DC. (draft)

van Leeuwen CJ, Van Der Zandt PTJ, Aidenberg T, Verhaar HJM, Hermens JLM. 1992. Application of QSARS, extrapolation and equilibrium partitioning in aquatic effects assessment. 1. Narcotic industrial pollutants. *Environ Toxicol Chem* 11:267-282.

van Wezel AP, de Vries DAM, Kostense S, Sijm DTHM, Opperhuizen A. 1995. Intraspecies variation in lethal body burdens of narcotic compounds. *Aquat Toxicol* 33:325-325.

van Wezel AP, de Vries DAM, Sijm DTHM, Opperhuizen A. 1996. Use of lethal body burden in the evaluation of mixture toxicity. *Ecotoxicol Environ Safety* 35:236-241.

Varanasi U, Reichert WL, Stein SE, Brown DW, Sanborn HR. 1985. Bioavailability and transformation of aromatic hydrocarbons in benthic organisms exposed to sediments from an urban estuary. *Environ Sci Technol* 19:836-841.

Veith GD, Call DJ, Brooke LT. 1983. Structure-toxicity relationships for the fathead minnow, Pimephales promelas: Narcotic industrial chemicals. *Can J Fish Aquat Sci* 40:743-748.

Veith GD, Mekenyan OG, Ankley GT, Call DJ. 1995a. QSAR evaluation of “-terthenial phototoxicity. *Environ Sci Technol* 29:1267-1272.

Veith GD, Mekenyan OG, Ankley GT, Call DJ. 1995b. A QSAR analysis of substituent effects on the photoinduced acute toxicity of PAHs. *Chemosphere* 30:2129-2142.

Venkatesan MI . 1988. Occurrence and possible sources of perylene in marine sediments - a review. *Mar Chem* 25: 1-27.

Verardo DJ. 1997. Charcoal analysis in marine sediments. *Limnol Oceanogr* 42:192-197.

Verhaar HJM, van Leeuwen CJ, Hermens JLM. 1992. Classifying environmental pollutants. 1. Structure-activity relationships for prediction of aquatic toxicity. *Chemosphere* 25:471-491.

Wakeham SG, Schaffner C, Giger W. 1980. Polycyclic aromatic hydrocarbons in Recent lake sediments - I. compounds having anthropogenic origins. *Geochim Cosmo Acta* 44: 403-413.

Wallen IE, Greer WC, Lasater R. 1957. Toxicity to *Gambusia affinis* of certain pure chemicals in turbid waters. *Stream Pollut* 29:695-711.

Ward GS, Parrish PR, Rigby RA. 1981. Early life stage toxicity tests with a saltwater fish: Effects of eight chemicals on survival, growth and development of sheepshead minnows (*Cyprinodon variegatus*). *J Toxicol Environ Health* 8:225-240.

Weinstein JE, Oris JT. 1999. Humic acids reduce the bioaccumulation and photoinduced toxicity of fluoranthene to fish. *Environ Toxicol Chem* 18:2087-2094.

Weinstein JE. 2002. Photoperiod effects on the UV-induced toxicity of fluoranthene to freshwater mussel glochidia: absence of repair during dark periods. *Aquatic Toxicol* 59:153-161.

Wernersson A-S, Dave G, Nilsson E. 1999. Combining sediment quality criteria and sediment bioassays with photoactivation for assessing sediment quality along the Swedish West Coast. *Aqua Ecosystem Health Manage* 2:379-389.

West CW, Kosian PA, Mount DR, Makynen EA , Pasha MS, Sibley PK, Ankley GT. 2001. Amendment of sediments with a carbonaceous resin reduces bioavailability of polycyclic aromatic hydrocarbons. *Environ Toxicol Chem* 20:1104-1111.

Wetzel. 1983. *Limnology*. 2nd Edition. Saunders College Printing, New York, U.S.A.

Whiteman FW, Ankley GT, Kahl MD, Rau DM, Balcer MD. 1996. Evaluation of interstitial water as a route of exposure for ammonia in sediment tests with benthic macroinvertebrates. *Environ Toxicol Chem* 15:794-801.

Wilkinson L. 1990. SYSTAT: The System for. SYSTAT, Inc., Evanston, IL.

Winkler KL, Duncan KL, Hose JE and Puffer JW. 1983. Effects of benzo[a]pyrene on the early development of California grunion, *Leuresthes tenuis* (pisces, atherinidae). *Fish Bull* 81: 473-481.

Wu Y, Zhang J, Mi T-z, Li B . 2001. Occurrence of n-alkanes and polycyclic aromatic hydrocarbons in the core sediments of the Yellow Sea. *Mar Chem* 76: 1-15.

Xie W-H, Shui W-Y, and Mackay D. 1997. A review of the effects of salt on the solubility of organic compounds in seawater. *Mar Environ Res* 44:429-4444.

Zaroogian G, Heltshe JF and Johnson M. 1985. Estimation of toxicity to marine species with structure-activity models developed to estimate toxicity to freshwater fish. *Aquat Toxicol* 6: 251-270.

Appendix A

Individual datasets which Comprise the Acute Lethality Database: Table from Di Toro et al. (2000).

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Freshwater					
Paramecium, *Tetrahymena elliotti*	24	S	U	10(12)	Rogerson et al., 1983
Hydra, *Hydra oligactis*	48	S	U	5	Slooff et al., 1983
Snail, *Lymnae stagnalis*	48	S	U	5	Slooff et al., 1983
Cladoceran, *Daphnia cucullata*	48	S	U	5	Canton and Adema, 1978
Cladoceran, *Daphnia magna*	24	S	U	21(28)	LeBlanc, 1980a
Cladoceran, *Daphnia magna*	48	S	U	72(78)	Abernethy et al., 1988; U.S. EPA, 1978; Canton and Adema, 1978 Rogerson et al., 1983; Bringman and Kuhn, 1959; Eastman et al., 1984; Dill, 1980
Cladoceran, *Daphnia magna*	48	S	U	19	EG&G Bionomics, 1982; Thurston et al., 1985; Adema, 1978; Oris et al., 1991; Brooke, 1991; Millemann et al., 1984; Munkrittrick et al., 1991
Cladoceran, *Daphnia magna*	48	FT,R	M	1(2)	EG&G Bionomics, 1982; Brooke, 1994
Cladoceran, *Daphnia pulex*	48	S	M	(1)	Trucco et al., 1983
Cladoceran, *Daphnia pulex*	48	S	U	6	Canton and Adema, 1978; Passino and Smith, 1987
Brine shrimp, *Artemia salina*	24	S	N	32(34)	Abernethy et al., 1988; Abernethy et al., 1986

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Crayfish, *Orconectes immunis*	96	FT	M	6	Thurston et al., 1985; Holcombe et al., 1987
Mosquito, *Aedes aegypti*	48	S	U	5	Slooff et al., 1983
Mosquito, *Culex pipiens*	48	S	U	5	Slooff et al., 1983
Midge, *Tanytarsus dissimilis*	48	S	M	9	Thurston et al., 1985; Call et al., 1983
Rainbow trout, *Oncorhynchus mykiss*	48	FT	M	7	Holcombe et al., 1987; Call et al., 1983
Rainbow trout, *Oncorhynchus mykiss*	24	FT	M	6	Call et al., 1983
Rainbow trout, *Oncorhynchus mykiss*	24	S	U	1(2)	Bently et al., 1975
Rainbow trout, *Oncorhynchus mykiss*	48	S	U	6	Slooff et al., 1983; Bently et al., 1975
Rainbow trout, *Oncorhynchus mykiss*	96	FT	M	22	Thurston et al., 1985; Call et al., 1983; Holcombe et al., 1987; Call et al., 1986; DeGraeve et al., 1982; Hodson et al., 1988
Rainbow trout, *Oncorhynchus mykiss*	96	S	M	1	Horne et al., 1983
Rainbow trout, *Oncorhynchus mykiss*	96	S	U	1	Bently et al., 1975
Bleak, *Alburnus alburnus*	96	S	I	7	Bengtsson et al., 1984

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Goldfish, *Carasius auratus*	24	S	M	26(28)	Bridie et al., 1979
Goldfish, *Carasius auratus*	24	S	U	5(6)	Pickering and Henderson, 1966
Goldfish, *Carasius auratus*	24	FT	M	1(2)	Brenniman et al., 1976
Goldfish, *Carasius auratus*	96	S	U	4	Pickering and Henderson, 1966
Goldfish, *Carasius auratus*	96	FT	M	1(2)	Brenniman et al., 1976
Goldfish, *Carasius auratus*	48	S	U	5(6)	Pickering and Henderson, 1966
Goldfish, *Carasius auratus*	48	FT	M	1(2)	Brenniman et al., 1976
Golden orfe, *Leuciscus idus melanotus*	24	S	i(ns)	26	Juhnke and Ludemann, 1978
Fathead minnow, *Pimephales promelas*	24	S	U	6	Pickering and Henderson, 1966
Fathead minnow, *Pimephales promelas*	24	FT	M	8	Ahmad et al., 1984
Fathead minnow, *Pimephales promelas*	48	S	U	11	Pickering and Henderson, 1966
Fathead minnow, *Pimephales promelas*	48	FT	M	8	Ahmad et al., 1984

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Fathead minnow, *Pimephales promelas*	96	FT	M	141(146)	Veith et al., 1983; Thurston et al., 1985; Holcombe et al., 1987; Ahmad et al., 1984; Dill,1980; DeGraeve et al., 1982; Alexander et al., 1978; Broderius and Kahl, 1985; Cairns and Nebeker, 1982; Hall et al., 1989; Hall et al., 1984; Call et al., 1985; CLSES, 1984; CLSES, 1985; CLSES, 1986; CLSES, 1988; CLSES, 1990; Kimball, 1978
Fathead minnow, *Pimephales promelas*	96	S	M	3(4)	Bridie et al., 1979; EG&G Bionomics, 1982; Gendussa, 1990; Horne et al., 1983
Fathead minnow, *Pimephales promelas*	96	R	U	1	Academy Natural Sci., 1981
Fathead minnow, *Pimephales promelas*	96	S	U	4	Pickering and Henderson, 1966
Channel catfish, *Ictalurus punctatus*	96	FT,S	M	7	Thurston et al., 1985; Holcombe et al., 1983; Gendussa, 1990
Medaka, *Oryzias latipes*	48	S	U	4(5)	Slooff et al., 1983
American flagfish, *Jordanella floridae*	24	FT	M	6	Smith et al., 1991
American flagfish, *Jordanella floridae*	48	FT	M	6	Smith et al., 1991
American flagfish, *Jordanella floridae*	96	FT	M	6	Smith et al., 1991
Mosquitofish, *Gambusia affinis*	24	S	U	(3)	Thurston et al., 1985
Mosquitofish, *Gambusia affinis*	48	S	U	(3)	Thurston et al., 1985

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Mosquitofish, *Gambusia affinis*	96	FT	M	5(6)	Thurston et al., 1985; Wallen et al., 1957
Mosquitofish, *Gambusia affinis*	96	S	U	3	Wallen et al., 1957
Guppy, *Poecilia reticulata*	24	S	U	(1)	Pickering and Henderson, 1966
Guppy, *Poecilia reticulata*	48	S	U	10(11)	Slooff et al., 1983; Pickering and Henderson, 1966
Guppy, *Poecilia reticulata*	96	S	U	4	Slooff et al., 1983
Bluegill, *Lepomis macrochirus*	24	S	U	18(19)	Pickering and Henderson, 1966; Buccafusco et al., 1981; Bently et al., 1975
Bluegill, *Lepomis macrochirus*	24	FT	M	1	Call et al., 1983
Bluegill, *Lepomis macrochirus*	48	FT	M	1	Call et al., 1983
Bluegill, *Lepomis macrochirus*	48	S	U	6(7)	Pickering and Henderson, 1966; Bently et al., 1975
Bluegill, *Lepomis macrochirus*	96	FT	M	8	Thurston et al., 1985; Bently et al., 1975; Call et al., 1983; Holcombe et al., 1987
Bluegill, *Lepomis macrochirus*	96	S	U	36(40)	Pickering and Henderson, 1966; U.S. EPA, 1978; LeBlanc, 1980b; ; Buccafusco et al., 1981; Bently et al., 1975; Dawson et al., 1977.
Tadpole, *Rana catesbeiana*	96	FT	M	5	Thurston et al., 1985

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Clawed toad, *Xenopus laevis*	48	S	U	5	Slooff and Baerselman, 1980
Mexican axolotl, *Ambystoma mexicanum*	48	S	U	5	Slooff and Baerselman, 1980
Saltwater					
Annelid worm, *Neanthes arenaceodentata*	96	S	U	4(5)	Horne et al., 1983; Rossi and Neff, 1978
Annelid worm, *Neanthes arenaceodentata*	96	R	U	(1)	Thursby et al., 1989a
Copepod, *Nitocra spinipes*	96	S	I	6	Bengtsson et al., 1984
Amphipod, *Leptocheirus plumulosus*	96	FT	M	4	Swartz, 1991a; Champlin and Poucher, 1992a; Boese et al., 1997
Mysid, *Americamysis bahia*	96	S	U	20(23)	U.S. EPA, 1978; Champlin and Poucher, 1992a; Zaroogian et al., 1985
Mysid, *Americamysis bahia*	96	S	M	1	EG&G Bionomics, 1982
Mysid, *Americamysis bahia*	96	R	U	1 8(9)	Thursby et al., 1989b
Mysid, *Americamysis bahia*	96	FT	M	8(9)	Battelle, 1987; Champlin and Poucher, 1992a; Horne et al., 1983; EG&G Bionomics, 1978; U.S. EPA, 1978; Kuhn and Lussier, 1987; Thursby, 1991b
Grass shrimp, *Palaemonetes pugio*	96	R	U	2	Battelle, 1987; Thursby et al., 1989a

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Grass shrimp, *Palaemonetes pugio*	96	S	U	4	Champlin and Poucher, 1992a; Horne et al., 1983; Thursby, 1991b; Tatem et al., 1978
Grass shrimp, *Palaemonetes pugio*	96	FT	M	1	Battelle, 1987
Grass shrimp, *Palaemonetes pugio*	96	S	M	1	Tatem, 1977
Crab, *Portunus pelagicus*	96	S	M	4	Mortimer and Connell, 1994
Inland silverside, *Menidia beryllina*	96	R	U	1	Thursby et al., 1989a
Inland silverside, *Menidia beryllina*	96	S	U	7(8)	Champlin and Poucher, 1992a; Dawson et al., 1977; Horne et al., 1983
Sheepshead minnow, *Cyprinodon variegatus*	24	S	U	7(8)	Heitmuller et al., 1981
Sheepshead minnow, *Cyprinodon variegatus*	48	S	U	11(12)	Heitmuller et al., 1981
Sheepshead minnow, *Cyprinodon variegatus*	96	S	U	13(15)	Heitmuller et al., 1981: U.S. EPA, 1978
Sheepshead minnow, *Cyprinodon variegatus*	96	FT	M	2	Ward et al., 1981; Battelle, 1987
Total Data Points				736 (796)	

[A]Method: S=static, FT=flow-through, R=renewal

[B]Concentration: U=unmeasured (nominal), M=chemical measured, I=initial

[C]Number of data points used; ()=number of data before screening for concentration>solubility and outliers.

Appendix B

Chemicals which Comprise the Acute Toxicity Database for Narcosis Chemicals in Section 2 of this Document: Table from Di Toro et al. (2000).

Chemical	CAS[A]	Class[B]	K_{OW}[C]	MW[D]	MV[E]	S[F]
triethylene glycol	112276	ao	-1.48	150.17	131	-
methanol	67561	ao	-0.715	32.04	41.0	13.5
2,4-pentanedione*	123546	k	-0.509	100.12	100	7.87
ethanol	64175	ao	-0.234	46.07	59.0	11.9
acetone	67641	k	-0.157	58.08	74.0	13.71
2-chloroethanol*	107073	ao	-0.048	80.51	65.0	9.09
2-(2-ethoxyethoxy)ethanol	111900	ao	0.011	134.17	111	-
1-chloro-2-propanol*	127004	ao	0.156	94.54	84.0	44.8
1,3-dichloro-2-propanol*	96231	ao	0.165	128.99	91.0	6.30
2-methyl-2,4-pentanediol	107415	ao	0.246	118.17	120	43.0
2-butanone	78933	k	0.316	72.11	90.0	2.81
2-propanol	67630	ao	0.341	60.10	77.0	13.6
3-chloro-1-propanol*	627305	ao	0.363	94.54	82.0	2.00
1-propanol	71238	ao	0.399	60.10	75.0	11.2
cyclopentanone	120923	k	0.453	84.12	89.0	1.11
2-methyl-2-propanol	75650	ao	0.663	74.12	95.0	16.5
methyl chloride	74873	al,ha	0.677	50.49	56.0	0.0666
2-butanol	78922	ao	0.717	74.12	93.0	14.9
methyl bromide*	74839	al,ha	0.791	94.94	57.0	0.154
3-methyl-2-butanone	563804	k	0.792	86.13	108	1.32
2,3-dibromopropanol*	96139	ao	0.819	217.90	96.0	5.97
cyclohexanone	108941	k	0.827	98.14	103	0.445
cyclopentanol	96413	ao	0.849	86.13	89.0	5.19
2-methyl-1-propanol	78831	ao	0.858	74.12	93.0	10.6
4-methyl-3-pente-2-one	141797	k	0.867	98.14	118	2.68
2-pentanone	107879	k	0.877	86.13	107	1.03
1-butanol	71363	ao	0.946	74.12	92.0	3.03
3-pentanone	96220	k	0.954	86.13	108	0.849
2-methyl-2-butanol	75854	ao	1.03	88.15	110	1.62
2-n-butoxyethanol	111762	ao	1.05	118.17	131	8.78
diethyleneglycolmono-n-butylether	112345	et	1.09	162.23	170	40.0
3,3-dimethyl-2-butanone	75978	k	1.09	100.16	125	0.954

Chemical	CAS[A]	Class[B]	K_{OW}[C]	MW[D]	MV[E]	S[F]
diethyl ether	60297	et	1.15	74.122	105	1.16
4-methoxy-4-methyl-2-pentane	107700	k	1.17	130.19	143	41.5
4-methyl-2-pentanone	108101	k	1.17	100.16	124	0.862
dichloromethane	75092	al,ha	1.18	84.93	65.0	0.211
t-butylmethyl ether	1634044	et	1.20	88.149	122	9.04
cyclohexanol	108930	ao	1.29	100.16	103	1.61
2-hexanone	591786	k	1.29	100.16	124	0.598
1,2-dichloroethane	107062	al,ha	1.40	98.96	79.0	0.114
1-pentanol	71410	ao	1.49	88.15	109	0.581
3-methyl-3-pentanol	77747	ao	1.49	102.18	125	3.79
2-phenoxyethanol	122996	ao	1.50	138.17	122	0.173
2,2,2-trichloroethanol	115208	ao	1.61	149.4	93.0	48.4
4-methyl-2-pentanol	108112	ao	1.66	102.18	126	2.25
3-hexanol	623370	ao	1.66	102.18	125	2.18
2-heptanone	110430	ke	1.67	114.19	141	0.312
5-methyl-2-hexanone	110123	ke	1.68	114.19	141	0.271
2,4-dimethyl-3-pentanol	600362	ao	1.78	116.2	140	3.05
6-methyl-5-heptene-2-one	110930	ke	1.82	126.2	151	0.487
2-hexanol	626937	ao	1.83	102.18	126	1.13
1,3-dichloropropane	142289	al,ha	1.84	112.99	97.0	0.0363
1,2-dichloropropane	78875	al,ha	1.86	112.99	99.0	0.0342
diisopropyl ether	108203	et	1.87	102.18	138	0.0918
chloroform	67663	al,ha	1.91	119.38	81.0	0.0319
1,1,2-trichloroethane	79005	al,ha	1.91	133.4	94.0	0.0369
1,4-dimethoxybenzene	150787	ar	1.95	138.165	132	0.0250
2,6-dimethoxytolunene	5673074	ar	1.99	152.19	147	0.0283
benzene	71432	ar	2.00	78.11	89.0	0.0260
1-hexanol	111273	ao	2.02	102.18	125	0.159
2-octanone	111137	ke	2.02	128.21	157	0.111
1-chloro-3-bromopropane	109706	al,ha	2.04	157.44	100	0.0184
5-methyl-3-heptanone	541855	ke	2.05	128.21	156	0.111
anisole	100663	ar	2.06	108.14	111	0.0148

Chemical	CAS[A]	Class[B]	K_{OW}[C]	MW[D]	MV[E]	S[F]
2,6-dimethyl-2,5-heptadiene	504201	ke	2.07	138.21	164	0.0171
t-1,2-dichloroethylene	156605	al,ha	2.10	96.94	81.0	0.0202
1,2,3-trichloroepropane	96184	al,ha	2.13	147.43	107	0.0177
1,1-dichloroethylene	75354	al,ha	2.19	96.94	81.0	0.0141
1,3-dibromopropane*	109648	al,ha	2.24	201.9	103	0.00930
bromoform	75252	al,ha	2.25	252.73	88.0	0.00650
1,1,2,2-tetrachloroethane	79345	al,ha	2.31	167.85	106	0.0181
1,4-dichlorobutane	110565	al,ha	2.33	127.01	113	0.00990
1,1-dichloropropane	78999	al,ha	2.36	112.99	101	0.00790
2-nonanone	821556	ke	2.38	142.24	174	0.0801
1,1,1-trichloroethane	71556	al,ha	2.38	133.4	101	0.00662
1,1,1,2-tetrachloroethane	630206	al,ha	2.43	167.85	110	0.0050
5-nonanone	502567	ke	2.44	142.24	174	0.0740
1-heptanol	111706	ao	2.57	116.2	142	0.0487
chlorobenzene	108907	ar,ha	2.58	112.56	102	0.00320
2-ethyl-1-hexanol	104767	ao	2.58	130.23	155	0.132
bicyclo(2,2,1)hepta-2,5-diene	121460	al	2.60	92.14	102	0.00490
toluene	108883	ar	2.62	92.14	107	0.00600
styrene	100425	ar	2.72	104.15	116	0.00550
tetrachloromethane	56235	al,ha	2.73	153.82	97.0	0.00248
2-decanone	693549	ke	2.73	156.27	190	0.0599
bromobenzene	108861	ar,ha	2.75	157.01	106	0.00196
cyclopentane	278923	al	2.76	70.134	95.0	0.00260
1,5-dichloropentane	628762	al,ha	2.76	141.04	130	0.00286
1,3,5-cycloheptatriene	544252	al	2.77	92.14	104	0.00377
trichloroethylene	79016	al,ha	2.81	131.39	90.0	0.00360
di-n-butyl ether	142961	et	2.89	130.23	170	0.00614
t-1,2-dichlorocyclohexane	822866	al,ha	2.90	153.05	128	0.00162
pentachloroethane	76017	al,ha	2.95	202.29	121	0.00111
2,4-hexadiene	592461	al	2.98	82.145	115	0.00237
butylphenyl ether	1126790	et	3.00	150.22	160	0.000790
benzophenone	119619	ke	3.05	182.22	163	0.000480

Chemical	CAS[A]	Class[B]	K_{OW}[C]	MW[D]	MV[E]	S[F]
ethylbenzene	100414	ar	3.06	106.17	123	0.00219
2,3-dimethyl-1,3-butadiene	513815	al	3.06	82.145	121	0.00162
2-undecanone	112129	ke	3.08	170.29	207	0.0459
1-octanol	118875	ao	3.10	130.23	158	0.0161
3-chlorotoluene	108418	ar,ha	3.12	126.59	118	0.000834
4-chlorotoluene	106434	ar,ha	3.13	126.59	118	0.000817
o-xylene	95476	ar	3.13	106.17	121	0.00191
m-xylene	108383	ar	3.19	106.17	124	0.00154
p-xylene	106423	ar	3.21	106.17	124	0.00146
1,4-dichlorobenzene	106467	ar,ha	3.24	147.00	113	0.000581
3,5,5-trimethyl-1-hexanol	3452979	ao	3.29	144.26	172	0.0117
1,2-dichlorobenzene	95501	ar,ha	3.31	147.00	113	0.000507
1,3-dichlorobenzene	541731	ar,ha	3.31	147.00	115	0.000524
napthalene	91203	pah	3.36	128.17	125	0.00110
cyclohexane	110827	al	3.38	84.16	109	0.000919
tetrachloroethylene	127184	al,ha	3.38	165.83	99.0	0.000710
2-dodecanone	6175491	ke	3.43	184.32	223	0.0357
cumene	98828	ar	3.49	120.19	140	0.000762
pentane	109660	al	3.50	72.15	116	0.000592
1,2-dibromobenzene	585539	ar,ha	3.56	235.92	119	0.000196
1,5-cyclooctadiene	111784	al	3.61	108.18	130	0.000386
1-nonanol	143088	ao	3.63	144.26	175	0.00552
1,2,4-trimethylbenzene	95636	ar	3.65	120.19	138	0.000487
n-propylbenzene	103651	ar	3.67	120.19	140	0.000467
dipentyl ether	693652	et	3.69	158.28	202	0.000757
1,3,5-trimethylbenzene	108678	ar	3.69	120.19	140	0.000414
hexachloroethane	67721	al,ha	3.73	236.74	132	0.0000936
2,4-dichlorotoluene	95738	ar,ha	3.79	161.03	129	0.000457
1-methylnaphthalene	90120	pah	3.84	142.20	140	0.000280
2-methylnaphthalene	91576	pah	3.86	142.20	141	0.000270
2-chloronaphthalene	91587	pah,ha	3.88	162.62	136	0.000100
1-chloronaphthalene	90131	pah,ha	3.88	162.62	136	0.000100
3,4-dichlorotoluene	95750	ar,ha	3.88	161.03	129	0.000120
biphenyl	92524	ar	3.91	154.21	150	0.000216

Chemical	CAS[A]	Class[B]	K_{OW}[C]	MW[D]	MV[E]	S[F]
1,3,5-trichlorobenzene	108703	ar,ha	3.97	181.45	125	0.0000933
1,2,3-trichlorobenzene	87616	ar,ha	3.98	181.45	124	0.0000870
1,2,4-trichlorobenzene	120821	ar,ha	4.00	181.45	126	0.0000886
acenaphthene	83329	pah	4.01	154.21	140	0.000100
2,5-dimethyl-2,4-hexadiene	764136	al	4.10	110.20	146	0.000133
methyl cyclohexane	108872	al	4.10	98.19	128	0.000155
1,2,4,5-tetramethylbenzene	95932	ar	4.11	134.22	152	0.000159
hexane	110543	al	4.12	86.18	132	0.000131
1,3-diethylbenzene	141935	ar	4.17	134.22	156	0.000135
1-decanol	112301	ao	4.19	158.28	192	0.00181
p-tert-butyltoluene	98511	ar	4.26	148.25	173	0.0000995
diphenylether	101848	et	4.36	170.21	152	0.0000595
amylbenzene	538681	ar	4.52	148.25	173	0.0000502
phenanthrene	85018	pah	4.57	178.23	161	0.0000340
1,2,4,5-tetrachlorobenzene	95943	ar,ha	4.64	215.89	136	0.0000151
1,2,3,4-tetrachlorobenzene	634662	ar,ha	4.64	215.89	136	0.0000145
1,2,3,5-tetrachlorobenzene	634902	ar,ha	4.64	215.89	136	0.0000148
1-undecanol	112425	ao	4.70	172.31	207	0.000640
pyrene	129000	pah	4.92	202.26	182	0.0000120
9-methylanthracene	779022	pah	5.01	192.26	175	0.00000980
fluoranthene	206440	pah	5.08	202.26	197	0.0000102
1-dodecanol	112538	ao	5.20	186.34	223	0.000238
pentachlorobenzene	608935	ar,ha	5.32	250.34	147	0.00000218
octane*	111659	al	5.34	114.23	164	0.00000625
1-tridecanol*	112709	ao	5.75	200.36	224	0.0000793
decane*	124185	al	6.56	142.28	229	0.000000300

*Chemical is not included: LC50>S.
[A]CAS=Chemical abstract number
[B]Class: ao=alcohol, ar=aromatic, ha=halogenated, et=ether, al=aliphatic, ke=ketone, pah=PAH
[C]$K_{OW}=\log_{10}(K_{OW})$;
[D]MW=molecular weight (gm/mol);
[E]V=molar volume (cm^3/mol);
[F]S=aqueous solubility(mol/L)

Appendix C

Summary of Data on the Acute Toxicity of PAHs to Freshwater and Saltwater Species and the Derivation of Genus Mean Acute Values.

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
FRESHWATER													
Hydra, *Hydra americana*	J	W,E	fluoranthene (206-44-0)	5.084	FT	M	70	0.3461	0.3461	22.06	22.06	_	Spehar et al., 1999
Hydra, *Hydra sp.*	X	W,E	phenanthrene (85-01-8)	4.571	FT	M	96	0.5386	0.5386	11.24	11.24	15.7	Call et al., 1986
Annelid, *Lumbriculus variegatus*	X	I	phenanthrene (85-01-8)	4.571	FT	M	>419	>2.351	>2.351	>49.07	_	_	Call et al., 1986
Annelid, *Lumbriculus variegatus*	A	I	fluoranthene (206-44-0)	5.084	FT	M	>178	>0.8801	>0.8801	>56.09	>52.46	>52.5	Spehar et al., 1999
Snail, *Mudalia potosensis*	X	E	fluorene (86-73-7)	4.208	S	U	**>1900**[G] (5600)	>11.42	>11.42	>108.2	>108.2	>108.2	Finger et al., 1985
Snail, *Aplexa hypnorum*	X	E	acenaphthene (83-32-9)	4.012	FT	M	>2040	>13.23	>13.23	>81.82	>81.82	>81.8	Holcombe et al., 1983
Snail, *Physa heterostropha*	X	E	fluoranthene (206-44-0)	5.084	S	U	137	0.6773	0.6773	43.17	43.17	43.2	Horne and Oblad, 1983
Snail, *Physella virgata*	A	E	fluoranthene (206-44-0)	5.084	FT	M	>178	>0.8801	>0.8801	>56.09	>56.09	>56.1	Spehar et al., 1999
Cladoceran, *Daphnia magna*	X	W	naphthalene (91-20-3)	3.356	S	U	8570	66.86	_	_	_	_	U.S. EPA, 1978
Cladoceran, *Daphnia magna*	J	W	naphthalene (91-20-3)	3.356	S	U	4723	36.85	_	_	_	_	Abernethy et al., 1986
Cladoceran, *Daphnia magna*	X	W	naphthalene (91-20-3)	3.356	S	M	2160	16.85	34.63	51.39	_	_	Millemann et al., 1984
Cladoceran, *Daphnia magna*	J	W	1-methyl naphthalene (90-12-0)	3.837	S	U	1420	9.986	9.986	42.20	_	_	Abernethy et al., 1986
Cladoceran, *Daphnia magna*	J	W	2-methyl naphthalene (91-57-6)	3.857	S	U	1491	10.49	10.49	46.29	_	_	Abernethy et al., 1986

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Cladoceran, *Daphnia magna*	X	W	acenaphthene (83-32-9)	4.012	S	U	3450	22.37	_	_	_	_	Randall and Knopp, 1980
Cladoceran, *Daphnia magna*	X	W	acenaphthene (83-32-9)	4.012	S	U	**>3800** (41000)	>24.64	_	_	_	_	LeBlanc, 1980a
Cladoceran, *Daphnia magna*	X	W	acenaphthene (83-32-9)	4.012	S	M	320	2.075	_	_	_	_	EG&G Bionomics, 1982
Cladoceran, *Daphnia magna*	X	W	acenaphthene (83-32-9)	4.012	S	M	1300	8.430	_	_	_	_	EG&G Bionomics, 1982
Cladoceran, *Daphnia magna*	X	W	acenaphthene (83-32-9)	4.012	FT	M	120	0.7782	0.7782	4.813	_	_	EG&G Bionomics, 1982
Cladoceran, *Daphnia magna*	X	W	fluorene (86-73-7)	4.208	S	U	430	2.585	2.585	24.49	_	_	Finger et al., 1985
Cladoceran, *Daphnia magna*	J	W	phenanthrene (85-01-8)	4.571	S	U	207	1.160	_	_	_	_	Abernethy et al., 1986
Cladoceran, *Daphnia magna*	X	W	phenanthrene (85-01-8)	4.571	S	U	843	4.730	_	_	_	_	Eastmond et al., 1984
Cladoceran, *Daphnia magna*	Neonate	W	phenanthrene (85-01-8)	4.571	S	M	700	3.928	_	_	_	_	Millemann et al., 1984
Cladoceran, *Daphnia magna*	Neonate	W	phenanthrene (85-01-8)	4.571	S,R	M	212	1.189	_	_	_	_	Brooke, 1994
Cladoceran, *Daphnia magna*	Neonate	W	phenanthrene (85-01-8)	4.571	FT	M	230	1.290	_	_	_	_	Brooke, 1993
Cladoceran, *Daphnia magna*	X	W	phenanthrene (85-01-8)	4.571	FT	M	117	0.6565	0.9204	19.21	_	_	Call et al., 1986
Cladoceran, *Daphnia magna*	J	W	pyrene (129-00-0)	4.922	S	U	90.9	0.4494	0.4494	20.13	_	_	Abernethy et al., 1986

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Cladoceran, *Daphnia magna*	J	W	9-methyl anthracene (779-02-2)	5.006	S	U	124.8	0.6491	0.6491	34.91	_	_	Abemethy et al., 1986
Cladoceran, *Daphnia magna*	J	W	fluoranthene (206-44-0)	5.084	S	U	**>260** (320000)	>1.285	_	_	_	_	LeBlanc, 1980a
Cladoceran, *Daphnia magna*	J	W	fluoranthene (206-44-0)	5.084	S	M	45	0.2225	_	_	_	_	Oris et al., 1991
Cladoceran, *Daphnia magna*	J	W	fluoranthene (206-44-0)	5.084	R	M	117	0.5785	_	_	_	_	Spehar et al., 1999
Cladoceran, *Daphnia magna*	X	W	fluoranthene (206-44-0)	5.084	S	M	105.7	0.5226	0.4067	25.92	25.23		Suedel ad Rodgers, 1996
Cladoceran, *Daphnia pulex*	X	W	naphthalene (91-20-3)	3.356	S	U	4663	36.38	36.38	53.99	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	X	W	fluorene (86-73-7)	4.208	S	U	212	1.275	1.275	12.08	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	X	W	1,3-dimethyl naphthalene (575-41-7)	4.367	S	U	767	4.917	4.917	65.84	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	X	W	2,6-dimethyl naphthalene (581-42-0)	4.373	S	U	193	1.237	1.237	16.78	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	X	W	anthracene (120-12-7)	4.534	S	U	**>45** (754)	>0.2528	>0.2528	>4.869[L]	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	Neonate	W	phenanthrene (85-01-8)	4.571	S	U	734	4.118	_	_	_	_	Passino and Smith, 1987
Cladoceran, *Daphnia pulex*	X	W	phenanthrene (85-01-8)	4.571	S	U	**>1100** (>1150)	>6.172	_	_	_	_	Geiger and Buikema, 1981, 1982

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Cladoceran, *Daphnia pulex*	X	W	phenanthrene (85-01-8)	4.571	S	U	350	1.964	_	_	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	X	W	phenanthrene (85-01-8)	4.571	S	M	100	0.5611	1.656	34.56	_	_	Trucco et al., 1983
Cladoceran, *Daphnia pulex*	X	W	2-methyl anthracene (613-12-7)	4.991	S	U	**>30** (96)	>0.1563	>0.1563	>8.134[L]	30.15	27.6	Smith et al., 1988
Amphipod, *Gammarus minus*	X	E	acenaphthene (83-32-9)	4.012	S	U	460	2.983	2.983	18.45	_	_	Horne et al., 1983
Amphipod, *Gammarus minus*	A	E	fluoranthene (206-44-0)	5.084	S	U	32	0.1582	0.1582	10.08	13.64	_	Horne and Oblad, 1983
Amphipod, *Gammarus pseudolimnaeus*	X	E	fluorene (86-73-7)	4.208	S	U	600	3.607	3.607	34.18	_	_	Finger et al., 1985
Amphipod, *Gammarus pseudolimnaeus*	X	E	phenanthrene (85-01-8)	4.571	FT	M	126	0.7070	0.7070	14.76	_	_	Call et al., 1986
Amphipod, *Gammarus pseudolimnaeus*	A	E	fluoranthene (206-44-0)	5.084	FT	M	43	0.2126	0.2126	13.55	18.98	16.1	Spehar et al., 1999
Amphipod, *Hyalella azteca*	J	E	fluoranthene (206-44-0)	5.084	FT	M	44	0.2175	0.2175	13.87	13.87	13.9	Spehar et al., 1999
Dragonfly, *Ophiogomphus* sp.	N	E	fluoranthene (206-44-0)	5.084	FT	M	>178	>0.8801	>0.8801	>56.09	>56.09	>56.1	Spehar et al., 1999
Stonefly, *Peltoperla maria*	X	E	acenaphthene (83-32-9)	4.012	S	U	240	1.556	1.556	9.626	_	_	Horne et al., 1983
Stonefly, *Peltoperla maria*	X	E	fluoranthene (206-44-0)	5.084	S	U	135	0.6675	0.6675	42.54	20.24	20.2	Horne and Oblad, 1983

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Midge, *Chironomus tentans*	L	I	naphthalene (91-20-3)	3.356	S	M	2810	21.92	21.92	32.53	_	_	Millemann et al., 1984
Midge, *Chironomus tentans*	L	I	phenanthrene (85-01-8)	4.571	S	M	490	2.749	2.749	57.39	_	_	Millemann et al., 1984
Midge, *Chironomus tentans*	L	I	fluoranthene (206-44-0)	5.084	S	M	>250	>1.236	>1.236	>78.78[L]	43.21	_	Suedel ad Rodgers, 1996
Midge, *Chironomus riparius*	L	I	fluorene (86-73-7)	4.208	S	U	**>1900** (2350)	>11.42	>11.42	>108.2	>108.2	>68.4	Finger et al., 1985
Midge, *Paratanytarsus sp.*	X	E	acenaphthene (83-32-9)	4.012	S	M	2000	12.97	_	_	_	_	Northwestern Aquatic Science Inc., 1982
Midge, *Paratanytarsus sp.*	X	E	acenaphthene (83-32-9)	4.012	S	M	2090	13.55	13.26	82.00	82.00	82.0	Northwestern Aquatic Science Inc., 1982
Midge, *Tanytarsus dissimilis*	L	I	naphthalene (91-20-3)	3.356	S	U	20700	161.5	_	_	_	_	Darville and Wilhm, 1984
Midge, *Tanytarsus dissimilis*	L	I	naphthalene (91-20-3)	3.356	S	U	12600	98.31	126.0	187.0	187.0	187	Darville and Wilhm, 1984
Coho salmon, *Oncorhynchus kisutch*	E	I	naphthalene (91-20-3)	3.356	R	M	>11800	>92.07	_	_	_	_	Korn and Rice, 1981
Coho salmon, *Oncorhynchus kisutch*	F	W	naphthalene (91-20-3)	3.356	R	M	5600	43.69	43.69	64.84	64.84	_	Korn and Rice, 1981
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	naphthalene (91-20-3)	3.356	S	U	1800	14.04	_	_	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	naphthalene (91-20-3)	3.356	S	U	6100	47.59	_	_	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	naphthalene (91-20-3)	3.356	S	U	2600	20.29	_	_	_	_	Edsall, C.C., 1991

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	naphthalene (91-20-3)	3.356	S	U	4400	34.33	_	_	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	naphthalene (91-20-3)	3.356	S	U	5500	42.91	_	_	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	J	W	naphthalene (91-20-3)	3.356	FT	M	1600	12.48	_	_	_	_	DeGraeve et al., 1982
Rainbow trout, *Oncorhynchus mykiss*	X	W	naphthalene (91-20-3)	3.356	FT	M	2300	17.94	14.97	22.21	_	_	DeGraeve et al., 1980
Rainbow trout, *Oncorhynchus mykiss*	J	W	acenaphthene (83-32-9)	4.012	FT	M	670	4.345	4.345	26.87	_	_	Holcombe et al., 1983
Rainbow trout, *Oncorhynchus mykiss*	J	W	fluorene (86-73-7)	4.208	S	U	820	4.930	4.930	46.71	_	_	Finger et al., 1985
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	1,3-dimethyl naphthalene (575-41-7)	4.367	S	U	1700	10.88	14.04	188.1[L]	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	L	W	phenanthrene (85-01-8)	4.571	S	U	**>1100** (3200)	>6.172	_	_	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	J	W	phenanthrene (85-01-8)	4.571	FT	M	375	2.104	2.104	43.92	_	_	Call et al., 1986
Rainbow trout, *Oncorhynchus mykiss*	X	W	fluoranthene (206-44-0)	5.084	S	M	187	0.9246	_	_	_	_	Horne and Oblad, 1983
Rainbow trout, *Oncorhynchus mykiss*	J	W	fluoranthene (206-44-0)	5.084	FT	M	26.0	0.1285	0.1285	8.193	25.13	40.4	Spehar et al., 1999
Brown trout, *Salmo trutta*	J	W	acenaphthene (83-32-9)	4.012	FT	M	580	3.761	3.761	23.26	23.26	23.3	Holcombe et al., 1983
Fathead minnow, *Pimephales promelas*	J	W	naphthalene (91-20-3)	3.356	S	M	1990	15.53	_	_	_	_	Millemann et al., 1984

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Fathead minnow, *Pimephales promelas*	J	W	naphthalene (91-20-3)	3.356	FT	M	7900	61.64	_	_	_	_	DeGraeve et al., 1982
Fathead minnow, *Pimephales promelas*	X	W	naphthalene (91-20-3)	3.356	FT	M	4900	38.23	_	_	_	_	DeGraeve et al., 1980
Fathead minnow, *Pimephales promelas*	J	W	naphthalene (91-20-3)	3.356	FT	M	6140	47.91	_	_	_	_	Geiger et al., 1985
Fathead minnow, *Pimephales promelas*	J	W	naphthalene (91-20-3)	3.356	FT	M	8900	69.44	_	_	_	_	DeGraeve et al., 1980
Fathead minnow, *Pimephales promelas*	J	W	naphthalene (91-20-3)	3.356	FT	M	6080	47.44	51.77	76.82	_	_	Holcombe et al., 1984
Fathead minnow, *Pimephales promelas*	J	W	1-methyl naphthalene (90-12-0)	3.837	S	U	9000	63.38	63.38	267.9	_	_	Mattson et al., 1976
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	S	M	3100	20.10	_	_	_	_	Marine Bioassay Lab., 1981
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	S	M	1500	9.727	_	_	_	_	EG&G Bionomics, 1982
Fathead minnow, *Pimephales promelas*	A	W	acenaphthene (83-32-9)	4.012	R	U	3700	23.99	_	_	_	_	Academy of Natural Sci., 1981
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	FT	M	1730	11.22	_	_	_	_	Geiger et al., 1985
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	FT	M	608	3.943	_	_	_	_	Cairns and Nebeker, 1982
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	FT	M	>1400	>9.079	_	_	_	_	EG&G Bionomics, 1982
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	FT	M	1600	10.38	7.713	47.71	_	_	Holcombe et al., 1983

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Fathead minnow, *Pimephales promelas*	X	W	fluorene (86-73-7)	4.208	S	U	**>1900** (100000)	>11.42	>11.42	>108.2[L]	_	_	Finger et al., 1985
Fathead minnow, *Pimephales promelas*	J	W	phenanthrene (85-01-8)	4.571	S	M	**>1100** (>1150)	>6.172	>6.172	>128.8[L]	_	_	U.S. EPA, 1978
Fathead minnow, *Pimephales promelas*	J	W	fluoranthene (206-44-0)	5.084	S	M	95	0.4697	_	_	_	_	Horne and Oblad, 1983
Fathead minnow, *Pimephales promelas*	J	W	fluoranthene (206-44-0)	5.084	S	M	7.71	0.0381	_	_	_	_	Gendusa, 1990
Fathead minnow, *Pimephales promelas*	A	W	fluoranthene (206-44-0)	5.084	FT	U	**>260** (>1000)	>1.285	_	_	_	_	Birge et al., 1982
Fathead minnow, *Pimephales promelas*	J	W	fluoranthene (206-44-0)	5.084	FT	M	69	0.3411	0.3411	21.74	67.97	68.0	Spehar et al., 1999
Channel catfish, *Ictalurus punctatus*	J	E	acenaphthene (83-32-9)	4.012	FT	M	1720	11.15	11.15	68.99	_	_	Holcombe et al., 1983
Channel catfish, *Ictalurus punctatus*	J	E	fluoranthene (206-44-0)	5.084	S	M	37.40	0.1849	0.1849	11.79	28.51	28.5	Gendusa, 1990
Bluegill, *Lepomis macrochirus*	J	W	acenaphthene (83-32-9)	4.012	S	U	1700	11.02	11.02	68.18	_	_	Buccafusco et al., 1981
Bluegill, *Lepomis macrochirus*	X	W	fluorene (86-73-7)	4.208	S	U	910	5.471	5.471	51.84	_	_	Finger et al., 1985
Bluegill, *Lepomis macrochirus*	J	W	phenanthrene (85-01-8)	4.571	FT	M	234	1.313	1.313	27.41	_	_	Call et al., 1986
Bluegill, *Lepomis macrochirus*	J	W	fluoranthene (206-44-0)	5.084	S	U	**>260** (4000)	>1.285	_	_	_	_	Buccafusco et al., 1981; EPA, 1978
Bluegill, *Lepomis macrochirus*	J	W	fluoranthene (206-44-0)	5.084	FT	M	44	0.2175	0.2175	13.87	34.04	34.0	Spehar et al., 1999

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
South african clawed frog *Xenopus laevis*	L	W	naphthalene (91-20-3)	3.356	FT	M	2100	16.38	_	_	_	_	Edmisten and Bantle, 1982
South african clawed frog *Xenopus laevis*	L	W	naphthalene (91-20-3)	3.356	FT	M	2100	16.38	16.38	24.31	24.31	24.3	Edmisten and Bantle, 1982
SALTWATER													
Annelid worm, *Neanthes arenaceodentata*	J	I	naphthalene (91-20-3)	3.356	S	U	3800	29.65	29.65	44.00	_	_	Rossi and Neff, 1978
Annelid worm, *Neanthes arenaceodentata*	X	I	acenaphthene (83-32-9)	4.012	S	U	3600	23.34	_	_	_	_	Horne et al., 1983
Annelid worm, *Neanthes arenaceodentata*	J	I	acenaphthene (83-32-9)	4.012	R	U	**>3800** (16440)	>24.64	23.34	144.4	_	_	Thursby et al., 1989a
Annelid worm, *Neanthes arenaceodentata*	A	I	phenanthrene (85-01-8)	4.571	S	U	600	3.366	3.366	70.27	_	_	Rossi and Neff, 1978
Annelid worm, *Neanthes arenaceodentata*	J	I	fluoranthene (206-44-0)	5.084	S	U	**>260** (500)	>1.285	_	_	_	_	Rossi and Neff, 1978
Annelid worm, *Neanthes arenaceodentata*	J	I	fluoranthene (206-44-0)	5.084	S	U	**>260** (20000)	> 1.285	>1.285	>81.93[L]	76.43	76.4	Spehar et al., 1999
Archiannelid, *Dinophilus gyrociliatus*	J	I	phenanthrene (85-01-8)	4.571	R	U	185.40	1.040	1.040	21.71	21.71	21.7	Battelle Ocean Sciences, 1987
Mud snail, *Nassarius obsoletus*	A	I,E	phenanthrene (85-01-8)	4.571	R	M	>245	>1.375	>1.375	>28.69	>28.69	>28.7	Battelle Ocean Sciences, 1987
Blue mussel, *Mytilus edulis*	A	E,W	phenanthrene (85-01-8)	4.571	R	M	>245	>1.375	>1.375	>28.69	>28.69	>28.7	Battelle Ocean Sciences, 1987

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Pacific oyster, *Crassostrea gigas*	E/L	W	naphthalene (91-20-3)	3.356	S	U	**>31000** (199000)	>241.9	>241.9	>358.9	>358.9	>359	U.S. EPA, 1980
Coot clam, *Mulinia lateralis*	J	E	pyrene (129-00-0)	4.922	FT	M	**>132** (>240)	>0.6526	>0.6526	>29.24	_	_	Champlin and Poucher, 1992a
Coot clam, *Mulinia lateralis*	J	E	fluoranthene (206-44-0)	5.084	S	U	**>260** (10710)	>1.285	>1.285	>81.93	>48.94	>48.9	Spehar et al., 1999
Soft-shell clam, *Mya arenaria*	A	I	phenanthrene (85-01-8)	4.571	R	M	>245	>1.375	>1.375	>28.69	>28.69	>28.7	Battelle Ocean Sciences, 1987
Calanoid copepod, *Eurytemora affinis*	A	X	naphthalene (91-20-3)	3.356	S	U	3798	22.58	22.58	33.51			Ott, et al., 1978
Calanoid copepod, *Eurytemora affinis*	A	X	2-methyl naphthalene (91-57-6)	3.857	S	U	1499	7.741	7.741	34.17	_	_	Ott, et al., 1978
Calanoid copepod, *Eurytemora affinis*	A	X	2,6-dimethyl naphthalene (581-42-0)	4.373	S	M	852	3.860	3.860	52.37	_	_	Ott, et al., 1978
Calanoid copepod, *Eurytemora affinis*	A	X	2,3,5-trimethyl naphthalene (2245-38-7)	4.856	S	M	316	1.271	1.271	49.53	41.51	41.5	Ott, et al., 1978
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	S	U	970	6.290	_	_	_	_	U.S. EPA, 1978;Ward et al., 1981
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	S	M	160	1.038	_	_	_	_	EG&G Bionomics, 1982
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	R	U	1190	7.717	_	_	_	_	Thursby et al., 1989a
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	FT	M	460	2.983	_	_	_	_	Thursby et al., 1989b

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	FT	M	190	1.232	_	_	_	_	EG&G Bionomics, 1982
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	FT	M	466.1	3.023	_	_	_	_	Horne et al., 1983;Thursby, 1991a
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	FT	M	271.9	1.763	2.104	13.01	_	_	Horne et al., 1983;Thursby, 1991a
Mysid, *Americamysis bahia*	J	E	phenanthrene (85-01-8)	4.571	FT	M	27.1	0.1521	_	_	_	_	Kuhn and Lussier, 1987
Mysid, *Americamysis bahia*	J	E	phenanthrene (85-01-8)	4.571	FT	M	17.7	0.0993	0.1229	2.565	_	_	Battelle Ocean Sciences, 1987
Mysid, *Americamysis bahia*	J	E	pyrene (129-00-0)	4.922	FT	M	28.28	0.1398	0.1398	6.264	_	_	Champlin and Poucher, 1992a
Mysid, *Americamysis bahia*	J	E	fluoranthene (206-44-0)	5.084	S	U	31	0.1533	_	_	_	_	Spehar et al., 1999
Mysid, *Americamysis bahia*	J	E	fluoranthene (206-44-0)	5.084	S	U	40	0.1978	_	_	_	_	U.S. EPA, 1978
Mysid, *Americamysis bahia*	J	E	fluoranthene (206-44-0)	5.084	FT	M	30.53	0.1509	_	_	_	_	Spehar et al., 1999
Mysid, *Americamysis bahia*	J	E	fluoranthene (206-44-0)	5.084	FT	M	87	0.4301	0.2548	16.24	7.633	7.63	EG&G Bionomics, 1978
Mysid, *Neomysis americana*	X	E	naphthalene (91-20-3)	3.356	S	M	1250	9.753	_	_	_	_	Hargreaves et al., 1982
Mysid, *Neomysis americana*	X	E	naphthalene (91-20-3)	3.356	S	M	1420	11.08	10.39	15.43	15.43	15.4	Hargreaves et al., 1982
Isopod *Excirolana vancouverensis*	J	I,E	fluoranthene (206-44-0)	5.084	R	M	>70	>0.3461	>0.3461	>22.06	>22.06	>22.1	Boese et al., 1997

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Amphipod, *Ampelisca abdita*	J	I	acenaphthene (83-32-9)	4.012	R	U	1125	7.295	7.295	45.12	_	_	Thursby et al., 1989a
Amphipod, *Ampelisca abdita*	J	I	fluoranthene (206-44-0)	5.084	S	U	67	0.3313	0.3313	21.11	30.86	30.9	Spehar et al., 1999
Amphipod, *Leptocheirus plumulosus*	A	E,I	acenaphthene (83-32-9)	4.012	FT	M	589.4	3.822	3.822	23.64	_	_	Swartz, 1991a
Amphipod, *Leptocheirus plumulosus*	A	E,I	phenanthrene (85-01-8)	4.571	FT	M	198.4	1.113	1.113	23.24	_	_	Swartz, 1991a
Amphipod, *Leptocheirus plumulosus*	J	E,I	pyrene (129-00-0)	4.922	FT	M	66.49	0.3287	0.3287	14.73	_	_	Champlin and Poucher , 1992a
Amphipod, *Leptocheirus plumulosus*	X	E,I	fluoranthene (206-44-0)	5.084	R	M	51	0.2522	0.2522	16.07	18.99	19.0	Boese et al., 1997
Amphipod, *Rhepoxynius abronius*	J	I	fluoranthene (206-44-0)	5.084	R	M	63	0.3115	0.3115	19.85	19.85	19.9	Boese et al., 1997
Amphipod, *Eohaustorius estuarius*	J	I	fluoranthene (206-44-0)	5.084	R	M	>70	>0.3461	>0.3461	>22.06	>22.06	>22.1	Boese et al., 1997
Amphipod, *Grandidierella japonica*	J	I	fluoranthene (206-44-0)	5.084	R	M	27	0.1335	0.1335	8.508	8.508	8.51	Boese et al., 1997
Amphipod, *Corophium insidiosum*	J	I	fluoranthene (206-44-0)	5.084	R	M	54	0.2670	0.2670	17.02	17.02	17.0	Boese et al., 1997
Amphipod, *Emerita analoga*	J	I,E	fluoranthene (206-44-0)	5.084	R	M	74	0.3659	0.3659	23.32	23.32	23.3	Boese et al., 1997
Kelp shrimp, *Eualis suckleyi*	X	W	naphthalene (91-20-3)	3.356	FT	M	1390	10.84	10.84	16.09	16.09	16.1	Rice and Thomas, 1989
Grass shrimp, *Palaemonetes pugio*	X	E,W	naphthalene (91-20-3)	3.356	S	M	2350	18.34	18.34	27.21	_	_	Tatem et al., 1978

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Grass shrimp, *Palaemonetes pugio*	X	E,W	acenaphthene (83-32-9)	4.012	S	U	676.8	4.389	_	_	_	_	Horne et al., 1983;Thursby, 1991b
Grass shrimp, *Palaemonetes pugio*	L	E,W	acenaphthene (83-32-9)	4.012	R	U	1697	11.00	6.950	42.98	_	_	Thursby et al., 1989a
Grass shrimp, *Palaemonetes pugio*	A	E,W	phenanthrene (85-01-8)	4.571	R	U	200.8	1.127	_	_	_	_	Battelle Ocean Sciences, 1987
Grass shrimp, *Palaemonetes pugio*	A	E,W	phenanthrene (85-01-8)	4.571	FT	M	145.4	0.8158	0.8158	17.03	_	_	Battelle Ocean Sciences, 1987
Grass shrimp, Palaemonetes pugio	J	E,W	fluoranthene (206-44-0)	5.084	S	U	142	0.7021	0.7021	44.75	30.72	30.7	Spehar et al., 1999
Sand shrimp, *Crangon septemspinosus*	X	E	acenaphthene (83-32-9)	4.012	S	U	245	1.589	1.589	9.826	9.826	9.83	Horne et al., 1983;Thursby , 1991b
American Lobster, *Homarus americanus*	L	_	fluoranthene (206-44-0)	5.084	R	U	**>260** (317)	1.285	1.285	81.93	81.93	81.9	Spehar et al., 1999
Hermit crab, *Paqurus longicarpus*	A	E	phenanthrene (85-01-8)	4.571	FT	M	163.7	0.9185	0.9185	19.17	19.17	19.2	Battelle Ocean Sciences, 1987
Slipper limpet, *Crepidula fornicata*	L	W	acenaphthene (83-32-9)	4.012	R	U	3426	22.28	22.28	137.8	137.8	138	Thursby et al., 1989a
Sea urchin, *Arbacia punctalata*	E	W	acenaphthene (83-32-9)	4.012	S	U	**>3800** (8163)	>24.64	>24.64	>152.4	_	_	Thursby et al., 1989a
Sea urchin, *Arbacia punctalata*	E	W	fluoranthene (206-44-0)	5.084	S	U	**>260** (20000)	>1.285	>1.285	>81.93	>117.2	>117	Spehar et al., 1999
Pink salmon, *Oncorhynchus gorbuscha*	Fry	W	naphthalene (91-20-3)	3.356	FT	M	960	7.490	_	_	_	_	Rice and Thomas, 1989
Pink salmon, *Oncorhynchus gorbuscha*	Fry	W	naphthalene (91-20-3)	3.356	FT	M	900	7.022	_	_	_	_	Rice and Thomas, 1989

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Pink salmon, *Oncorhynchus gorbuscha*	Fry	W	naphthalene (91-20-3)	3.356	FT	M	990	7.724	_	_	_	_	Rice and Thomas, 1989
Pink salmon, *Oncorhynchus gorbuscha*	Fry	W	naphthalene (91-20-3)	3.356	FT	M	1010	7.880	_	_	_	_	Rice and Thomas, 1989
Pink salmon, *Oncorhynchus gorbuscha*	Fry	W	naphthalene (91-20-3)	3.356	FT	M	890	6.944	7.40	10.99	10.99	11.0	Rice and Thomas, 1989
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	acenaphthene (83-32-9)	4.012	S	U	2200	14.27	_	_	_	_	Heitmuller et al., 1981
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	acenaphthene (83-32-9)	4.012	R	U	**>3800** (50000)	>25.00	_	_	_	_	Thursby et al., 1989a
Sheepshead minnow, *Cyprinodon variegatus*	A	E,W	acenaphthene (83-32-9)	4.012	FT	M	3100	20.10	20.10	124.3	_	_	Ward et al., 1981
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	phenanthrene (85-01-8)	4.571	R	U	>245	>1.375	_	_	_	_	Battelle Ocean Sciences, 1987
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	phenanthrene (85-01-8)	4.571	FT	M	429.4	2.409	2.409	50.29	_	_	Battelle Ocean Sciences, 1987
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	pyrene (129-00-0)	4.922	FT	M	**>132** (>640)	>0.6526	>0.6526	>29.24	_	_	Champlin and Poucher, 1992a
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	fluoranthene (206-44-0)	5.084	S	U	**>260** (>20000)	>1.285	_	_	_	_	Spehar et al., 1999
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	fluoranthene (206-44-0)	5.084	S	U	**>260** (>560000)	>1.285	>1.285	>81.93[L]	79.07	79.1	Heitmuller et al., 1981;U.S EPA, 1978
Inland silverside, *Menidia beryllina*	X	W	acenaphthene (83-32-9)	4.012	S	U	2300	14.91	_	_	_	_	Horne et al., 1983
Inland silverside, *Menidia beryllina*	J	W	acenaphthene (83-32-9)	4.012	R	U	**>3800** (5564)	>24.64	>19.17	>118.6	_	_	Thursby et al., 1989a

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Inland silverside, *Menidia beryllina*	J	W	pyrene (192-00-0)	4.922	FT	M	**>132** (>188.17)	>0.6526	>0.6526	>29.24	_	_	Champlin and Poucher, 1992a
Inland silverside, *Menidia beryllina*	J	W	fluoranthene (206-44-0)	5.084	S	U	**>260** (>616)	>1.285	>1.285	>81.93	>65.73	_	Spehar et al., 1999
Atlantic silverside, *Menidia menidia*	A	W	phenanthrene (85-01-8)	4.571	FT	M	108	0.6060	0.6060	12.65	12.65	28.8	Battelle Ocean Sciences, 1987
Winter flounder, *Pseudopleuronectes americanus*	J	_	fluoranthene (206-44-0)	5.084	S	M	>188	>0.9295	>0.9295	>59.24	>59.24	>59.2	Spehar et al., 1999

[A]Life-stage: A = adult, J = juvenile, L = larvae, E = embryo, U = life-stage and habitat unknown, X = life-stage unknown but habitat known.
[B]Habitat: I = infauna, E = epibenthic, W = water column.
[C]log K_{OW}: Predicted using SPARC (Karickoff et al, 1991).
[D]Method: S= static, R = renewal, FT= flow-through.
[E] Concentration: U = unmeasured (nominal), M = chemical measured.
[F] Acute Values: 96 hour LC50 or EC50, except for *Daphnia* and *Tanytarsus* which are 48 hours duration.
[G] Bolded acute values are the water solubilities of the PAH (Mackay et al., 1992). For these tests the acute values exceeded solubility. Therefore, solubilities are used instead of the acute value for further calculations.
[H] PAH-specific SMAV: Geometric mean of the acute values by PAH and species.
[I] PAH-specific SMAVs at a log K_{OW}=1.0; calculated as antilog ($\log_{10}$LC50 + 0.945$\log_{10}K_{OW}$)/1000 (see Equation 2-33).
[J] Species SMAV: Geometric mean of K_{OW}-normalized SMAVs for a species across PAHs.
[K] GMAV: Geometric mean of SMAVs for all species within a genus.
[L] Not used in calculations.

Appendix D

Comparison of PAH-specific Equilibrium Partitioning Sediment Benchmarks (ESBs) Derived from Narcosis Theory and the Median Response Concentration of Benthic Species for Individual PAHs in Spiked-sediment Toxicity Tests.

Common Name, *Scientific Name*	Chemical	Response	Median Response Conc.[A] (µg/goc)	$C_{OC,PAHi,FCVi}$ (µg/goc)	Test-Specific $ESBTU_{FCVi}$[B] (Unitless)	PAH-Specific SMAV[C]	GMAV[D]	References[E]
Oligochaete, *Lumbriculus variegatus*	pyrene	7 d LC50	**>9090** (61100)	694	**>13.1**	-	-	Kukkonen and Landrum, 1994
Oligochaete, *Lumbriculus variegatus*	pyrene	7 d EC50-SA	**>9090** (51400)	694	**>13.1**	-	-	Kukkonen and Landrum, 1994
Oligochaete, *Limnodrilus hoffmeisteri*	phenanthrene	10 d LC50	**>34300** (42500)	593	**>57.8**	**>57.8**	**>57.8**	Lotufo and Fleeger, 1996
Oligochaete, *Limnodrilus hoffmeisteri*	phenanthrene	28 d EC25-R	5790	593	9.80	-	-	Lotufo and Fleeger, 1996
Oligochaete, *Limnodrilus hoffmeisteri*	pyrene	28 d EC25-R	8440	694	12.2	-	-	Lotufo and Fleeger, 1996
Cladoceran, *Daphnia magna*	fluoranthene	10 d LC50	2380	704	-	-	-	Suedel et al., 1993
Cladoceran, *Daphnia magna*	fluoranthene	10 d LC50	955	704	-	-	-	Suedel et al., 1993
Cladoceran, *Daphnia magna*	fluoranthene	10 d LC50	3260	704	-	-	-	Suedel et al., 1993
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	**>23900** (37649)	704	-	-	-	Driscoll et al., 1997a
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	1250	704	-	-	-	Suedel et al., 1993
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	1480	704	-	-	-	Suedel et al., 1993
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	500	704	-	-	-	Suedel et al., 1993
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	22000	704	31.3	-	-	Harkey et al., 1997
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	5130	704	7.29	15.1	15.1	DeWitt et al., 1989
Amphipod, *Corophium spinicorne*	fluoranthene	10 d LC50	2830	704	4.02	-	-	Swartz et al., 1990

Common Name, *Scientific Name*	Chemical	Response	Median Response Conc.[A] (µg/goc)	$C_{OC,PAHi,FCVi}$ (µg/goc)	Test-Specific $ESBTU_{FCVi}$[B] (Unitless)	PAH-Specific SMAV[C]	GMAV[D]	References[E]
Amphipod, *Corophium spinicorne*	fluoranthene	10 d LC50	4390	704	6.23	5.01	5.01	Swartz et al., 1990
Amphipod, *Leptocheirus plumulosus*	acenapthene	10 d LC50	10900	489	22.3	-	-	Swartz et al., 1991a
Amphipod, *Leptocheirus plumulosus*	acenapthene	10 d LC50	23500	489	48.1	-	-	Swartz et al., 1991a
Amphipod, *Leptocheirus plumulosus*	acenapthene	10 d LC50	8450	489	17.3	26.4	-	Swartz et al., 1991a
Amphipod, *Leptocheirus plumulosus*	phenanthrene	10 d LC50	6870	593	11.59	-	-	Swartz et al., 1991a
Amphipod, *Leptocheirus plumulosus*	phenanthrene	10 d LC50	8080	593	13.63	-	-	Swartz et al., 1991a
Amphipod, *Leptocheirus plumulosus*	phenanthrene	10 d LC50	8180	593	13.8	13.0	18.5	Swartz et al., 1991a
Amphipod, *Rhepoxynius abronius*	acenapthene	10 d LC50	2310	489	4.72	-	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	acenapthene	10 d LC50	2110	489	4.31	4.51	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	phenanthrene	10 d LC50	3080	593	5.19	-	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	phenanthrene	10 d LC50	2220	593	3.74	4.41	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	pyrene	10 d LC50	1220	694	1.76	-	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	pyrene	10 d LC50	2810	694	4.05	2.67	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	>4360	704	>6.19	-	-	DeWitt et al., 1992
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	4410	704	6.26	-	-	DeWitt et al., 1992

Common Name, *Scientific Name*	Chemical	Response	Median Response Conc.[A] (µg/goc)	$C_{OC,PAHi,FCVi}$ (µg/goc)	Test-Specific $ESBTU_{FCVi}$[B] (Unitless)	PAH-Specific SMAV[C]	GMAV[D]	References[E]
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	3080	704	4.38	-	-	DeWitt et al., 1992
Amphipod, Rhepoxynius abronius	fluoranthene	10 d LC50	2230	704	3.17	-	-	Swartz et al., 1990
Amphipod, Rhepoxynius abronius	fluoranthene	10 d LC50	3150	704	4.50	-	-	DeWitt et al., 1992
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	1890	704	2.68	-	-	Swartz et al., 1990
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	2790	704	3.96	-	-	De Witt et al., 1992
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	2320	704	3.30	-	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	1700	704	2.41	-	-	DeWitt et al., 1989
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	1030	704	1.47	-	-	Swartz et al., 1988
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	2100	704	2.98	-	-	Swartz et al., 1990
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	3310	704	4.70	3.56	3.67	Swartz et al., 1997
Amphipod, *Eohaustorius estuarius*	acenapthene	10 d LC50	1630	489	3.33	-	-	Swartz et al., 1991a
Amphipod, *Eohaustorius estuarius*	acenapthene	10 d LC50	4180	489	8.55	-	-	Swartz et al., 1991a
Amphipod, *Eohaustorius estuarius*	acenapthene	10 d LC50	1920	489	3.93	4.82	-	Swartz et al., 1991a
Amphipod, *Eohaustorius estuarius*	phenanthrene	10 d LC50	4210	593	7.10	-	-	Swartz et al., 1991a
Amphipod, *Eohaustorius estuarius*	phenanthrene	10 d LC50	3760	593	6.34	-	-	Swartz et al., 1991a

Common Name, *Scientific Name*	Chemical	Response	Median Response Conc.[A] (µg/goc)	$C_{OC,PAHi,FCVi}$ (µg/goc)	Test-Specific $ESBTU_{FCVi}$[B] (Unitless)	PAH-Specific SMAV[C]	GMAV[D]	References[E]
Amphipod, *Eohaustorius estuarius*	phenanthrene	10 d LC50	4060	593	6.85	6.75	-	Swartz et al., 1991a
Amphipod, *Eohaustorius estuarius*	fluoranthene	10 d LC50	3100	704	4.40	-	-	DeWitt et al., 1989
Amphipod, *Eohaustorius estuarius*	fluoranthene	10 d LC50	3930	704	5.59	-	-	DeWitt et al., 1989
Amphipod, *Eohaustorius estuarius*	fluoranthene	10 d LC50	3570	704	5.07	5.00	5.46	DeWitt et al., 1989
Midge, *Chironomus tentans*	fluoranthene	10 d LC50	1590	704	-	-	-	Suedel et al., 1993
Midge, *Chironomus tentans*	fluoranthene	10 d LC50	1740	704	-	-	-	Suedel et al., 1993
Midge, *Chironomus tentans*	fluoranthene	10 d LC50	682	704	-	-	-	Suedel et al., 1993
Amphipod, *Diporeia* sp.	pyrene	31 d LC50	**>9090**	694	**>13.1**	-	-	Landrum et al., 1994
Amphipod, *Diporeia* sp.	fluoranthene	10 d LC50	(147000) **>23900** (29300)	704	**>34.0**	**>34.0**	**>34.0**	Driscoll et al., 1997a

[A] Bolded median response concentration (acute) values are the $C_{OC,PAHi,Maxi}$ based on the water solubilities of the PAH (Mackay et al., 1992). For these tests the interstitial water concentration at the median response concentration exceeded solubility. Therefore, solubilities are used instead of the acute value for further calculations.

[B] Test-specific ESBTUs: Quotient of the median response concentration (µg/goc) and $C_{OC,PAHi,FCVi}$ (from Table 3-4).

[C] PAH-specific SMAV: Geometric mean of the test-specific $ESBTU_{FCVi}$ values from 10-d LC50 tests by species and PAH. Test-specific $ESBTU_{FCVi}$ values greater that solubility included only if they are the sole 10-d LC50 for the species.

[D] GMAV: Geometric mean of the PAH-specific SMAVs for all species within a genus.

[E] Spiked sediments from Suedel et al. (1993) were unlikely at equilibrium; i.e., organisms were tested after only 18 to 24 hours after spiking.

Appendix E

CAS#, Molecular Weight and Solid Solubility of Selected PAHs.

PAH	CAS #[A]	Molecular Weight (µg/µmol)	Mackay Solid Solubility[B] (µg/L)
indan	496117	118.18	100000
naphthalene	91203	128.17	30995
C1-naphthalenes	-	142.20	??
1-methylnaphthalene	90120	142.20	28001
2-methylnaphthalene	91576	142.20	25000
acenaphthylene	208968	152.20	16314
acenaphthene	83329	154.21	3800
1-ethylnaphthalene	1127760	156.23	10100
2-ethylnaphthalene	939275	156.23	8001
C2-naphthalenes	-	156.23	??
1,4-dimethylnaphthalene	571584	156.23	11400
1,3-dimethylnaphthalene	575417	156.23	8001
2,6-dimethylnaphthalene	581420	156.23	1700
2,3-dimethylnaphthalene	581408	156.23	2500
1,5-dimethylnaphthalene	571619	156.23	3100
fluorene	86737	166.22	1900
C3-naphthalenes	-	170.25	??
2,3,5-trimethylnaphthalene	2245387	170.26	??
1,4,5-trimethylnaphthalene	213411	170.20	2100
anthracene	120127	178.12	45.00
phenanthrene	85018	178.23	1100
C1-fluorenes	-	180.25	??
1-methylfluorene	1730376	180.25	1090
C4-naphthalenes	-	184.28	??
2-methylanthracene	613127	192.26	29.99
1-methylanthracene	610480	192.26	??
9-methylanthracene	779022	192.26	261.1
2-methylphenanthrene	2531842	192.26	??
1-methylphenanthrene	832699	192.26	269.9
C1-phenanthrene/anthracenes	-	192.26	??
9-ethylfluorene	2294828	194.28	??
C2-fluorenes	-	194.27	??
pyrene	129000	202.26	131.9
fluoranthene	206440	202.26	239.9
2-ethylanthracene	52251715	206.29	??
C2-phenanthrene/anthracenes	-	206.29	??
9,10-dimethylanthracene	781431	206.29	55.90
3,6-dimethylphenanthrene	1576676	206.29	??
C3-fluorenes	-	208.3	??
C1-pyrene/fluoranthenes	-	216.29	?
2,3-benzofluorene	243174	216.28	2.001

PAH	CAS #[A]	Molecular Weight (µg/µmol)	Mackay Solid Solubility[B] (µg/L)
benzo(a)fluorene	238843	216.29	45.00
C3-phenanthrene/anthracenes	-	220.32	??
naphthacene	92240	228.30	0.600
benz(a)anthracene	56553	228.29	11.00
chrysene	218019	228.29	2.000
triphenylene	217594	228.3	43.00
C2-pyrene/fluoranthenes	-	230.13	??
C4-phenanthrenes/anthracenes	-	234.23	??
C1-benzanthracene/chrysenes	-	242.32	??
C3-pyrene/fluoranthenes	-	244.32	??
benzo(a)pyrene	50328	252.31	3.810
perylene	198550	252.31	0.4012
benzo(e)pyrene	192972	252.32	4.012
benzo(b)fluoranthene	205992	252.32	1.501
benzo(j)fluoranthene	205822	252.32	2.500
benzo(k)fluoranthene	207089	252.32	0.7999
C2-benzanthracene/chrysenes	-	256.23	??
9,10-dimethylbenz(a)anthracene	56564	256.35	43.50
7,12-dimethylbenz(a)anthracene	57976	256.35	49.99
7-methylbenzo(a)pyrene	63041770	266.35	??
benzo(ghi)perylene	191242	276.23	0.2600
C3-benzanthracene/chrysenes	-	270.36	??
indeno(1,2,3-cd)pyrene	193395	276.23	??
dibenz(a,h)anthracene	53703	278.35	0.6012
dibenz(a,j)anthracene	58703	278.35	12.00
dibenz(a,c)anthracene	215587	278.35	1.601
C4-benzanthracene/chrysenes	-	284.38	??
C1-dibenz(a,h)anthracenes	-	292.37	??
coronene	191071	300.36	0.1400
C2-dibenz(a,h)anthracenes	-	306.39	??
C3-dibenz(a,h)anthracenes	-	320.41	??

[A] For C#-PAHs, a CAS is not available.
[B] Mackay et al. (1992).

Appendix F

Water-only and Interstitial Water LC50s used in Table 5-1.

Chemical Test Species	Method[A]	Water-only LC50 (µg/L)	Interstitial Water LC50 (µg/L)	References
Freshwater				
Fluoranthene				
Diporeia sp.	FT,M/10	>194	>381.3	Driscoll et al., 1997a,b
Hyalella azteca	FT,M/10	130.7	>75.4	Driscollet al., 1997a,b
Hyalella azteca	S,M/10	44.9	45.9	Suedel et al., 1993
Hyalella azteca	S,M/10	44.9	236.5	Suedel et al., 1993
Hyalella azteca	S,M/10	44.9	97.6	Suedel et al., 1993
Chironomus tentans	S,M/10	31.9	91.2	Suedel et al., 1993
Chironomus tentans	S,M/10	31.9	251	Suedel et al., 1993
Chironomus tentans	S,M/10	31.9	75.7	Suedel et al., 1993
Saltwater				
Acenaphthene				
Eohaustorius estuarius	FT,M/10	374	800	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	374	609	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	374	542	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	678	>1,720	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	678	1410	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	678	1490	Swartz, 1991a
Fluoranthene				
Leptocheirus plumulosus	S/10	39.2	-	Driscoll et al., 1998
Phenanthrene				
Eohaustorius estuarius	FT,M/10	131	138	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	131	139	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	131	146	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	185	387	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	185	306	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	185	360	Swartz, 1991a
2,6-dimethylnaphthlene				
Rhepoxynius abronius	S,M/10	-	200	Ozretich et al., 2000a
2,3,5-trimethylnaphthlene				
Rhepoxynius abronius	S,M/10	-	153	Ozretich et al., 2000a
1-methylfluorene				
Rhepoxynius abronius	S,M/10	-	44	Ozretich et al., 2000a
2-methylphenanthrene				
Rhepoxynius abronius	S,M/10	-	70	Ozretich et al., 2000a
9-methylanthracene				
Rhepoxynius abronius	S,M/10	-	32	Ozretich et al., 2000a
Acenaphthene				
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997

Chemical Test Species	Method[A]	Water-only LC50 (µg/L)	Interstitial Water LC50 (µg/L)	References
Naphthalene				
Rhepoxynius abronius	S,M/10	-	10440	Ozretich et al., 2000a
Phenanthrene				
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997
Pyrene				
Rhepoxynius abronius	S,M/10	-	28.1	Ozretich et al., 2000a
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997
Fluoranthene				
Rhepoxynius abronius	S,M/10	13.9	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	13.9	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	13.9	22.7	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	13.9	29.4	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	13.9	24.2	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	13.9	> 315	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	13.9	14.1	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	13.9	26.6	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	13.9	19.2	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	13.9	9.38	DeWitt et al., 1992
		Mean LC50 ratio =	1.6	

[A] Test conditions for water-only toxicity tests: S = static, FT = flow-through, M = measured, 10 = 10-d duration.

Appendix G

Teratogenic Effects from Laboratory Exposure to PAHs.

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
fathead minnow (embryos), *Pimephales promelas*	maternal via water	lab; flow-through	Anthracene	6.66 µg/L 11.6 µg/L	6 wks 3 wks	-yolk-sac malformations -edema -eye deformities	8.8a µg/g (eggs)	Effects on embryos incubated with solar ultraviolet light radiation	Hall and Oris, 1991
freshwater topminnows, *Poeciliopsis monacha Poeciliopsis lucida*	water; acetone carrier	lab; static renewal	BaP	1,000 µg/L nominal; 1,250 µg/L was acutely lethal	24 h followed by 6 mo. of monitoring	-increased AHH and EROD activities	9.0 µg/g converted from 35.7 nmol/g wet wt.	Implied effect - increased AHH and EROD activity indicative of carcinogenic and teratogenic metabolites formed during metabolism of BaP by MFO-system	Goddard et al., 1987
English sole (embryos), *Parophrys vetulus*	maternal via oral	lab; wild-caught	BaP	8,000 µg/L (8 mg/kg force-fed)	-	-malformation of tail regions -insufficient yolk-sac -reduced fin-fold size -reduced hatching success	51.2 and 263 µg/g (eggs) - avg. = 157; Tissue conc. from 80 mg/kg i.p. maternal injection	-Eggs maintained 11 days until yolk-sac absorbed; static. -Incidence of effect 4 times greater than controls (Chai-square df=3.81)	Hose et al., 1981
Rainbow trout (embryos), *Oncorhynchus mykiss*	aqueous from BaP spiked to sediment	lab; static renewal (7-10d)	BaP	0.21 µg/L measured	through to 36 d post-hatch	-nuclear pycnosis -lack of body pigment -insufficient yolk-sac -abnormalities of eyes -increased mortality (at 2.40 µg/L in aqueous) -muscle necrosis -abnormal mitosis in eyes and brains	1.93 µg/g (eggs), 12.34 µg/g (alevins), from exposure to 2.40 µg/L BaP	Poor control survival (52% mortality)	Hannah et al., 1982; Hose et al., 1984
Sand sole (embryos), *Psettichthys melanostichus*	water; static	lab	BaP	0.1 µg/L measured; range (0.08 - 0.12)	through to yolk-sac absorption (7 -10 d)	-overgrowth of tissues -arrested development -twinning; Effects only after 48 h, i.e., during organogenesis	2.1 µg/g wet weight	effects only exhibited in 5% of animals; average hatching success of controls only 57% versus 28% BaP-treated	Hose et al., 1982

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
Flathead sole (embryos), *Hippoglossoide selassodon*	water; static	lab	BaP bound to bovine serum albumin	4.2 µg/L decreasing to <0.05 µg/L (DL)	through to yolk-sac absorption (7 -10 d)	-hatching success sig. decrease -nuclear pycnosis and general disruption of neural and ocular tissues	-	very low hatching success in controls and experimentals; 5.5 and 11.5%, respectively	Hose et al., 1982
English sole (embryos), *Parophrys vetulus*	water	lab	BaP	2.1 µg/L measured	through to yolk-sac absorption (7 -10 d)	none	-	-	Hose et al., 1982
gizzard shad, *Dorosoma cepedianum*	water via treated sediment	lab; static	BaP	1.38 µg/g sediment (initial); 0.74 µg/g sediment (mean of days 4,8 and 15)	22 d	none	BDL in all but 2 fish on day 4 - (0.001 and 0.0002 µg/g wet weight)	-40 ligated shad in 250 L H_2O with 4.15 kg sediment -no sig. decline in sediment conc. after day 4.	Kolok et al., 1996
gizzard shad, *Dorosoma cepedianum*	water and/or sediment ingestion	lab; static	BaP	1.02 µg/g sediment (initial); 0.63 µg/g sediment (mean of days 4,8, and 15)	22 days	none	ligated fish: 0.010 µg/g wet weight (n=4) non- ligated: 0.012 µg/g wet weight (n=14)	-50 shad, 30 ligated; 20 non-ligated, in 500 L H_2O with 3.15 kg sediment -no sig. decline in sediment conc. after day 4 -all other tissue concs. BDL (n=26 ligated; n=6 non- ligated)	Kolok et al., 1996
estuarine clams, *Rangia cuneata*	water; acetone carrier	lab; static	BaP	30.5 µg/L	24 h	none	7.2 µg/g wet weight	-majority of BaP concentrated in the viscera (~75%) -n=5	Neff and Anderson, 1975
estuarine clams, *Rangia cuneata*	water; acetone carrier	lab; static	BaP	30.5 µg/L	24 h	none	5.7 µg/g wet weight	-majority of BaP concentrated in the viscera (~65%) -n=8	Neff and Anderson, 1975

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
coho salmon (24 h Post fertilization), *Oncorhynchus kisutch*	water; 0.5% DMSO	lab; static exposure then flow-through	BaP	25,000 µg/L	24 h	none	-	Effects on hatching, orientation, and foraging only.	Ostrander et al., 1988
coho salmon, (32 d post fertilization), *Oncorhynchus kisutch*	water; 0.5% DMSO	lab; static exposure then flow-through	BaP	25,000 µg/L	24 h	none	-	Effects on hatching, orientation, and foraging only.	Ostrander et al., 1988
coho salmon, (24 h Post fertilization), *Oncorhynchus kisutch*	water; 0.5% DMSO	lab; static exposure then flow-through	BaP	25,000 µg/L	24 h	none	0.54 decreasing to 0.15 nmol/mg protien from 2 to 68 d post fertilization	Conc. of BaP in tissue are not converted because wet weights were not given; only the mg protein/animal. Can possibly borrow weights from earlier paper.	Ostrander et al., 1989
coho salmon, (32 d post fertilization), *Oncorhynchus kisutch*	water; 0.5% DMSO	lab; static exposure then flow-through	BaP	25,000 µg/L	24 h	none	4.47 decreasing to 0.33 nmol/mg protien from 2 to 68 d post fertilization	Conc. of BaP in tissue are not converted because wet weights were not given; only the mg protein/animal. Can possibly borrow weights from earlier paper.	Ostrander et al., 1989
Calif. grunion (embryos), *Leuresthes tenuis*	water	lab; static	BaP	measured: 5 µg/L (steady-state); 24 µg/L (initial)	15 days	-reduction in % hatch -lateral folding of tail -absence of caudal fin folds -hemorrhagic lesion or congested vasculature in caudal region	day 15: 0.992 ppm (wet weight); 6.872 ppm (dry weight)	steady state concentration reached in 4 to 10 days	Winkler et al., 1983

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
Calif. grunion (embryos), *Leuresthes tenuis*	water	lab; static	BaP	measured: 5-24 µg/L (steady state); 24-361 µg/L (initial)	15 days	-retarded growth (14d) -sporadic heart beat -displaced head relative to yolk-sac -absence of melanophores near lateral lines -absence of lens formation -lesions as larvae (above)	day 15:0.92 to 10.48 µg/g wet weight; 6.87 to 62.80 µg/g (dry weight)	steady state concentration reached in 4 to 10 days	Winkler et al., 1983
Calif. grunion (embryos), *Leuresthes tenuis*	water	lab; static	BaP	measured: 869 ppb (initial); steady-state not reached	15 days	-retarded growth (14d) -lateral curvature mid-body -absent melanophores -unused yolk sac -lesions as larvae (above)	day 15 - 19.98 µg/g wet weight; 112.03 µg/g dry weight	steady-state concentration never reached	Winkler et al., 1983
Pacific herring (embryos), *Clupea pallasi*	seawater contaminated by contact with oiled gravel - experiment 1; less weathered	lab; static	Field Mixture[A]	9.1 µ/L	16 days	-yolk sac edema	13.7 µg/g wet weight	Crude Oil characterized for PAHs only; concentrations of individual PAHs not given	Carls et al., 1999
Pacific herring (embryos), *Clupea pallasi*	seawater contaminated by contact with oiled gravel - experiment 2; more weathered	lab; state	Field Mixture[A]	0.41 µ/L to 0.72 µ/L	16 days	- yolk sac edema -pericardial edema - skeletal, spinal, and craniofacial abnormalities - anaphase aberration	0.022 µg/g wet weight	Crude Oil characterized for PAHs only; concentrations of individual PAHs not given	Carls et al., 1999

[A]Artificially weathered Alaska North Slope crude oil.

Appendix H

Carcinogenic Effects from Laboratory and Field Exposure to PAHs and PAH Mixtures.

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
Japanese Medaka, *Oryzia latipes* (6-10 d old)	Water; dimethyl-formamide carrier.	Lab; static	BaP	261 µg/L	2 x 6h, 1 week apart	Neoplastic lesions in livers and other tissues after 36 weeks 36% vs 1% (controls); 20 fish with adenoma, 6 with hepatocellular carcinoma	-	Exposures carried out at 26°C in the dark; concentration exceeds saturation solubility of BaP	Hawkins et al, 1988; Hawkins et al., 1990
guppy, *Poecilia reticulata* (6-10 d old)	Water; dimethyl-formamide carrier.	Lab; static	BaP	209 µg/L	2 x 6h, 1 week apart	Neoplastic lesions in livers and other tissues after 52 weeks 23% vs 0% (controls); 1 altered foci, 5 adenoma, 4 with hepatocellular carcinoma	-	Studies carried out longer because tumorigenic response in guppy is slower than in medaka	Hawkins et al, 1988; Hawkins et al., 1990
Rainbow trout (fingerlings), *Oncorhynchus mykiss*	oral	Lab	BaP	1,000 ppm per feeding	12 and 18 months	Incidence of neoplasms on liver 15% (1.0/liver) at 12 months 25% (7.7/liver) at 18 months	-	MFO info also available 0% at 6 months 0% on other organs	Hendricks et al., 1985
Rainbow trout (juvenile), *Oncorhynchus mykiss* (10 mo)	ip injection	Lab	BaP	1 mg B(a)P in 0.4 ml PG (1/month for 12 months)	18 months (6 months after final injection)	Incidence of neoplasms in various organs = 46% (x = 7.7 tumors/organ)	-	Organs examined = gonads, swim bladder, liver, spleen, head and trunk kidneys, pancreas, intestines, and stomach	Hendricks et al., 1985
Poeciliopsis lucida and *Poeciliopsis monacha* (1-7 months old)	water; acetone carrier	Lab: (multiple exposures) 3 to 4 exposure periods of 5-20 hours each week	7,12-dimethylbenz(a)-anthracene	5 ppm (per exposure)	7 - 8 months (from initial exposure)	incidence of hepatic tumors = 48%	-	only survivors examined = (55% mortality in 5 ppm treatment) (13% mortality in control)	Schultz and Schultz 1982

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
Poeciliopsis lucida and *Poeciliopsis monacha* (1-6 weeks old)	water; acetone carrier	Lab: (multiple exposures) 5 exposures periods of 6 hours each week	7,12-dimethylbenz(a)-anthracene	5 ppm (per exposure)	6 - 7 months	Incidence of hepatic tumors = 41.8%	-	22% mortality in treatment 16% mortality in control Tumor-bearing livers enlarged, yellow-white to greenish and granular.	Schultz and Schultz 1982
Bullheads	Direct skin (river sediment extract)	Lab	Field Mixture[A]	5% RSE painted once per week	18 months	23% of survivors hyperplastic 9% with multiple papillomas	-	Survival of control and experimental fish was 31%.	Black, 1983
Japanese Medaka, *Poecilia reticulata* (6-10 d old)	Water via Sediment extract re-dissolved in acetone	Lab	Field Mixture[B]	182 ppb TPAH Black River, OH extract; 254 ppb TPAH Fox River, WI extract	24 h	hepatocellular carcinoma - Black River Ex. (2/15 fish); Pancreatic-duct cell adenoma - Fox River Ex. (1/15 fish)	-	No incidence of carcinomas in controls up to 270 days post-exposure; one incidence of lymphoma after 360 days of exposure.	Fabacher et al., 1991
Rainbow trout (embryos), *Oncorhynchus mykiss*	injection of sediment extract into yolk sac	Lab	Field Mixture[C]	Doses[D]: (Exp I) 0.006 g (Exp II) 0.012 g 0.006 g 0.003 g	1 year	Hepatic carcinomas (I) 8.9% (11/123) (II) 8.1% (12/148) 4.0% (5/148) 3.1% (2/65)	-	Note; PCBs also present sediment from Hamilton Harbour	Metcalfe et al 1988

[A] Buffalo River, NY; total no. PAHs measured = 13, total no. of carcinogenic PAHs = 6.
[B] Black River, OH. And Fox River, WI; full compliment of measured PAHs.
[C] Hamilton Harbor, ON, Canada; total no. PAHs measured = 13, total no. of carcinogenic PAHs = 6.
[D] Doses are calculated as gram equivalent wet weight of sediment represented by the volume of extract micro-injected into each trout sac-fry.