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EXAMPLE APPROACH FOR THE DEVELOPMENT OF SITE-SPECIFIC PRELIMINARY REMEDIATION GOALS FOR PROTECTION OF ECOLOGICAL AND HUMAN HEALTH AT NAVY AQUATIC SITES

Prepared by:

Science Applications International Corporation

Under Contract to:

Naval Facilities Engineering Service Center
Port Hueneme, Ca

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EXECUTIVE SUMMARY

There is presently a lack of established scientifically based procedures for the development of site-specific cleanup goals, here called Preliminary Remediation Goals (PRGs), for ecological and human receptors at aquatic sites. This document provides remedial project managers and technical support contractors involved in site remediation with an example approach for calculating site-specific PRGs.

This approach, implemented towards the end of the Remedial Investigation (RI) phase, uses the results from the human health risk assessment (HHRA) and ecological risk assessment (ERA) to establish threshold concentrations of sediment contaminants below which adverse effects on ecological and human receptors are not expected to occur. Once developed, the PRGs are used to support the remedial alternatives evaluation in the Feasibility Study (FS), in accordance with the requirements of the National Contingency Plan (NCP) and the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA).

The basic assumption of the PRG development approach for aquatic ecological receptors is that the concentrations of each chemical in the sediment, sediment pore water, surface water, and biotic components are in equilibrium. PRGs based on one of the ecosystem components (*e.g.*, sediment) can result in protection of all the components. PRGs for protection of terrestrial and human receptors from contaminant uptake from site media (e.g., shellfish tissue) are developed using conventional exposure models and are converted into sediment-based concentrations using bioaccumulation factors.

The proposed PRG development approach integrates various exposure pathways using a consistent and systematic seven-step process separated into two phases. In the derivation phase (steps 1-5), information from the risk assessments is used to determine the "Limiting" Contaminants of Concern (L-CoCs) and to calculate protective concentrations (PRGs). In the implementation phase (steps 6-7), site specific conditions and the practicality of the PRGs are considered in the analysis for supporting reduction of identified risks and Applicable or Relevant and Appropriate (ARAR) compliance.

This approach has been adopted by Northern Division Naval Facilities Engineering Command and has been demonstrated at several sites in U.S. EPA Region 1. Additional information on the approach is available from the Northern Division environmental risk assessment team.

TABLE OF CONTENTS

1. INTRODUCTION 1

1.1. Objectives 1
1.2. Background 2
1.3. Case Study Risk Assessment Summary 4
1.4. Organization of this Report 7

2. PRG DERIVATION 8

2.1. Aquatic PRG Derivation 9
2.1.1. Aquatic Threshold Effects Value Derivation Approach 9
2.1.2. Aquatic Case Study Example 19
2.2. Avian Predator PRG Derivation 22
2.2.1. Avian Predator Threshold Effects Value Derivation Approach 22
2.2.2. Avian Predator Case Study Example 24
2.3. Human Health PRG Derivation 25
2.3.1. Human Health Threshold Effects Value Derivation Approach 25
2.3.2. Human Health Case Study Example 29

3. PRG IMPLEMENTATION 30

3.1. Translation of Threshold Effects Values into PRGs 30
3.1.1. Example Aquatic PRGs Calculation 30
3.1.2. Example Avian Predator/Human Health PRGs Calculation 32
3.1.3. Case Study Results 33
3.2. Example Approach for Spatial Implementation of PRGs 33
3.3. Assessment of PRGs for Risk Reduction 34
3.3.1. General PRG Assessment Approach 34
3.3.2. Case Study Aquatic PRG Assessment 35
3.3.3. Case Study Avian Predator PRG Assessment 39
3.3.4. Case Study Human Health PRG Assessment. 39
3.4. Overall Summary 40

4. CONCLUSIONS/RECOMMENDATIONS 42

5. LITERATURE CITED 43

LIST OF FIGURES

Figure 1.3-1. Derecktor Shipyard case study area.

Figure 2.1-1. Water quality screening value selection process and associated data qualifiers.

Figure 2.1-2. Equilibrium partitioning relationships for CoCs among environmental media.

Figure 3.2-1. Thiessen Polygons for PRG implementation for the Derecktor Shipyard/Coddington Cove study area.

Figure 3.2-2. Risk Probability for the Derecktor Shipyard/Coddington Cove study area.

Figure 3.3-1. Summary of CoCs exceeding PRGs for protection of aquatic biota by location for sediments in the Derecktor Shipyard/Coddington Cove study area: Bedded Sediment Exposure Pathway for HMW PAHs.

Figure 3.3-2. Summary of CoCs exceeding PRGs for protection of aquatic biota by location for sediments in the Derecktor Shipyard/Coddington Cove study area: Bedded Sediment Exposure Pathway for Total PCBs.

Figure 3.3-3. Summary of CoCs exceeding aquatic PRGs by location for sediments from the Derecktor Shipyard/Coddington Cove study area: Recommended PRG Implementation for the Bedded Sediment Exposure Pathway.

Figure 3.3-4. Summary of CoCs exceeding PRGs for protection of aquatic biota by location for sediments in the Derecktor Shipyard/Coddington Cove study area: Resuspended Sediment Exposure Pathway for Copper.

Figure 3.3-5. Summary of CoCs exceeding PRGs for protection of aquatic biota by location for sediments in the Derecktor Shipyard/Coddington Cove study area: Resuspended Sediment Exposure Pathway for Lead.

Figure 3.3-6. Summary of CoCs exceeding PRGs for protection of aquatic biota by location for sediments in the Derecktor Shipyard/Coddington Cove study area: Resuspended Sediment Exposure Pathway for Total PCBs.

Figure 3.3-7. Summary of CoCs exceeding aquatic PRGs by location for sediments from the Derecktor Shipyard/Coddington Cove study area: Recommended PRG Implementation for the Resuspended Sediment Exposure Pathway.

Figure 3.3-8. Summary of CoCs exceeding PRGs for protection of avian predators by location for sediments from the Derecktor Shipyard/Coddington Cove study area: Avian Predator Exposure Pathway for Zinc.

Figure 3.3-9. Summary of CoCs exceeding PRGs for protection of avian predators by location for sediments from the Derecktor Shipyard/Coddington Cove study area: Avian Predator Exposure Pathway for Total PCBs.

Figure 3.3.10. Summary of CoCs exceeding PRGs for protection of human health by location for sediments from the Derecktor Shipyard/Coddington Cove study area: Human Health Exposure Pathway for Benzo(a)pyrene.

Figure 3.3-11. Summary of CoCs exceeding human health PRGs by location for sediments from the Derecktor Shipyard/Coddington Cove study area: Recommended PRG Implementation for the Human Health Exposure Pathway.

LIST OF TABLES

Table 1.2-1. Potential Remedial Action Objectives for the case study area.

Table 1.2-2. Chemical-specific ARARs for the case study area.

Table 1.3-1. Overall summary of exposure- and effects-based weights of evidence and characterization of risk for the marine ecological risk assessment of the case study area.

Table 2.0-1. Generic procedure for Preliminary Remediation Goals (PRGs) development for aquatic, avian predator and human health exposure pathways in the case study area.

Table 2.1-1. Water Quality Criteria for target analytes selected for aquatic PRG development and derived Water Quality Screening Values.

Table 2.1-2. Summary of partitioning coefficients used in calculations of organic contaminant concentrations in pore waters by equilibrium partitioning.

Table 2.1-3. Distribution of toxic and non-toxic aquatic Hazard Quotients and derivation of No Effect Quotients for the aquatic receptors exposed to analytes via bedded and resuspended sediment exposure pathways for the case study area.

Table 2.1-4. Derivation of Threshold Effects Values for the aquatic exposure pathway in the case study area.

Table 2.1-5. Summary of bedded and resuspended pathway Hazard Quotients and identification of "limiting" CoCs for the aquatic exposure pathway in the case study area.

Table 2.2-1. Documentation of Toxicity Reference Values used for calculation of risks for the avian predator exposure pathway in the case study area.

Table 2.2-2. Derivation of Threshold Effects Values for the avian predator exposure pathway in the case study area.

Table 2.2-3. Summary of Threshold Effects Value Hazard Quotients and identification of "Limiting" CoCs for the avian predator exposure pathway in the case study area.

Table 2.3-1. Summary of Exposure Parameter Values used in estimating exposures via shellfish consumption by recreational fishermen.

Table 2.3-2. Summary of Risk Based Values used for the calculation of risks for the human health exposure pathway in the case study area.

Table 2.3-3. Derivation of Threshold Effects Values for the human health exposure pathway in the case study area.

Table 3.1-1. Summary of baseline Preliminary Remediation Goals (PRGs) for aquatic, avian predator and human health exposure pathways for the case study area.

Table 3.1-2. Summary of baseline and recommended Preliminary Remediation Goals (PRGs) for aquatic, avian predator, and human health exposure for the case study area expressed as sediment concentration-based values.

LIST OF EXHIBITS

Exhibit 1A. Calculation of pore water concentration for organic contaminants using Equilibrium Partitioning.

Exhibit 1B. Example calculation of pore water hazard quotients for aquatic biota exposed to CoCs in bedded sediments.

Exhibit 2. Calculation of the 95% UCL and MAX HQ_{PW} statistics from collocated chemistry/toxicity data.

Exhibit 3. Derivation of No Effects Quotients for the aquatic exposure pathway.

Exhibit 4. Derivation of Threshold Effects Values for the aquatic exposure pathway.

Exhibit 5. "Limiting" CoC selection process for the aquatic exposure pathway.

Exhibit 6. Calculation of baseline Preliminary Remediation Goals for the aquatic exposure pathway.

Exhibit 7. Preliminary Remediation Goal-Hazard Quotients for implementation for the aquatic exposure pathway.

LIST OF ACRONYMS

ACL	*Ambient Concentration Limit*
AET	Apparent Effects Threshold
ARAR	Applicable or Relevant and Appropriate
AVS	Acid Volatile Sulfide
AWQC	Ambient Water Quality Criteria
BAF	Bioaccumulation Factor
BCF	Bioconcentration Factor
BSAF	Biota-Sediment Accumulation Factor
BW	Body Weight
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
CoC	Chemical of Concern
EF	Exposure Factor
ELU	Elutriate
EMAP	Environmental Monitoring and Assessment Program
EPA	Environmental Protection Agency
EqP	*Equilibrium Partitioning*
ER-L	Effects Range Low
ER-M	Effects Range Medium
ERA	Ecological Risk Assessment
FA	Freshwater Acute
FC	Freshwater Chronic
FCR	Food Consumption Rate
FF	Feeding Fraction
FFA	Federal Facility Agreement
FS	Feasibility Study
HHRA	Human Health Risk Assessment
HI	Hazard Index
HMW	High Molecular Weight
HQ	Hazard Quotient
Koc	Organic carbon partition coefficient
Kow	Octanol-water partition coefficient
L-CoC	Limiting Chemical of Concern
MCL	*Maximum Concentration Limit*
MF	Migratory Factor
NA	*Not Available*
NR	*Not Recommended*
NOAA	National Oceanic and Atmospheric Administration
NOEC	No Effects Concentration
NOEQ	*No Effects Quotient*
OSWER	*Office of Solid Waste Enforcement Regulations*
PAH	Polycyclic Aromatic Hydrocarbon

LIST OF ACRONYMS (Continued)

PCB	Polychlorinated Biphenyl
PEL	*Permissible Exposure Level*
PRG	Preliminary Remediation Goal
PW	Pore water
RAOs	Remedial Action Objectives
RBV	Risk Based Value
RCRA	*Resource Conservation and Recovery Act*
RfD	Reference Dose
RIDEM	*Rhode Island Department of Environmental Management*
RIDOH	*Rhode Island Department of Health*
RIGL	*Rhode Island General Law*
RME	Reasonable Maximum Exposure
RoC	Receptor of Concern
RPRG	*Recommended Preliminary Remediation Goal*
RSV	*Reference Screening Value*
SA	Saltwater Acute
SC	Saltwater Chronic
SD	Standard Deviation
SDWA	Safe Drinking Water Act
SEM	Simultaneously Extractable Metals
SF	Slope Factor
SQC	Sediment Quality Criteria
SQRT	Square Root
TBC	To Be Considered
TCLP	*Toxic Concentration Leachate Procedure*
TEV	Threshold Effects Value
TRV	Toxicity Reference Value
TOC	Total Organic Carbon
TSCA	*Toxic Substances Control Act*
UCL	Upper Confidence Limit
WQC	Water Quality Criteria
WQS	*Water Quality Standard*
WQSV	Water Quality Screening Value

Note: The above acronyms listed in italics are used in exhibits, figures or tables only.

1. INTRODUCTION

Preliminary Remediation Goals (PRGs) are a list of Chemicals of Concern (CoCs) and associated concentrations that represent safe levels for ecological and/or human receptors within a given chemical exposure pathway. When PRGs are implemented as part of a remedial action involving contaminated sediments, the sediment CoC concentrations are reduced to below PRG levels by dredging, capping, or alternative treatment technologies.

1.1. OBJECTIVES

There is presently a lack of established procedures for developing site-specific PRGs, particularly for aquatic ecological receptors. To address human health risks, for example, the typical approach to develop clean-up goals is the re-arrangement and back-calculation of safe concentrations from the risk analysis equations. However, this process does not integrate background concentrations and does not include an evaluation of the extent of habitat disruption in relation to the extent of risk to be addressed. Also, for ecological risks based on a weight-of-evidence approach, such equations do not exist, and variable responses among the lines-of-evidence introduce uncertainty in the relationship between risk and the chemical exposure concentration.

Here, a PRG development process is presented that demonstrates how to evaluate and integrate multiple lines of evidence, exposure pathways and associated uncertainties to derive site-specific cleanup criteria to address all site-related risks. The document is intended to provide project managers and technical support staff involved in aquatic site remediation with an example approach for calculating site-specific PRGs. The ecological and human health PRGs developed in this report are not intended for the purpose of screening a site. Rather, they represent site-specific, risk-based concentrations that can be used to delineate sediment hot spots and cleanup volumes as refined in a Feasibility Study (FS) or an Engineering Evaluation/Cost Analysis (EE/CA).

Specific objectives for this site-specific PRG development process are aimed to accomplish the following:

- Derive initial, site-specific remediation goals (*i.e.*, cleanup levels) using data collected from the risk assessment; and
- Refine the calculated cleanup levels based on the magnitude and extent of observed risk and Applicable or Relevant and Appropriate (ARAR) compliance.

It must be emphasized that the calculated PRGs are not meant to be used to determine the final spatial extent of remedial action. This would be determined by the selection of final remediation goals by risk managers and results of a pre-design survey.

In this document, the PRG development approach is demonstrated through example calculations and a case study implemented at a Navy facility. While it is hoped that this example has relevance to a wide array of Navy site applications, it may not apply to every Navy site.

1.2. BACKGROUND

Experience with the implementation of this PRG development process within U.S. EPA Region I has yielded a list of "frequently asked questions" that are common to all sites. These questions with answers are provided below.

What guidance exists that pertains to site-specific Remediation Goal development? The available guidance for development of human health PRGs, contained in *the Risk Assessment Guidance for Superfund: Volume 1 - Human Health Evaluation Manual, Part B, Development of Risk-based Preliminary Remediation Goals* (U.S. EPA, 1991a), is not site-specific. Similarly, for ecological receptors, general guidance is presented in *Preliminary Remediation Goals for Ecological Endpoints* (Efroymson *et al.*, 1996). Again, this document also does not incorporate site-specific environmental conditions.

The recent document *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments* (U.S. EPA, 1997a) takes an important step forward in requiring that a key output of the risk characterization be *"contamination concentrations that bound the threshold for estimated adverse ecological effects..."* However, this EPA guidance does not include specific examples of such calculations. The case study presented here meets this need by illustrating how such calculations may be performed for different ecological exposure pathways (*e.g.*, aquatic, avian predator, human health), as well as how to spatially integrate PRGs developed for protection of human health.

What is the relationship between these PRGs and the risk assessments? The example approach described in this document does not supercede or replace the role that the ERA or HHRA plays in determining whether some "actionable" risk exists at the site. Rather, PRG development is initiated only when the results of the assessments indicate actionable risk. In addition, this approach does not require the collection of new data if the ERA/HHRA studies are reasonably complete.

What is the relationship between these PRGs and remediation goals? When risk managers reach a finding of actionable risk, remediation options must be developed and evaluated as part of the Feasibility Study (FS). The example PRG process and resulting site-specific PRG concentrations presented in this document are an integral part of a site's FS. In an FS, nine criteria are used to evaluate proposed remediation options. Two threshold criteria (overall protection of human health and the environment, and compliance with ARARs) and one of five "balancing" criteria (reduction of toxicity) that are used to evaluate the Remedial Action Objectives (RAOs) are directly applicable to PRG selection. The other balancing criteria (long-term effectiveness and permanence; reduction of toxicity, mobility, or volume through treatment; short-term effectiveness; implementability and cost) are also evaluated in the FS and directly affect the acceptability of various remedial alternatives. For this reason, PRGs generated by this procedure do not necessarily represent absolute levels of contaminants that must be removed from the site. Rather, it is the balance between the degree of risk reduction achieved, feasibility and cost that is the determining factor in selecting the final chemically based remedial goals documented in the Record of Decision (ROD) for the site.

Can the PRG approach be applied to any site? Navy facilities often have various chemical contamination issues related to landfills, shipyards, firing ranges and numerous other sources. As part of the remedial investigation, surveys have typically been conducted to determine the type and extent of CoCs in soil, groundwater, and offshore sediment and shellfish, including assessment of associated risks to the environment and human health. The PRG approach is broadly applicable to a wide variety of sites and data types, particularly where actionable risk is identified for a number of exposure pathways.

Do PRGs developed by this process meet regulatory requirements (e.g., ARARS)? The process presented herein is designed to support the evaluation of remedial alternatives in accordance with the requirements of the National Contingency Plan (NCP) and Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) guidance. As discussed above, it is assumed that if a need for PRGs exists, then investigations have been performed *a priori* that reveal elevated ecological and/or human health risks due to site-related chemical exposure. Under the Navy's Installation Restoration (IR) Program, these findings would result in the preparation of a FS for the site, the purpose of which is to outline options for remedial actions to address the chemicals causing risk.

Remedy options are generally evaluated by their effectiveness in meeting the objectives for mitigation of existing and potential threats to public health and the environment. These objectives are based upon the knowledge of the types of CoCs, the environmental media (*e.g.,* soil, water, and sediment) in which they are found or could be found in the future, and the projected use of the site. Therefore, the remedy must provide a mechanism to comply with the Federal and State ARARs or To-Be-Considered (TBC) standards listed as RAOs for the site.

Typical RAOs such as those adopted for this case study are listed in Table 1.2-1. In most cases, the measure of compliance is that residual CoC concentrations do not remain at concentrations above the RAOs. PRGs must comply with RAOs and may be modified based on feasibility and cost (among other factors) before becoming part of the remediation goals for the site. Thus, it is noted that these site-specific PRG values may differ significantly from generic PRGs published for use as screening values to include or exclude a site that is under consideration for remedial investigation.

How do these PRGs relate to CoCs identified during the risk assessment? The CoCs and associated concentrations which constitute the PRGs are supposed to be risk-based, *i.e.*, reflective of the results of the risk assessment with respect to the selection of those CoCs that "limit" remediation (U.S. EPA, 1991a). Like a nutrient that controls the growth of a plant because of limited supply, "Limiting" CoCs are those analytes that control the extent of risk because of high concentrations and/or enhanced exposure. While a risk assessment may identify dozens of chemicals that exceed threshold toxicity benchmarks, typically the chemicals having the greatest exceedences determine the level of risk. By remediating these CoCs to below risk-causing concentrations, collocated CoCs present at lower concentrations (relative to effect levels) will be proportionally reduced and therefore remediated as well. It is for this select group of CoCs or "Limiting" CoCs, that PRGs are developed, monitored, and evaluated for compliance with the RAOs.

Does the implementation of these PRGs address site-related chemical risks? An underlying assumption of the "Limiting" CoC approach is that implementing a PRG for a chemical causing the highest risk will lead to reduction of lesser risks caused by other CoCs. Those CoCs that are incorporated in the PRGs are assumed to adequately represent the risks posed by all site-related CoCs. This assumption would be flawed if there existed novel chemicals at high concentrations that have not yet been detected or are present in a form that is more bioavailable than has been previously measured. This, however, is a limitation of the risk assessment, not the PRG process. In addition, the "Limiting" CoC approach will be effective only when various chemical contaminants and exposure pathways remain collocated when removed from the sampling location. "Dislocation" of CoCs from one another might arise from application of treatment technologies that preferentially remove one CoC class over another (e.g., chemical specific bioremediation). If such a practice is instituted, then the available data must be re-evaluated for each CoC class and exposure pathway to ensure all receptors are adequately protected.

What kind of data do you need in order to develop these PRGs? Ideally, data needs would be determined during the Problem Formulation phase of the baseline HHRA and ERA in order to have sufficient information to implement the PRG evaluation. For aquatic sites, exposure to semi-aquatic mammals, seabirds and human receptors may occur from incidental ingestion of surface water and sediment, or from consumption of fish and shellfish containing site-related CoCs. In this case, the applicable chemical-specific ARARs/TBCs usually include U.S. EPA Water Quality Criteria and proposed U.S. EPA Sediment Quality Criteria, State-mandated sediment and soil criteria, U.S. FDA action limits for fish/shellfish consumption and tolerance ranges for carcinogenic risks for certain CoCs (Table 1.2-2). In this instance, the primary data needs (in addition to the risk conclusions) would be measurements of:

- CoC concentrations in sediment and pore water (particularly metals);
- Organic carbon content and particle size of sediment;
- Collocated sediment toxicity tests; and
- Collocated tissue residues and lipid content of biota.

Additional data needs include the exposure parameters used for modeling CoC partitioning between sediment and pore water, aquatic receptor bioaccumulation factors, as well as human shellfish consumption exposure factors. In many cases, these parameters are reported in the ERA/HHRA studies; but for sake of completeness the calculation methods are provided in this document (for example, while the HHRA calculates risks, the safe fish/shellfish concentrations are not typically reported). Again, all the necessary information for development of aquatic, terrestrial and human health PRGs should be available from the risk assessments.

1.3. CASE STUDY RISK ASSESSMENT SUMMARY

The case study selected for the PRG development demonstration is Derecktor Shipyard, which is part of the Naval Station Newport (NSN) and Coddington Cove in Rhode Island (Figure 1.3-1) where risks to aquatic biota, avian receptors and human health were identified (SAIC and URI, 1997; TTNUS, 1998). The ERA/HHRA for the case study was conducted in accordance with the U.S. EPA's ERA framework, EPA Region I guidance, and the ERA

Guidance for Superfund, Vol. 2, Environmental Evaluation Manual (1989b). The study was conducted prior to publication of the Eight Step process contained in the Ecological Risk Assessment process for Superfund (U.S. EPA, 1997a), therefore some overlap may be apparent in some of the steps. These guidance documents were used to generate and interpret data required to complete the assessments. The need for PRGs is the result of a finding of actionable risks. This section summarizes the risk assessment findings leading to the development of site-specific PRGs.

Sediment and tissue samples collected in 1993 and 1994 as part of a prior site characterization were examined for chemical concentrations of contaminants. These findings led to the conduct of a full baseline ERA investigation. The study objective was to assess ecological and human health risks to offshore environments of Narragansett Bay from chemical stressors associated with Derecktor Shipyard; these data supported risk management decisions regarding site-specific remedial options. Problem Formulation was conducted to determine the nature and extent of possible contamination of offshore media associated with Derecktor Shipyard sources. Specifically, this activity involved the identification of CoCs and potentially contaminated media, evaluation of the spatial extent of contamination, identification of the ecological receptors potentially at risk from CoCs, and identification of appropriate assessment and measurement endpoints. The assessment endpoints included the vitality of the pelagic community (via measurements from deployed blue mussels, fish), epibenthic community (via measurements of indigenous blue mussels, lobster, winter flounder, benthic community), infaunal community (via measurements on hard clams, benthic community), and endpoints representing pelagic and avian predators.

In accordance with established data quality objectives, a sampling plan was devised to provide better assessment of the chemical exposure to biological populations in surficial sediments adjacent to the site, potential migration of contaminants to adjacent embayments, and ecological risks to endemic populations (biotic receptors) in Narragansett Bay. Seventeen sampling stations located in Coddington Cove, both immediately adjacent to and in the wider area surrounding Derecktor Shipyard, were selected. Reference stations were located in similar habitat but away from site influences. These reference stations represented up-gradient ("upstream") and down-gradient ("downstream") positions along the regional chemical concentration gradient in Narragansett Bay.

Data collection occurred primarily during the summer months, since field conditions were considered most biologically dynamic and potentially stressed during this time. Side-scan sonar, sub-bottom profiling sonar, and sediment core surveys were undertaken to determine the characteristics of both surface and underlying sediments within the Derecktor Shipyard/ Coddington Cove study area. This combination of techniques provided more complete information than could be obtained by a limited number of surface and core samples. In addition, surveys to measure current velocity and water column profiles of conductivity, temperature, and depth were undertaken to determine patterns of water circulation within the study area. These water column data were used to assist in the selection of sampling locations.

Multiple lines of evidence were identified to provide a sufficient basis to support risk assessment and risk management decisions. These included sediment organic and inorganic

chemical analysis, elutriate (resuspended sediment) analysis, and extraction of sediment pore water for metals analysis. In addition, samples of blue mussels (*Mytilus edulis*), hard clams (*Mercenaria mercenaria*), cunner fish (*Tautogolabrus adspersus*), and American lobster (*Homarus americanus*) were collected and analyzed to characterize both contaminant exposure and effects on these receptors. Caged blue mussels were deployed at selected locations to assess pelagic exposure pathways for contaminants. Tissue residue data were used to assess food chain transfer of CoCs to avian aquatic predators and recreational shellfishermen.

Site-specific evaluations of sediment toxicity were conducted using the 10-day amphipod (*Ampelisca abdita*) mortality test. For sediment elutriates, the sea urchin (*Arbacia punctulata*) fertilization and larval development tests were used. Evaluations of effects on aquatic biota included assessments of infaunal and epifaunal benthic community structure, biotic condition of indigenous and deployed bivalves, hematopoietic neoplasia in bivalves, fecal pollution indicators in bivalves, and cytochrome P450 activity in cunner.

The interpretation of ecological risk in the case study was based on a weight of evidence approach. Measures of exposure and effects were first evaluated independently. The exposure-based weights of evidence included:

- Comparison of sediment concentrations with commonly used benchmarks (*e.g.,* NOAA Effect Range-Low and Effect Range-Median (ER-L/ER-M; Long and Morgan, 1990; Long *et al.*, 1995),
- Divalent metal exposure measures (Simultaneously Extracted Metal; Acid Volatile Sulfides (DiToro *et al.*, 1991), and
- Comparison of site and reference shellfish tissue concentrations for each target receptor.

Effects-based measures included comparison of:

- Tissue concentrations against effects-based benchmarks,
- Laboratory toxicity test results,
- Field measurements (benthic community structure, bivalve condition and neoplasia, P450 activity in cunner, and fecal pollution indicators in bivalve tissue) and
- Exposure to avian aquatic receptors from ingestion of contaminated prey.

The findings of exposure and effects weights of evidence were evaluated jointly for the strength of exposure-response relationships to interpret the probability of adverse ecological risks at each station (Table 1.3-1). A scoring approach allowed results to be grouped into four primary classes (baseline, low, intermediate, and high risk). Conclusions and recommendations were presented in this form to provide a range of considerations for risk management.

High ecological risk was designated at a given sampling station when numerous weights of evidence suggested adverse exposure/effects and demonstrable exposure-response relationships were observed. Two of 18 stations were categorized as high-risk stations (Table 1.3-1). The high concentrations of contaminants in the sediments suggested that risks were likely to persist over a long period of time. The spatial extent of apparent impact might be limited, as nearby stations exhibited fewer and lower levels of risk.

Intermediate ecological risks were assigned to nine stations (Table 1.3-1). Indication of CoC exposure at these stations was most evident from comparison of tissue concentrations to reference station values, while effects indicators included tissue residue effects, toxicity in the laboratory tests, and observed effects on natural biota. However, clear exposure-response relationships were generally not evident at these stations, resulting in intermediate risk being assigned.

A low risk probability was assigned to eight of 18 stations and one reference station (Table 1.3-1). For these stations, the data suggested low risks based on the majority of exposure and effects-based weights of evidence, and no exposure-response relationships were observed. Finally, a baseline risk was assigned to the one remaining reference location. Here, exceedences of sediment benchmarks were not observed, and laboratory and field effects were not readily apparent.

Based on the above information indicating a finding of actionable risk for the site, a FS was deemed necessary including the development of PRGs. Results of PRG development for the site serve as the basis for the case study example presented in this report. Aspects of derivation and implementation procedures for PRGs are presented below in Sections 2 and 3, respectively.

1.4. ORGANIZATION OF THIS REPORT

The example PRG approach presented in this document integrates various exposure pathways using a consistent and systematic process separated into a derivation phase and implementation phase. In the derivation phase (Section 2), a five-step process is used to refine the list of CoCs and determine protective concentrations for various exposure pathways, here generically called threshold effects values (TEVs). In the implementation phase (Section 3), two additional steps are employed to convert the threshold effect values into sediment-based units (termed “baseline” PRGs), and to evaluate the baseline PRGs with respect to site-specificity and practicality for supporting risk reduction and ARAR compliance. This latter evaluation leads to selection of recommended PRGs for the site. For each of the seven steps, the generic approach and rationale is presented along with example calculations, and subsequently, case study results are provided. Finally, in Section 4, conclusions and recommendations for application of the approach to Navy sites in other EPA regions are discussed based on experience with the process at EPA Region 1 sites.

2. PRG DERIVATION

PRG derivation procedures for aquatic, avian and human health pathways share a common element: the determination of a site-specific threshold effects value. The threshold effects value is the media-specific concentration of a chemical of concern that can be used to discriminate between an exposure that is unlikely to result in biological effects and those concentrations that have been associated with adverse effects at the site. The principal difference between the threshold effects value and a PRG is that the former is expressed in the exposure pathway concentration units (e.g., sediment pore water for aquatic biota, shellfish tissue concentrations for avian and human health consumption), while the latter is expressed in sediment concentration units to be used during remediation or monitoring. Hence, the main requirement for PRG derivation is determination of the threshold effects value that relates to multiple biological receptors.

The five PRG derivation steps that lead to determining the list of CoCs and associated threshold effects values for use in PRG implementation (Section 3) are shown in Table 2.0-1. These steps are generically identified as follows:

- Consolidation of literature-based benchmarks into a single, pathway-specific list of "no effects concentrations" and application to site data for estimating hazard;
- Review of site-specific conditions and refinement of chemical exposure assumptions;
- Review of ERA/HHRA CoC list and modification based on refined exposure assumptions and predicted chemical hazards;
- Refinement of pathway-specific no effects concentrations to account for background CoC levels (called threshold effects values); and
- Assessment of CoC exceedences of threshold effects values to identify pathway-specific "Limiting" CoCs for PRG selection.

Each of the above steps is often a component of ERA/HHRA investigations for determination of risks. However, there is a wide range of approaches in how these steps are performed and applied, particularly since the majority of existing ERA studies were initiated prior to formalization of the recent ERA eight-step guidance. Thus, the emphasis here is to establish procedures that lead to consistency in the PRG development approach. Ultimately, the degree of review, refinement and recalculation of data obtained from the ERA/HHRA will only depend upon the degree of consistency with the PRG derivation approach recommended here. As the recommended procedures are implemented within the risk assessments, the need for these steps (and the PRG derivation process in general) will become obsolete.

While the generic steps in PRG derivation are similar across pathways, the details and data types for specific pathways differ sufficiently such that they are discussed separately in Section 2.1 (aquatic), Section 2.2 (avian) and Section 2.3 (human health) below. To facilitate the presentation, for each pathway the general approach and technical background for derivation is presented first, followed by a case study example using data from an ecological and human health assessment performed for a Navy shipyard.

2.1. AQUATIC PRG DERIVATION

As discussed above, the procedures for aquatic PRG derivation is provided below in two sections. The general approach and technical background for derivation is presented in Section 2.1.1, while in Section 2.1.2, a case study example for PRG derivation is presented using data from an ERA performed for a Navy shipyard.

2.1.1. Aquatic Threshold Effects Value Derivation Approach

Step 1. Benchmark consolidation and hazard calculation. Benchmarks are derived from media-specific concentrations that represent a range of measured concentrations for a given chemical where biological effects have and have not been observed. A variety of benchmarks are available for sediment, water and tissue residues and should be obtainable from the risk assessment, however not all chemicals have benchmarks. For purposes of PRG derivation, there is the need for an array of benchmarks that is as complete as possible in providing the most consistent levels of protection. In many cases, this requires the conversion of a benchmark developed for one medium (e.g., sediment) into equivalent concentrations for another medium (e.g., water or tissue). Step 1 of the process is to assemble a cohesive set of benchmarks that here are generically called no effect concentrations. *It is important to note that these no effect concentrations do not directly represent PRGs for the site. Rather, they represent the beginning step in the process towards derivation of the PRGs.*

For the aquatic exposure pathway, chemical concentrations in pore water are used as the primary indicator of exposure in sediments, whereas the concentrations in the water of sediment slurries (called elutriates) are assumed to represent chemical exposure of resuspended sediments. Hence, the no effect concentrations for the aquatic pathway are water-based and are best obtained from Water Quality Criteria. However, Water Quality Criteria values are not available or directly applicable in many cases, such that alternate benchmarks must be used. This process of selecting and converting alternate benchmarks into the aquatic-based no effect concentrations needed for PRG derivation is discussed below.

*Benchmark consolidation***.** As discussed above, contaminant exposure for the aquatic exposure pathway is based on the concentration of contaminants in the water of bedded (*i.e.*, pore water) and resuspended (*i.e.*, elutriate) sediments. This decision is based on the fact that Water Quality Criteria are, for the most part, the only available aquatic benchmarks that are derived from single chemical dose-response relationships. Most sediment benchmarks, in contrast, are derived from field data sets where multiple chemicals are present in the sample where effect measurements were made and hence the reliability of the effect concentration is reduced. Water Quality Criteria for surface waters are common ARARs for aquatic environments and therefore this approach ensures that the PRGs will support ARAR compliance.

For purposes of the example case study presented below, values equivalent to a Water Quality Criteria - Saltwater Chronic, i.e., low-level effect concentrations, values were assumed to represent the no effect concentration. Wide ranges of Water Quality Criteria values are available, requiring careful evaluation prior to selection (Table 2.1-1).

A number of important contaminants do not have promulgated Water Quality Criteria-saltwater chronic values, requiring use of alternative water-based contaminant criteria following the decision tree logic presented in Figure 2.1-1. This approach allows for calculation of "Water Quality Criteria- saltwater chronic equivalent" benchmarks, and assigns a data qualifier to identify the benchmark source. The data qualifier "A" is applied to benchmarks derived directly from existing Water Quality Criteria- saltwater chronic values (Table 2.1-1; under "Source" heading). For contaminants having Water Quality Criteria-saltwater acute (WQC-SA) values, the equivalent Water Quality Criteria- saltwater chronic value (WQC-SC) was derived by dividing the WQC-SA value by eight (data qualifier = "B"). The conversion factor was derived from the mean overall acute effect: chronic effect ratio for paired chemical data contained in the U.S. EPA AQUIRE database (Shepard, 1998). AQUIRE is a compilation of published data on effects of chemicals to aquatic organisms. Freshwater chronic data are used directly as screening values, with assigned data qualifier "C." As with WQC-SA values, Water Quality Criteria-Freshwater Acute values (WQC-FA) were converted to chronic values by dividing by 8 and assigned a data qualifier of "D".

Research by the U.S. EPA into the development of sediment quality criteria for divalent metals (Cu, Cd, Pb, Ni, and Zn) has shown that sediment toxicity can be predicted when the quantity of Simultaneously Extractable Metal (SEM) present is in excess of the Acid Volatile Sulfide (AVS) concentration (Berry *et al.*, 1996). Analogous to equilibrium partitioning of organic contaminants, metals preferentially bind to sediment in the presence of AVS (i.e., more AVS means less metal diffused into surrounding water). The expression of SEM relative to AVS has been historically expressed as the SEM/AVS ratio, although the difference of SEM and AVS (SEM-AVS) is now the preferred convention. The ratio metric is considered less sensitive when AVS is near detection limits (*e.g.*, resulting in very high SEM/AVS ratios). The use of SEM-AVS calculation is based on the fact that AVS will bind divalent metals in direct proportion to their respective molar concentrations (Hansen *et al.*, 1996).

In the U.S. EPA National Sediment Quality Inventory (U.S. EPA, 1996a), the SEM-AVS value of 5 µmol/g dry wt is recommended as the screening benchmarks for potential sediment toxicity. It should be noted that the SEM-AVS method is not directly amenable to PRG development since the SEM calculation is not metal-specific and does not directly assist in identification of contaminant-specific PRGs. However, the data are useful for the validation of the derived values as appropriate site-specific PRGs.

Sediment-based correlative benchmarks are derived to complete the assessment of site-related contaminants where Water Quality Criteria benchmarks are lacking. For the example case study presented here, Effects Range-Low (ER-L) bulk sediment concentrations (Long *et al.*, 1995) were selected and translated into pore water equivalent concentrations (data qualifier = "E") using an equilibrium partitioning model (discussed below). Whereas the previous benchmarks used to derive the water quality screening values (WQSVs) are based on effects observed in laboratory exposures, NOAA sediment effects database contains sediment concentrations at which biological effects were observed in both laboratory and field studies. Still, they appear to afford a comparable level of protection; the water quality criteria are expected to protect 95% of species from sub-lethal or chronic impacts, while the ER-L is defined as the 10th percentile value where 90% of the reported effects occur at higher concentrations.

Thus in the PRG development process, it is assumed that using an ER-L as a basis for derivation of water quality screening values will provide a level of protection similar to other water quality based benchmarks.

Benchmark Derivation based on equilibrium partitioning. EPA's Sediment Quality Criteria Program has demonstrated the general applicability of Water Quality Criteria for prediction of sediment toxicity when partitioning characteristics of the chemical between water and the sediment are taken into account (Figure 2.1-2). This is accomplished by using the equilibrium partitioning model of DiToro *et al.* (1991):

1) $$C_{PW} = C_{Sed} / (foc * Koc)$$

where C_{PW} is the pore water concentration, C_{Sed} is the sediment concentration, f_{oc} is the fraction of organic carbon in dry sediment, and K_{oc} is the organic carbon/water partitioning coefficient. In Equation 1, organic chemical pore water concentrations (in µg/L) are calculated from the corresponding sediment concentration (in µg/kg) divided by the product of the fraction of organic carbon in the site sediment (as %TOC/100) and the organic carbon/water partitioning coefficient for the contaminant (in L/kg).

Values of K_{oc} for typical organic contaminants are listed in Table 2.1-2. Example calculations of pore water concentrations are presented in Exhibit 1A. The K_{oc} describes the relative affinity of the contaminant to associate (attach) to organic carbon or partition (diffuse) into the surrounding water. Thus, a K_{oc} of 5.6 for benzo(a)anthracene (from Table 2.1-2) means that this chemical is partitioned such that 398,107 ($\log_{10}$ 5.6) times more chemical is found bound with the organic matter/sediment matrix than in surrounding water. Values for K_{oc} are determined from the octanol/water partition coefficient (K_{ow}) using the relationship developed by the U.S. EPA (Karickhoff, 1989):

2) $$\log_{10} K_{oc} = 0.00028 + 0.983 * \log_{10} K_{ow}$$

Because the equilibrium partitioning model permits the calculation of the chemical concentration in pore water from the sediment concentration, the appropriate sediment benchmark (e.g.., the ER-L) can be converted into a water-based value. This value is assumed to afford a comparable level of protection selected for other chemicals (i.e., the Water Quality Criteria - Saltwater Chronic value). For example, in the PRG case study, the anthracene ER-L (85.3 ng/g) is 0.29 µg/L when converted into a water-based concentration using the model.

Exhibit 1A. Calculation of porewater concentration for organic contaminants using Equilibrium Partitioning.

EqP model for organics:
$C_p = C_s/(f_{oc} * K_{oc})$ (DiToro *et al.*, 1991)

Pathway	Station	Contaminant	Cs	foc	Koc	Cp
Aquatic Bedded	DSY-40	Benzo(a)anthracene (H)	234	0.0148	1.01E+05	0.04
		Benzo(a)pyrene (H)	317	"	1.01E+06	0.02
		Benzo(e)pyrene (H)	333	"	1.01E+06	0.02
		Chrysene (H)	444	"	4.01E+05	0.07
		Dibenz(a,h)anthracene (H)	52.7	"	3.77E+06	9.52E-04
		Fluoranthene (H)	779	"	1.08E+05	0.49
		Pyrene (H)	1190	"	1.06E+05	0.76
		HMW PAHs			Sum:	1.41

(H) – High Molecular Weight PAH analyte
C_p - organic chemical porewater concentrations (µg/L)
C_s - sediment concentration (µg/kg dry weight)
f_{oc} - fraction of organic carbon (%TOC/100)
K_{oc} - organic carbon/water partitioning coefficient; derived from $\log_{10}K_{ow}$
($\log_{10}K_{oc} = 0.00028 + 0.983*\log_{10}K_{ow}$); where K_{ow} = the octanol/water partition coefficient

As demonstrated in Exhibit 1A, chemical-specific field measurements of sediment concentrations can also be converted into pore water concentrations for comparison against actual or calculated no effect concentrations. The model incorporates site-specific conditions in the form of organic carbon content measured as total organic carbon (TOC) of sediment because TOC controls sediment-pore water partitioning and hence chemical exposure. These data should be available from the risk assessment. For individual metals, similar effective partitioning models do not exist. Therefore, direct measurement of metals in pore water as part of the risk assessment is recommended.

***Hazard Normalization*.** Using the no effect concentration data[1] and location-specific concentrations of CoCs in pore water, pore water hazard quotients are calculated to assess the degree of chemical exposure to aquatic biota associated with bedded sediments. This step normalizes, as a ratio, the inherent hazard of the contaminants so that the relative risks of each contaminant can be compared.

Example calculations are shown in Exhibit 1B. The results demonstrate the calculation of individual analyte hazard quotients as well as the sum of hazard quotients for high molecular weight PAHs. While hazard quotients exceeding unity suggest a possible role of the chemical in

1. In the case study example, the no effect concentration data for the bedded sediment and resuspended sediment aquatic exposure pathways were called Water Quality Screening Values (WQSV).

contributing to adverse effects to aquatic biota living in bedded sediment, the hazard quotient data are carried forward into Step 2, below, to assess whether observed exceedences were associated with measured toxicity in field samples taken from the site.

Exhibit 1B. Example calculation of pore water hazard quotients for aquatic biota exposed to CoCs in bedded sediments.

Pathway	Station	CoC	C_P	WQSV[1]	HQ_{PW}[2]
Aquatic Bedded	DSY-40	Total PCBs	2.39E-03	0.03	0.08
		Anthracene (L)[3]	0.54	0.29	1.87
		Benzo(a)anthracene (H)	0.04	0.07	0.61
		Benzo(a)pyrene (H)	0.02	0.04	0.50
		Chrysene (H)	0.08	0.10	0.79
		Dibenz(a,h)anthracene (H)	9.52E-04	1.70E-03	0.57
		Fluoranthene (H)	0.49	16.00	0.03
		Pyrene (H)	0.77	0.63	1.22
		HMW PAHs[4]	-	-	3.72

1 - WQSV = Water Quality Screening Value
2 - HQ_{PW} = Pore Water Hazard Quotient (unitless; C_P/WQSV)
3 - (L) = Low Molecular Weight PAH analyte
4 - HMW PAH value = sum of High Molecular Weight PAH analyte HQ_{PW}s.

Depending on the pathways evaluated in the ERA, effects related to sediments that may be subject to resuspension can be of concern as was the case for the present ERA. Hence, chemical concentrations were also measured in sediment elutriates and used to assess chemical exposure due to resuspended sediment[2]. As with the pore water data, elutriate hazard quotients are calculated as the concentration measured in sediment elutriates divided by the respective no effect concentration.

Step 2. Evaluate analyte exposure under site-specific conditions. For each analyte, site-specific factors may exist that modify the degree of chemical exposure to target receptors. For example, site-specific factors related to the bound form of the analyte in the environment (*e.g.*, some analytes present as paint chips, scrap metal, sand blast material, etc.) may make the true analyte exposure to aquatic receptors less than that predicted directly by bulk sediment or even water-based benchmarks. Hence, the second step in the PRG development process is to

2. Elutriate chemical concentrations are typically measured in waters that have been mixed with sediments in a three part water to one part sediment ratio, allowing one hour for settling.

calculate under site-specific conditions the highest degree of departure from the literature-based benchmark that can allowed for a given chemical without an adverse effect being likely to occur. This value, calculated from the site-specific data, is called the no effects concentration.

The approach used to determine the site-specific no effects concentration was adopted from correlative benchmark approaches (e.g., NOAA Effects Range values), where the goal is to establish statistical confidence around thresholds of sediment concentrations that are collocated with measured biological effects. For example, the ER-L benchmark was developed by matching of chemical concentrations with incidence of benthic effects (*e.g.*, toxicity, reduced benthic composition, biomarker response) measured in field samples or laboratory studies (Long and Morgan, 1990; Long *et al.*, 1995). For the case study ERA, the primary indicator for site-specific analyte exposure in bedded sediment was derived from results of the amphipod (*Ampelisca abdita*) 10-day solid-phase bulk sediment toxicity test. For resuspended sediments, results from the sea urchin (*Arbacia punctulata*) fertilization and larval development elutriate tests were used. An amphipod or sea urchin toxicity test was conducted at each location where a bulk sediment or elutriate chemical sample was collected, respectively. In the sea urchin tests, a series of elutriate dilutions are tested to determine the elutriate concentration causing 10% reduction in the endpoint (i.e., IC_{10}).

Once the paired toxicity-chemistry data sets are assembled, the hazard-normalized data (from Step 2) are segregated into non-toxic and toxic datasets as determined in the ERA investigation[3]. It is important to note that any set of similar endpoints could be used for this purpose and different sites are likely in fact to have conducted tests with other species and life stages. For the case study, for example, a sample size of 17 matched chemistry/toxicity data pairs for the amphipod was available (a subset of the data is shown in Exhibit 2 to demo this concept). Statistics are generated independently for the toxic and non-toxic data sets: Calculation of the mean and 95% upper confidence limit (UCL) of non-toxic pore water hazard quotients is needed for Step 3, and the maximum pore water hazard quotient value is used in Step 4, discussed below.

3. In the case study example, toxicity was defined as follows: amphipod survival $\geq$ 80% = non-toxic; and sea urchin fertilization/larval development $IC_{10} \geq 50\%$ = non-toxic.

Exhibit 2. Calculation of the 95% UCL and maximum HQ_{PW} statistics from collocated chemistry/toxicity data.

1 - HMW PAH value represents the sum of six HMW PAH analyte HQPW values.
2 - Only a subset of stations is shown to demonstrate the concept.
3 - 95% UCL = Upper Confidence Limit (unit-less; calculated from t-test statistical analysis)
4 - Where n≤3, the non-toxic maximum HQ_{PW} is used instead of the 95% UCL due to small sample size.

HMW PAHs	Bulk Sediment Non-Toxic to Amphipods	Bulk Sediment Toxic to Amphipods
Station[2]	HQ_{PW}[1]	HQ_{PW}
DSY-25	2.98	-
DSY-26	4.53	-
DSY-27	-	3.41
DSY-28	-	1.25
DSY-29	6.07	-
DSY-40	3.72	-
DSY-41	0.34	-
Sample size (n)	15	2
Mean HQ_{PW}	1.89	-
95% UCL[3]	2.87	-
Max HQ_{PW}[4]	-	3.41

Step 3. Retain analytes likely to substantially contribute to risk at the site. An objective of PRG derivation is to identify and retain only analytes for which PRG implementation will lead to effective risk reduction at the site. Thus, it is assumed that if an analyte is a substantial risk contributor, the concentration associated with toxic samples must be greater than the non-toxic samples (represented by the no effect concentration). This assumption holds whether the data are expressed in concentration units (*e.g.*, ng/g) or concentrations relative to benchmarks *(e.g.,* hazard quotients).

In Step 3, the test-specific no effects concentration (hazard-normalized) is taken either as the 95% upper confidence limit or unity (test NOEQ = 1). Test NOEQ values less than one are not adopted because it is considered unlikely that site-specific factors could increase toxicity of the analyte to levels above that represented by the existing benchmarks (e.g., Ambient Water Quality Criteria for metals, ER-L equivalents for organics). In the case of Total PCBs, for example, the selection of the default value is justified based on the assumption that a lack of data exists in the upper end of the non-toxic exposure range to fully characterize the sample distribution (Exhibit 3).

Exhibit 3. Derivation of the No Effect Quotient (NOEQ) for the aquatic exposure pathway.

•The NOEQ is the highest departure from the HQ = 1 equivalent concentration (i.e., WQSV) for which site-specific adverse effects are unlikely to occur.

•Only those CoCs for which the maximum HQ_{PW} of toxic samples exceeds the NOEQ are retained for further PRG development.

	Nontoxic Samples			Toxic Samples			
Analyte	n	95% UCL HQ_{PW}	Test NOEQ	n	Max PW-HQ	Max PW-HQ>NOEQ?	Path NOEQ
Anthracene	15	1.38	1.38	2	1.21	NO	
HMW PAHs	15	2.87	2.87	2	3.41	YES	2.85
Total PCBs	15	0.11	1.00	2	1.78	YES	1.00

For 95% UCL values < 1, a test NOEQ = 1 is adopted.

To complete Step 3, the maximum toxic hazard quotient and no effect quotient are compared for each analyte. All analytes satisfying the requirement where the maximum toxic hazard quotient is greater than the no effect quotient are retained for further consideration as PRGs. In the example, anthracene did not meet this criterion and thus was not retained for further PRG development (Exhibit 3). This criterion was met for HMW PAHs and Total PCBs; hence these CoCs are carried forward into Step 4 for further development as potential PRGs.

Step 4. Comparison of protective concentrations with background. Because of the general exchange of water and sediment of an aquatic site, it must be assumed that it would not be technically feasible in the long term to remediate the site to concentrations lower than that generally found in the region. Hence, the regional CoC concentrations are determined to ensure that the pathway-specific PRG will be greater than or equal to the regional concentration estimate. Here, this concentration is generically identified as the "background value" such that the method for derivation can be exactly defined within the PRG development procedure and therefore eliminate confusion with soil-based methods for determining background that may or may not apply to sediments.

The best source of background data is from reference locations used in the ERA investigation. Depending on the scope of the ERA, however, sufficient data may not be available and additional locations may be needed to include as wide a spatial coverage as possible. Should this be required it is necessary to show that the environments of reference locations among various studies are comparable to the study area. This may be accomplished by

inspection of geotechnical data (*e.g.*, sediment particle size and organic carbon content) and demonstrating that the additional locations are within the range observed for stations in the study area.

Once the reference locations have been selected, the reference database can be populated with the chemical data. In the case study example, the reference database for the aquatic exposure pathways includes both pore water and elutriate concentrations, as discussed in Step 1. At present, the background value is based on the upper 95% confidence range of available background data. In cases where the availability of reference data is insufficient to calculate the upper confidence limit (*e.g.*, $n \leq 3$), the maximum value is used in lieu of the limit. This value is intended to ensure that regional contamination will not compromise meeting the remediation goals. As discussed above, this particular method may differ from other procedures used to estimate background, and if another procedure has been agreed upon that is applicable to sediment, it should be used.

As a last procedure in Step 4, the threshold effects value is determined. The goal of this process is to establish a threshold effect value for each chemical that will not be lower than the upper range of background concentrations. First, the no effect quotient is converted into the equivalent no effect concentration by multiplying by the water quality screening value (Exhibit 4). Subsequently, the greater of the no effect concentration and the background value is taken as the threshold effect value. The process is repeated for each CoC and pathway where a no effect quotient was previously retained in Step 3.

Exhibit 4. Derivation of Threshold Effects Values (TEVs) for the aquatic exposure pathway.

- The NOEQ values are converted into the no effects concentration (*e.g.*, water concentration units)
- The reference screening value is calculated as the 95% UCL of reference contaminant concentrations after outlier removal;
- The greater of the no effects concentration and reference screening value is taken as the threshold effects value.

Pathway	CoC	NOEQ[1] *	WQSV[2] =	NOEC[3]	RSV[4]	TEV[5]
AQ-PW[6]	HMW PAHs	2.87	0.29	0.83	0.18	0.83
	Total PCBs	1.00	0.03	0.03	1.96E-04	0.03
AQ-ELU[7]	Arsenic	1.12	36.0	40.3	18.3	40.3
	Copper	1.00	2.90	2.90	1.25	2.90
	Lead	1.06	8.50	9.01	13.2	13.2
	HMW PAHs	7.32	0.29	2.12	0.21	2.12
	Total PCBs	2.31	0.03	0.07	0.05	0.07
	o,p'-DDE	2.8	1.00E-03	2.80E-03	3.59E-03	3.59E-03

1 - NOEQ = No Effect Quotient (unit-less)
2 - WQSV = Water Quality Screening Value (µg/L)
3 - NOEC = No Observable Effect Concentration (µg/L)
4 - RSV = Reference Screening Value (µg/L)
5 - TEV = Threshold Effects Value (µg/L)
6 - AQ-PW = Aquatic Pore Water (Bedded sediment)
7 - AQ-ELU = Aquatic Elutriate (Resuspended sediment)

In the example, it can be seen that the threshold effect value for lead and o,p'-DDE are determined by the background value; all others are based on the site-specific no effect concentration. These CoCs and associated threshold effects values are brought forward into Step 5 discussed below.

Step 5. Assess CoC exceedences of threshold effects values to identify "limiting," pathway-specific CoCs for PRG selection. The CoCs and associated concentrations to be used as PRGs are intended to be risk-based, *i.e.*, reflective of the results of the risk assessment with respect to the selection of those CoCs that "limit" remediation (U.S. EPA, 1991a). In the case study, limiting CoCs were typically those analytes that were responsible for much of the baseline risk (because of high concentrations and/or strong correlation with high toxicity). By remediating for these limiting CoCs to their PRG concentrations, it follows that collocated CoCs will also be remediated to levels much lower than their corresponding goals.

In Step 5, Hazard Quotients based on threshold effects value data are calculated by dividing the site pore water concentrations by the respective threshold effects values. These data are then inter-compared for each station to determine the CoC causing maximum risk at each station (Exhibit 5). This CoC is designated as the "Limiting CoC" and is retained for further PRG development.

Exhibit 5. Example of "Limiting" CoC selection process for the aquatic exposure pathway.

- HQ_{TEV} values among analytes are compared within each station.
- PW $HQ_{TEV} = C_P/TEV$.
- CoC with the Maximum $HQ_{TEV} > 1$ at each station selected as a "limiting" CoC.
- CoC also selected at each station where Sum $HQ_{TEV} > 1$.

Pathway	CoC	PW HQ_{TEV} Station			
		DSY-25	DSY-27	DSY-28	DSY-40
AQ-PW	HMW PAHs	0.95	1.13	0.42	1.80
	Total PCBs	0.15	1.78	0.06	0.08
	Sum HQ_{TEV}	1.09	2.91	0.48	1.88
	Max HQ_{TEV}	0.95	1.87	-	1.80
	"Limiting" CoC	HMW PAHs	Total PCBs	-	HMW PAHs

As an additional procedure in Step 5, the analyte with the maximum threshold effects value hazard quotient for the station is also retained whenever the sum of threshold effect value HQs is greater than unity (e.g., DSY-25, Exhibit 5). This step is taken to further address the uncertainty in the collocation assumption by identifying any CoC that might substantially contribute to risk at the site. Note that application of this latter procedure should only be performed for chemicals that have similar modes of toxic action. In the case study data set, the identified CoCs (PCBs, PAHs, pesticides, selected metals) have a common mode of toxic action, called narcosis (McCarty and McKay, 1993). Hence for the purposes of the present study, it was determined that it was appropriate to sum the threshold effect value HQs across chemicals. However, the appropriateness of this step for other Navy sites with different CoCs and modes of toxic action should be assessed.

The above process is repeated for all sampled locations to identify the collection of all possible limiting CoCs for PRG implementation. It should be noted that the extent to which the relative mixtures of CoC reflect the site as a whole might have an effect on whether all the necessary limiting CoCs are identified. If the number of stations is low, and it is likely that unsampled areas would indicate that other chemicals are also important risk drivers, then the process will not identify the proper limiting CoCs. Still, if such a condition did exist, the certainty of the ERA/HHRA risk prediction would be equally affected. Thus it is assumed for purposes of PRG development that a sufficient number of stations have been sampled to characterize the risks and thus important CoCs will not be overlooked.

2.1.2. Aquatic Case Study Example

Procedures leading to the identification of limiting CoCs in Steps 1 - 5 above were applied to the case study data set, as discussed below.

Step 1. In Step 1, the literature-based benchmarks representing no effect concentrations are obtained from the ERA or are otherwise derived as shown in Figure 2.1-1. Results are presented in Table 2.1-1. In the case study presented in this document, the aquatic pathways to be addressed included both bedded (*i.e.*, in-place) and resuspended sediment effects on aquatic biota. The measurement endpoints representing chemical exposure by these pathways are the sediment pore water and sediment elutriate concentrations, respectively. Lacking water-based benchmarks for several of the organic chemicals, water-equivalent concentrations were calculated using the equilibrium partitioning model discussed in Section 2.1.1, which rely upon knowledge of chemical specific partitioning coefficients (Table 2.1-2). Of course, the exposure pathways being addressed by PRGs are determined by the risk assessments and hence appropriate benchmarks representing other pathways of concern may be required. Also, the specific details of the model and preferred partitioning constants may vary depending on the site and therefore a consultation with the regulatory community on these aspects should be sought.

Step 2. In Step 2, hazard quotients values associated with non-toxic and toxic samples are calculated for each exposure pathway. For the bedded sediment exposure pathway in the present study, hazard quotient values were calculated for chemicals found in the pore water of bedded sediment samples and the data were segregated into toxic and non-toxic data sets based on measured survival of amphipods. Similarly, for the resuspended sediment exposure pathway in the present study, hazard quotient values were calculated for chemicals found in the water of

sediment elutriate samples and the data were segregated into toxic and non-toxic data sets based on measured fertilization and larval development success of sea urchins. The same approach can be applied for other pathways and receptors applicable to other Navy sites as long as the toxicity and chemistry data come from the same sample.

For the bedded sediment exposure pathway in the present study, the lack of individual pore water metal data precluded the calculation of hazard quotients for this chemical class. Also, the measurement of organic concentrations in pore water was not practical given the high sample volume requirements (~1 L) for chemical analyses. Hence, the pore water organic concentrations were predicted from the measured sediment concentrations and the sample total organic carbon concentration (using the same equilibrium model and coefficients for benchmark derivation discussed in Section 2.1.1, above). Ideally, direct measurements of pore water concentrations should be always be used where possible; high volume pore water extraction methods are becoming more readily available to meet this need.

In contrast to the bedded sediment pathway, the resuspended sediment pathway assessment relies upon chemistry data obtained from elutriate preparations (made by proportional mixing of sediment and site water). In this case, adequate sample volumes are easily obtainable, and hence in the present study, both metals and organic chemical concentrations were measured in the elutriate samples. Also, unlike the modeling approach of predicting pore water concentrations in sediment, a comparable modeling approach for similar predictions of chemicals in sediment elutriates is not available. Thus, making direct measurements of elutriate water is particularly necessary for assessing the importance of chemical exposures for this pathway.

Summary results of the hazard quotient data for each exposure pathway and toxicity result are presented in Table 2.1-3. For non-toxic samples, the 95% UCL hazard quotient values reflect the highest chemical concentration (normalized to the benchmark) for which no effects should be observed 95% of the time. For toxic sample, the value simply reflects the maximum observed hazard quotient. As indicated by the sample numbers, the majority (15 of 17) of amphipod samples for the bedded sediment exposure pathway were toxic (Table 2.1-3a), while for the resuspended sediment exposure pathway, elutriates were non-toxic to sea urchin fertilization but were frequently toxic (70-80%) to sea urchin larval development (Table 2.1-3b). The comparison of non-toxic and toxic hazard quotient data is discussed in Step 3, below.

Step 3. In Step 3, CoCs are retained for further PRG derivation when the maximum hazard quotient associated with toxic samples exceeds the no effect quotient (determined from non-toxic samples). Subsequently, the no effects quotient for the selected CoCs is taken as the largest of the 95% upper confidence limit of the non-toxic hazard quotient data for the exposure pathway and the default hazard quotient (*e.g.*, HQ=1). Finally, as discussed in Section 2.1.1, when the *analyte-specific* no effect quotient was less than 1, a default value of 1 was retained.

For the bedded sediment exposure pathway, the retained analytes included High Molecular Weight (HMW) PAHs and Total PCBs (Table 2.1-3a), while for resuspended sediments, the analytes included arsenic, copper, lead, HMW PAHs, Total PAHs, Total PCBs, and the pesticide o,p'-DDE (Table 2.1-3a).

For most analytes, the 95% upper confidence limit of the non-toxic hazard quotient was less than unity, indicating good agreement with the literature-based no effect concentration data. That is, toxicity was not observed where criteria values predict that toxicity should not occur. As shown in Table 2.1-3b, for example, the sea urchin response to sediment elutriate exposure correlated particularly well with calculated hazard quotients since for the nine analytes with hazard quotients greater than unity, all were associated with elutriate toxicity in at least some samples.

The results of Step 3 with the case study data indicate that toxicity was generally observed when hazard quotients exceeded unity. This finding confirms that the test species employed in the ERA were sensitive to chemicals when concentrations were above the screening values. For the analytes where the no effect quotient did exceed unity, this is attributed to site-specific conditions that have reduced CoC exposure relative to conditions under which the Water Quality Criteria are derived (*i.e.*, single-species, water-only laboratory bioassays).

Step 4. In Step 4, the background value is determined and then compared to the no effect concentration (from Step 3). As discussed previously, the purpose of this step is to ensure that the calculated threshold effect value will exceed "ambient" concentrations (defined as the 95% UCL of the mean background). Otherwise, the remediated area could be re-contaminated by off-site CoCs over time and therefore long-term clean up would be unattainable.

Background value calculation. In the example case study, an outlier screening procedure was instituted because of irresolvable uncertainty concerning some analytes at some of the reference locations being biased high because of potential point source pollution. These values, it was argued, would unreasonably elevate the calculated background concentration. Thus for the purposes of the present study, it was agreed that rather than excluding the reference location outright and therefore eliminate all the data, analyte concentrations greater than the mean plus 95% upper confidence limit of the reference data set would be excluded and the 95% upper limit recalculated to establish the background value. While this procedure was not ideal, the agreement did allow the PRG development process to move forward to meet the Federal Facilities Agreement (FFA) schedule without the need for a costly and lengthy background survey. Such a procedure may not be necessary or appropriate at other sites, particularly if a formal background study has been completed.

To complete Step 4, the calculated background values were compared to the no effect concentrations. For the case study, the no effect concentration for the majority of CoCs was found to be much greater than the background value (Table 2.1-4). This finding was reassuring since it indicated that the PRGs would be determined by risks at the site rather than background concentrations. Exceptions were noted for lead and o,p'-DDE, however, as the background value exceeded the no effects concentration. Thus, for these CoCs, the background value was selected as the threshold effects value, and these CoCs were retained in the process.

Step 5. Limiting CoC selection is performed in Step 5. Table 2.1-5 presents a summary of maximum threshold effects value hazard quotients observed by exposure media and station for the aquatic exposure pathway. Results show that among all the possible limiting CoC

candidates, only a small number had hazard quotients greater than unity. For the bedded sediment aquatic exposure pathway, the two limiting CoCs were HMW PAHs and Total PCBs. Arsenic, copper, lead, Total PCBs, and o,p'-DDE were identified as limiting CoCs for the resuspended sediment exposure pathway. These CoCs are brought forward into PRG implementation, discussed in Section 3.

2.2. AVIAN PREDATOR PRG DERIVATION

The site-specific PRG derivation approach for the avian predator exposure pathway presented in this section follow the same process as that documented for the aquatic exposure pathway (Section 2.1). In the derivation phase (Section 2), a five-step process is used to refine the list of CoCs and determine protective concentrations, called threshold effects values. For each of the steps, the generic approach and rationale is presented (focusing on the nuances specific for this pathway), and subsequently, case study results are provided.

2.2.1. Avian Predator Threshold Effects Value Derivation Approach

As identified in Table 2.0-1, steps required for avian predator PRG derivation include:

- Benchmark selection and expression of contaminant concentration data as hazard quotients;
- Evaluation of contaminant exposure under site specific conditions;
- Retention of analytes substantially contributing to avian predator risk at the site;
- Evaluation of the feasibility of the CoC and pathway-specific Toxicity Reference Value (TRV) as a long-term remediation goal; and
- Assessment of CoC exceedences of threshold effects values to identify limiting pathway-specific CoCs for PRG selection. These sequential steps are explained in the following sections.

Step 1. Benchmark consolidation and hazard calculation. As for the aquatic exposure pathway, the first step in the PRG derivation process is the identification of literature-based benchmarks applicable to an avian exposure pathway. As stated previously, the benchmarks should normally be available from the ERA investigation, but for the sake of consistency and completeness, the method of calculation is presented below.

Benchmark consolidation. The avian benchmarks found in the literature typically represent various acute and chronic effect concentrations based on dietary contaminant exposures to domesticated test organisms (Table 2.2-1). Where possible, aquatic bird test data are selected in preference to data for other bird species. The various test types (endpoints) and associated values (endpoint values) are converted into no effect concentrations by application of a safety factor that depends on the duration of the test. Finally, the receptor no effect concentration is obtained by scaling the test species no effect concentration to that of the receptor based on differences in body size according to the following equations of Sample *et al.*, (1996):

3) $$NOEC_{RoC} = NOEC_{test} * [BW_{test} / BW_{RoC}]^{1/3},$$

where $NOEC_{RoC}$ is the No Effect Concentration of the Receptor of Concern (mg CoC/kg bird/-day), BW_{RoC} is the body weight of receptor species (kg), BW_{test} = body weight of the test species (kg) and $NOEC_{test}$ is the experimental dose to test species representing the no effect concentration (mg CoC/kg bird-day).

In the ERA investigation, risks were identified for herring gulls caused by the consumption of CoCs in shellfish from the site. Intake of contaminants via other exposure routes such as water and sediment ingestion was determined to be minimal in comparison to intake via food ingestion. The next step is to determine the chemical concentration in prey that would result in a dose equivalent to the no effect concentration. This value is called the toxicity reference value, and is calculated by dividing the no effect concentration by the amount of food consumed by the receptor as follows:

4) $$TRV = NOEC_{RoC} / f$$

where the TRV is the toxicity reference value and *f* is the amount of food consumed per unit body weight per day (kg prey/kg bird/day). The food factor for the bird receptor is derived by normalizing the Food Consumption Rate (FCR; kg prey/day, dry wt) to the receptor body weight (BW; kg bird, dry wt):

5) $$f = FCR / BW_{RoC}$$

where the food consumption rate for the aquatic receptor is estimated from published allometric regression models. For example, the food consumption rate model of Nagy (1987) for the herring gull is as follows:

6) $$FCR = 0.495 * (BW_{RoC})^{0.704}$$

<u>*Hazard calculation*</u>. Given the receptor-specific toxicity reference values, hazard quotient values for avian predators are derived by dividing the prey species tissue concentration by the toxicity reference value. The resulting hazard quotients may be adjusted depending upon site specific factors as described in Step 2, below.

Step 2. Evaluate analyte exposure under site-specific conditions. As discussed for aquatic receptors, site-specific factors may exist that modify the degree of chemical exposure to target receptors. Exposure model parameters selected in the ERA to assess the degree of chemical exposure to the avian receptor are often conservative, and may not consider all possible site-specific factors. In the case study ERA, for example, the parameters used in the exposure model (Sample *et al.*, 1996)) were the site-specific foraging range factor (FR; fraction of range/area represented by the site relative to the total feeding area), migration factor (MF; fraction of the year bird is in the area) and the feeding fraction (FF, the contribution of the prey type to the total diet). These parameters can be adjusted to calculate an Exposure Factor (EF) as follows:

7) $EF = FR * MF * FF$

The exposure factor can in turn be multiplied against the prey species concentrations to refine either the chemical dose (mg CoC/kg bird/day) of CoC to the receptor (in Step 1, above) or alternatively, multiplied against the hazard quotients values derived in Step 2.

Step 3. Retain analytes likely to substantially contribute to risk at the site. Similar to the aquatic exposure pathway, the purpose of this step is to identify and retain those analytes for which PRG implementation will lead to effective risk reduction at the site. Unlike the aquatic approach, there is no measured site-specific toxicity response that can be used to assess localized chemical exposure conditions. Still, this step affords the opportunity to select more realistic exposure assumptions than might have been employed in the ERA (e.g., the receptor feeds exclusively from the site or station through its lifetime). This could lead to modification of the hazard quotients in Step 2, or alternatively, a more robust screening of CoCs (*e.g.*, retain only CoCs with HQ > 10) may be performed to further refine those analytes most important in contributing to risk. Whether such adjustments are needed or can be made will depend upon the conservatism of the original model assumptions and pragmatic management decisions based on evaluations of the probability of risks against the practicality of potential risk reduction.

Step 4. Comparison of protective concentrations with background. Following the general approach taken for the aquatic background value derivation, a reference database consisting of prey species tissue concentrations is developed for CoCs carried forward from Step 3 to derive the avian background value. The database assembly includes calculation of the mean + 95% UCL of the data after outlier removal.

The resulting avian predator background values are compared against toxicity reference values (see Exhibit 4), with the greater of the two taken as the avian threshold effects value. This procedure ensures that PRGs are not set to concentrations below regional background values, and therefore ensuring their practicality as remediation concentrations.

Step 5. Assess exceedences of Threshold Effects Values to identify "limiting," pathway-specific CoCs for PRG selection. Finally, in Step 5, the CoCs to be retained as limiting CoCs are derived by comparing the hazard quotients (based on the measured shellfish tissue concentration (Table 2.2-1) and selecting the CoC with the maximum value on a station-by-station basis (see Exhibit 5). As was performed for the aquatic pathway, a CoC is retained for the avian pathway at each station whenever the sample sum of hazard quotients exceeded unity (Table 2.2-1). Again, CoCs were summed only for chemicals that have similar modes of toxic action. The appropriateness of this step for other Navy sites with different CoCs should be assessed.

2.2.2. Avian Predator Case Study Example

Step 1-3. Literature-based TRVs (toxicity reference values) required as part of Step 1 were obtained from the ERA investigation (Table 2.2-1). The list of CoCs that were brought

forward from the ERA included eight metals as well as Total PCBs (Table 2.2-2). For purposes of the case study, no change in the exposure factor assumptions were made (Step 2), hence those CoCs identified in the ERA were retained as potentially important contributors to risk (Step 3).

Step 4. A background database of shellfish tissue was assembled from several ERA investigations conducted in the area. These data were inspected for potential outliers (none were found) and the 95% upper confidence limit of each chemical in the database was determined. To complete Step 4, the resulting background values were compared against the toxicity reference value, shown in Table 2.2-2. The greater of the two values was selected as the avian threshold effects value. The data comparison showed that the toxicity reference value was in all cases at least four-fold higher than the background value. This suggests that the threshold effects value represent obtainable remediation concentrations, if necessary.

Step 5. Finally, as part of Step 5, hazard quotients based on threshold effects values were inter-compared by CoC at each of the stations (Table 2.2-3). Maximum hazard quotient values exceeding unity was observed only for Total PCBs (but at several stations), while silver and zinc were also retained as limiting CoCs for PRG implementation because the sum hazard quotients also exceeded unity at several stations. It is noted again that CoCs are retained in the process even though chemical-specific hazard quotients were less than one in order to reduce the uncertainty about potential CoC concentrations for unsampled locations. This decision is revisited during PRG implementation (Section 3) where the effectiveness and practicality of selected CoCs is addressed.

2.3. HUMAN HEALTH PRG DERIVATION

The methodology for human health PRG derivation generally follows the same five step process as the previous PRG derivation approaches for the aquatic and avian predator exposure pathways, leading to site-specific protective concentrations, called threshold effects values. As before, the generic approach and rationale is presented and case study results are provided.

2.3.1. Human Health Threshold Effects Value Derivation Approach

PRGs for protection of human health are dependent upon exposure assumptions regarding both the vector of exposure (e.g., food, sediment, and water) and intended use of the site (e.g., subsistence, recreational, site-worker). While sources of PRGs for human health do include concentrations based on ARARs (*e.g.*, maximum contaminant level goals set under the Safe Drinking Water Act), the focus here is the derivation of PRGs based on concentration limits using carcinogenic and/or non-carcinogenic toxicity values under site-specific exposure conditions.

As identified in Table 2.0-1, steps required for human health PRG derivation include:

- Benchmark consolidation and CoC hazard calculation combined with evaluation of analyte exposure under site specific conditions (Steps 1-2);
- Retention of CoCs substantially contributing to human health risk at the site (Step 3);
- Evaluation of the feasibility of the CoC as a long-term remediation goal (Step 4); and

- Assessment of CoC exceedences of threshold effects values to identify pathway-specific limiting CoCs for PRG selection (Step 5).

Each of these procedures is separately addressed in the following sections.

Steps 1 and 2. Benchmark consolidation and hazard calculation; Evaluate analyte exposure under site-specific conditions. As with the ecological receptors, Hazard Quotients for human receptors are defined as the concentration in the exposure media divided by the media-specific threshold for adverse effects. Unlike the Steps 1 and 2 for ecological receptors, the process of benchmark derivation and exposure evaluation must be combined due to the nature of the human health models and effects endpoints.

The exposure pathway of concern for PRG derivation is identified in the human health risk assessment (HHRA). In the present example, the identified pathway was for contaminants in shellfish tissue residues consumed by recreational shellfishermen. Four indigenous species were characterized for PRG evaluation, including hard-shell clams (*Mercenaria mercenaria* and *Pitar morrhuana*), blue mussels (*Mytilus edulis*) and lobster (*Homarus americanus*). Both non-carcinogenic and carcinogenic chemicals, discussed in the following sections, caused risks.

Non-carcinogens. The non-carcinogenic effect benchmark is typically taken as the concentration in shellfish that is unlikely to cause adverse health effects, even in sensitive populations (U.S. EPA, 1991a). The risk-based shellfish concentration (C) representing a non-carcinogenic baseline hazard (Hazard Quotient=1) to humans from ingestion of contaminants follows U.S. EPA guidance (U.S. EPA, 1989a) as follows:

8) $$C_{Tis} = (HQ * RfD * BW * AT)/(IF * CF * FI * EF * ED * RAF)$$

Where:

C_{Tis}	=	Safe concentration in shellfish tissue (mg/kg)
RfD	=	Reference dose (acceptable daily intake level; mg CoC/kg-day^{-1})
HQ	=	Acceptable Hazard Quotient; ratio of average daily intake level to reference dose (unity)
BW	=	Body weight (kg)
AT	=	Averaging time (days)
IF	=	Intake factor (i.e., shellfish consumption rate, g/day)
CF	=	Conversion factor (1 kg/10^6 mg)
FI	=	Fraction ingested (i.e., fraction of shellfish ingested)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
RAF	=	Relative absorption factor (unit-less; analyte-specific)

Values for the majority of the exposure parameters should be found in the HHRA. Most commonly the parameters are not site-specific and are typically obtained directly from the U.S. EPA Standard Default Exposure Factors Handbook (U.S. EPA, 1993d; Table 2.3-1). In the absence of relevant site-specific data, it is also typically assumed that all of the shellfish consumption by local subsistence fishermen occurs in the vicinity of the site, but uncertainty

regarding this conservative assumption should be addressed when assessing the selection of PRGs for the site.

Values for the Reference Dose are chemical specific, and should be contained in the toxicity profiles provided in the HHRA. Similarly, values for the Relative Absorption Factor are receptor specific, and should also be contained in the toxicity profiles provided in the HHRA Finally, one additional factor that should be considered is the annual shellfish consumption rate of the regional population. In New England, a population survey of shellfish consumption showed that the 95th percentile of total shellfish consumption for adults in the range of 18 to 65 years of age was 15.6 g/day (Rupp *et al.*, 1980). This value ranges two-three fold across the U.S. and therefore will have substantial influence on the resulting PRGs; hence the values appropriate to the Navy site under investigation should be available in the site HHRA.

Taking the above into consideration and substituting the values in Table 2.3-1 for the reasonable maximum exposure (RME) scenario, Equation 8 reduces to:

9) $$C_{RME} = (4679.5 * RfD) / RAF$$

Procedures to calculate safe concentrations of carcinogenic chemicals are generally similar and discussed in the following section

Carcinogens. Carcinogenic risk is estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen (U.S. EPA, 1991a). This is based on U.S. EPA's interpretation of the significance of the cancer risk estimate as stated in the National Oil and Hazardous Substances Pollution Contingency Plan (40 CFR Part 300). The carcinogenic risk-based shellfish concentration benchmark, C_{Tis}, is described as follows:

10) $$C_{Tis} = (Risk * BW * AT) / (SF * IF * CF * FI * EF * ED * RAF)$$

Where:

Risk = Acceptable probability of an exposed individual developing cancer
SF = Cancer slope factor (mg/kg-day^{-1})

The remaining exposure parameters are as defined for the non-carcinogenic model. It can be seen that the two deviations are the risk and SF parameters, whereas previously the HQ and RfD were used for non-carcinogenic chemicals. Hence, Equation 10 shown above can be simplified to calculate safe shellfish tissue concentrations for carcinogens as follows:

11) $$C_{RME} = 0.011 / (SF * RAF)$$

Here, the equation assumes a reasonable maximum exposure and a 10^{-6} cancer risk level. The equation should be adjusted if necessary to use exposure parameters appropriate for the region where the Navy site is located.

Step 3. Retain analytes likely to substantially contribute to risk at the site. Using the reasonable maximum exposure benchmark values calculated from the above models, analytes found in environmental samples above the reasonable maximum exposure benchmark are retained for further PRG derivation. Typically, the HHRA has already performed this calculation in effect and identified those CoCs presenting possible cancer and non-cancer risks. In the present process, the lesser of the reasonable maximum exposure benchmarks for carcinogenic and non-carcinogenic risks is adopted as the "risk-based value". A more robust evaluation of the exposure assumptions (*e.g.*, 10^{-6} vs. 10^{-4} cancer risk assumption) and exposure parameters should be performed and updated to ensure reasonableness. This may afford the opportunity to correct for overly conservative assumptions in the risk assessment and incorporate the most recent literature values published since the HHRA was completed.

Step 4. Comparison of protective concentrations with background. Just as described for ecological receptors, the efficacy of the human health Risk-Based Values are further evaluated by comparison against human health background values. These reference values were derived from measured CoC concentrations in shellfish harvested from reference locations. This analysis was calculated in the same basic manner as for the avian receptor (*e.g.,* upper 95% of shellfish tissue concentrations after outliers were removed). However, consideration was given to only those species used for human consumption (some species used by avian receptors are not palatable to humans), as well as the form in which the food was ingested (*e.g.,* whole body vs. muscle).

Following the approaches used for aquatic and avian receptors, the human health threshold effects values were obtained by selecting the greater of the risk-based value and the background value for each CoC (following Exhibit 4). Note that the risk-based values compared against reference shellfish concentrations for the study area are in wet (*e.g.,* live) weight units. Hence, the % solids content (*i.e.*, g dry/g live wt) for tissue samples may be needed from the ERA/HHRA to permit conversion of the Risk-Based Values to dry weight units, as necessary.

Step 5. Assess CoC exceedences of Threshold Effects Values to identify pathway-specific limiting CoCs for PRG selection. The list of CoCs to be retained as limiting CoCs for human health are derived following the same procedures employed for the aquatic and avian predator exposure pathways. The threshold effect value hazard quotients (measured shellfish tissue concentration divided by the threshold effects value) are compared across CoCs by station with any CoC possessing the maximum hazard quotient for that station being selected as the limiting CoC (See Exhibit 5). Also as was done previously, a CoC is retained whenever the sum of the threshold effect value hazard quotients for the station was greater than one.

It should be noted that the final list of limiting CoCs would usually reflect the same CoCs identified in the HHRA, since the same models and input parameters are used. Should this not be the case, reasons for discrepancies with the HHRA findings should be noted. One case where this might occur is if the TEV value has been determined by the background concentration, and concentrations at the specific stations do not exceed background. Also, the site-specific parameter values may be updated during the PRG development process if sufficient information is available, and if agreements with the regulators can be obtained that the most conservative

approach (e.g., as typically employed in the HHRA) is not feasible or practical from a management perspective.

2.3.2. Human Health Case Study Example

Steps 1-2. Table 2.3-1 presents the reasonable maximum exposure benchmarks (reported as wet weight) for CoCs in shellfish tissue consumed by recreational shellfishermen in the case study area, following Steps 1 and 2, above. These benchmarks include "site-specific" factors, in this case being for example, the shellfish consumption practices of individuals likely to live near and frequent the site.

Step 3. Consistent with Step 3, the analyte list was reduced to only those CoCs likely to contribute substantially to risk (*i.e.*, CoCs with cancer risk > 10^{-6} or non-cancer HQ > 1). The risk-based values for CoCs in shellfish tissue are the minimum of the carcinogenic and non-carcinogenic reasonable maximum exposure values by CoC (reported in dry wt units). In the case study data set, good agreement in solids content for various species was observed, allowing use of the average of 14% solids content (*i.e.*, 86% water content) for conversion of wet weight values into dry weight concentrations. In all cases, risk-based values were based on carcinogenic risks. Therefore, risk-based values were calculated by dividing 14% or 0.14 into the cancer risk (*i.e.*, 1 X 10^{-6}).

Step 4. Following procedures outlined in Step 4, Risk-Based Values were compared with background values. With the exception of arsenic, the risk-based values were higher than the background values (Table 2.3-2).

Step 5. Finally, following Step 5, the comparison of CoC-specific Hazard Quotients by station indicated that arsenic and benzo(a)pyrene were the limiting CoCs for the human health pathway (Table 2.3-3).

3. PRG IMPLEMENTATION

The second phase of PRG development involves a qualitative assessment of the practicality for spatial implementation (*i.e.*, ensures the spatial implementation of the PRG effectively target areas of increased risk as identified in the ERA/HHRA reports). This requires that the Threshold Effects Values, which represent the site-specific no effect concentration (HQ = 1) for limiting CoCs, be translated into sediment-based units (*i.e.*, PRGs) (Step 6). In this regard, the calculated PRG values represent baseline (HQ = 1) risk thresholds for protection of ecological and human health. While not strictly required for PRG development, a graphical approach facilitating a spatial analysis of site concentrations is presented.

Subsequently, the baseline PRGs are "tested" through comparison against measured chemical concentrations at the site, such that the relationship between the areas of PRG exceedence and the degree of risk as concluded in the ERA and HHRA can be ascertained (Step 7). This analysis leads to the development of "recommended" PRGs, which are PRGs considered to be appropriate from a risk- and ARAR-based perspective (Section 3.3) based on best professional judgment. Finally, summary recommendations are made as to the use of the baseline and recommended PRGs for input into the Feasibility Study for the evaluation of remedial alternatives.

3.1. TRANSLATION OF THRESHOLD EFFECTS VALUES INTO PRGS

In Step 6 of the PRG development process, the Threshold Effects Values are recalculated as necessary into appropriate (sediment-based) concentration units to be implemented during site remediation. The methods and examples for this calculation are presented separately for the aquatic (Section 3.1.1) and the avian predator/human health exposure pathways (Section 3.1.2). Baseline PRG results for the ERA/HHRA case studies are presented in Section 3.1.3.

3.1.1. Example Aquatic PRGs Calculation

Step 6. Determine PRGs for limiting CoCs (i.e., express Threshold Effects Values in concentration-based units to be used during remediation). The primary intention of this calculation is to derive a PRG number that protects the receptor. Thus, it is expected that when the PRG is applied to measured sediment chemistry, a comparable degree of exceedence of the PRG will be observed as indicated by the matrix-specific risk indicator (*i.e.*, the hazard quotient based on threshold effects value data) as follows:

12) $$HQ_{PRG} = HQ_{TEV}$$

In this concept of "risk equivalency", it is assumed that risks associated with the matrix representative of ecological concern (pore water) are in equilibrium with risks associated with the matrix selected for remedial action (sediment). Hence, the magnitude of risk at a given location, when expressed as a unit-less quotient, should theoretically be the same regardless of whether the measurement of exposure is pore water-based or sediment-based. Thus, a sediment pore water concentration that is two-fold above the threshold effects value hazard quotient (*e.g.*, $HQ_{TEV} = 2$), should also have a corresponding PRG concentration that will reduce the risk by a

factor of two when implemented (*e.g.*, from $HQ_{PRG} = 2$ to a $HQ_{PRG} < 1$).

Following this assumption, the relationship described in Equation 12, above, can be used to solve for the PRG. Substituting for HQ_{PRG} :

13) $$HQ_{TEV} = C_{Sed} / PRG ,$$

and given the HQ_{TEV} and associated sediment concentration (C_S) at each location, the baseline PRG concentration is determined as follows:

14) $$PRG = \frac{1}{n} (C_{Sed} / HQ_{TEV}) / N ,$$

where N is the number of locations at the site.

From Equation 14, it is seen that the mean of station- and CoC-specific PRG estimates are used to derive the site-wide baseline (HQ = 1) PRG concentration. An example of this calculation is presented in Exhibit 6.

Exhibit 6.
Calculation of Baseline Preliminary Remediation Goals for the aquatic exposure pathway.

The TEV values are translated into sediment based units (i.e., baseline PRGs). All available sediment data at the site can be considered with respect to PRG compliance and risk reduction.

Pathway	Station	"Limiting" CoC	Cs	HQ_{TEV}[1]	Cs/HQ_{TEV}
Aquatic Bedded	DSY-25	HMW PAHs	4065	0.95	4298
	DSY-26	HMW PAHs	4939	1.50	3300
	DSY-27	HMW PAHs	9818	1.13	8714
	DSY-40	HMW PAHs	4613	1.80	2558
	DSY-41	HMW PAHs	241	0.15	1604
$PRG = \Sigma(C_S/HQ_{TEV})/n$				Sum:	105459
				n[2]:	17
				Baseline PRG[3]:	6204

1 - HQTEV = Threshold Effect Value-Hazard Quotient (unitless); 2 - n = Number of Observations; only a respresentative sample of stations are shown. 3 - PRG = Preliminary Remediation Goal (µg/kg)

An important feature of this threshold effects value-to-PRG translation method is that the measured risk data are used to derive the PRG value, whereas the equilibrium models were used to calculate the pore water-based exposure concentration PRGs. For example, data at

Station DSY-27 shows that high TOC content can result in low pore water concentrations and accordingly low TEV-HQ values despite the high sediment concentration. In some cases, an estimated PRG may fall outside the expected range of the values particularly if characteristics of sample imply non-equilibrium conditions (*e.g.*, low TOC content of sediment, inert CoC materials such as metal fragments). In these instances, the predicted values can be validated against model estimates and the cause for atypical (high or low) PRG values can be isolated.

3.1.2. Example Avian Predator/Human Health PRGs Calculation

The translation of both avian predator and human health PRGs require the conversion of tissue-based threshold effects values to sediment-based concentrations. For metals, the method of translation involves the calculation of site- and CoC-specific Bioaccumulation Factors (BAF_{CoC}), as follows:

15) $$BAF_{CoC} = \frac{1}{n}(C_{Tis} / C_{Sed}) / N$$

where C_{Tis} and C_{Sed} are the dry weight CoC concentrations in organism tissue and sediment, respectively, and N is the number of samples.

BAF values for metals tend to be site-specific and should be obtained or calculated from data in the ERA report. In the example data set, BAF values for arsenic (0.875), copper (0.33), lead (5.0E-6), silver (6.0E-4), and zinc (1.05) were determined. Subsequently, the PRG metals baseline in µg/g dry wt is calculated from the tissue-based threshold effects value concentration (TEV_{CoC}, µg/g dry tissue) according to the formula:

16) $$PRG_{Baseline} = \frac{1}{n}(TEV_{CoC} / BAF_{CoC}) / N$$

For organic CoCs, the Biota-Sediment Accumulation Factor (BSAF) is determined by the formula:

17) $$BSAF = \frac{1}{n}([C_{Tis} / Lipid_{Tis}] / [C_{Sed} / TOC_{Sed}] / N)$$

where the chemical concentrations (ng/g dry wt) and concentrations of tissue lipid ($Lipid_{Tis}$, %) and TOC in the sediment (TOC_{Sed}, %) employed in the equation are species- and station-specific.

The site-specific BSAF values for PCBs, PAHs and pesticides may be available from the ERA. Our experience suggests that the site-specific values compare well with literature BSAF values reported by U.S. EPA (U.S. EPA, 1997b) and do not differ substantially by analyte or species as long as receptors are sediment-associated (Tracey and Hansen, 1996). Therefore, the following equation applies:

18) $$PRG_{Baseline} = \frac{1}{n}((TOC_{Sed} * TEV / Lipid_{Tis}]) / BSAF) / n$$

Given class-specific BSAF values, the above calculation uses the mean sediment TOC and the mean biota lipid. These values or supporting data should be available from the ERA investigation. In the example data set, BSAFs for PAHs, PCBs and pesticides were 0.12, 5.0, and 3.85, respectively.

3.1.3. Case Study Results

Table 3.1-1 presents a summary of baseline (*e.g.*, HQ = 1) PRGs for the aquatic (bedded and resuspended), avian predator and human health exposure pathways using the procedures described in the above section. All values are reported as dry weight concentrations in sediment. Note that PRG concentrations are calculated for only limiting CoCs (*i.e.*, those CoCs identified as having the maximum hazard quotient based on the threshold effects value data by station for the pathway).

The Baseline PRGs that should theoretically represent values that address all of the site-related risk. However, there are uncertainties in the process not least of which is the fact that the risk assessment may be based on multiple lines of evidence, all of which are not quantitatively incorporated in the PRG calculation. Hence, the next step is to spatially compare the PRGs to all available sediment chemistry as well as the results of the risk assessment to determine where and the extent to which site-related risks would be addressed.

3.2. EXAMPLE APPROACH FOR SPATIAL IMPLEMENTATION OF PRGS

Implementation of PRGs to the spatial extent of potential remedial action requires that the CoC data obtained from point samples be extrapolated to non-sampled locations. Numerous methods for spatial extrapolation of point data to larger areas (such as contouring) have been developed for various environments and sampling strategies where the assumptions of continuity (*e.g.,* constant CoC dilution with distance) and gradation (*e.g.,* regular spacing of sampling locations) are met. In the case of site remediation, however, these assumptions are often not met due to heterogeneous CoC distributions and station clustering in focused areas. Hence, a method was developed for PRG implementation that does not require the presumption of continuity in the data (ESRI, 1989). This approach involves the use of Thiessen polygons and was modeled, in part, after the U.S. EPA EMAP Demonstration Study for the Virginian Province (Weisberg *et al.*, 1993). The Thiessen polygon technique involves the creation of irregularly shaped polygons around sampled locations. The polygon geometry is such that any location in the polygon area is closer to the sampled point than to any other sampled point. The area of the entire polygon is then assumed to take on the concentration at the sampled location, assuming that proximity is the best predictor of conditions most likely to occur at the unsampled location.

An example of the Thiessen polygon model constructed for the case study area is shown in Figure 3.2-1. Geographic Information Systems software (ESRI, 1989) was used for polygon construction and subsequent generation of PRG implementation maps. The inshore boundary of the site polygons was established as the shoreline at high tide, while the polygons are arbitrarily bounded at the offshore. Shading of polygons was used to demonstrate the locations where CoC concentrations exceed the PRG, hence the area of potential remedial action. It is noted, however, that the polygonal area does not necessarily represent the final remediation area, since the final area will depend on final PRG selection and design sampling to improve spatial resolution.

An objective of PRG implementation is to utilize as much of the available site-specific data as possible to reduce spatial uncertainty. The available data may extend beyond the ERA data set and include results obtained from prior characterization studies. For the case study example, a previous investigation of sediment contamination included 24 sampling locations

(Stations 1 - 24). These data are in addition to the 19 sampling locations investigated as part of the ERA (Stations 25 - 41). Thus for the example data set, the two studies were combined, hence accounting for the total number of polygons shown in Figure 3.2-1.

Ideally, both the ERA and any previous investigations would have used similar sampling protocols and chemical analytical procedures, such that the data would meet the QA/QC and data quality objectives for the study. For example, in the case study, one notable difference was the depth of sediment sampling for the two investigations (0-2 cm vs. 0-15 cm depth). This potential effect of sampling variation on data comparability (hence usability) can be addressed by a comparison of chemical results obtained from closely located stations between the two studies. If the degree of observed variation is within the range generally considered to be acceptable among field duplicates (*i.e.*, 30-40%), it may be concluded that the two data sets are sufficiently comparable to permit the incorporation of the prior data set into the PRG assessment. This was found to be true for the case study data sets.

Summary findings of the ERA study are used to classify the study area polygons based on the probability of adverse ecological risk caused by site-related CoCs to aquatic and avian aquatic predator receptors. Results for the case study ERA are shown in Figure 3.2-2. The map shows the highest probability of adverse ecological risk ("+++") occurring at harbor-front stations DSY-27 and DSY-29, while the lowest probability of risk ("+") was observed for outer cove areas.

Risk assessments for human health typically do not provide sufficient information, on the spatial distribution or magnitude of risk. Rather it is often concluded only that adverse risk due to CoCs in resident biota does or does not exist within the study area. This was the case for the HHRA results available for the present study. However, because the implementation of PRGs is intended to reduce risk, the spatial distribution of human health PRG exceedences must also be considered when evaluating the PRG for remediation as discussed in the following section.

3.3. ASSESSMENT OF PRGS FOR RISK REDUCTION

The assessment of the suitability of PRGs as cleanup goals involves an analyte-by-analyte evaluation of baseline PRGs in order to define the relationship between the degree of PRG exceedence and risk at the site (Step 7). As part of this evaluation, the calculated values are also assessed in relation to traditional benchmarks, to gauge the relative degree of protection afforded to exposure pathways by the site-specific PRGs.

A general assessment approach for PRGs is presented in Section 3.3.1, followed by case study examples for site-specific PRGs for aquatic (Section 3.3.2), avian predator (Section 3.3.3) and human health (Section 3.3.4) exposure pathways. Conclusions and recommendations are presented in Section 3.3.5.

3.3.1. General PRG Assessment Approach

Step 7. Evaluate the practicality of the PRGs for effective risk reduction. In evaluating the practicality of PRGs for effective risk reduction, candidate PRGs which would result in risk reduction in the most affected areas are favored over other candidate PRGs that do not. Unlike

the previous steps, this step is a qualitative, risk-based interpretation based on best professional judgment. This is primarily a spatial analysis, where the location of PRG exceedences (*e.g.*, $HQ_{PRG} > 1$) for each of the limiting CoCs is reviewed with respect to the spatial distribution of observed risks at the site as concluded from the results of the ERA or HHRA. The relationship between the apparent risk reduction and the remediated area is discussed and presented as recommended PRGs to provide input into risk management decisions regarding the setting of final Remediation Goals (as part of the final Record of Decision for the site).

3.3.2. Case Study Aquatic PRG Assessment

In the case study ERA, potential exposure pathways of concern included chemicals available to aquatic biota from in-place (bedded) sediments as well as chemicals made available by mixing of sediments into the water column (resuspended sediments). The site-specific PRGs for each of these exposure pathways are discussed in separate sections below. In this interpretation it was assumed that the extent of PRG exceedence should be proportional to risks; that is, higher risks will be associated with sediments having chemical concentrations that exceed the PRG to a greater degree. Accordingly, the point of departure where the relationship can no longer be discerned can be considered as a lower limit of predictability for the PRG. The analysis focuses on determining this lower limit because implementation of a PRG below this level would have uncertain risk reduction benefits. This concept is further clarified in the following section.

Bedded sediments. In the case study example, the baseline PRGs developed for the bedded sediment exposure pathway included HMW PAHs and Total PCBs (Table 3.1-1). The discussion below presents the logic used to select recommended PRGs that were adopted for the case study area.

HMW PAHs. The PRG for HMW PAHs (6203 ng/g dry weight) was exceeded at eight case study area stations (Figure 3.3-1), seven of which the exceedence was the highest observed for the location. PRG exceedences were greatest for the harbor front stations, particularly high-risk area stations DSY-3 and DSY-29 where sediment concentrations were 11 and 4.6 times the baseline PRG, respectively (Exhibit 7). Other exceedences included intermediate risk area stations DSY-20 (PRG HQ = 2.0) and DSY-18 (PRG-HQ = 3.1) as well as low risk area stations DSY-30 (PRG-HQ = 1.5) and DSY-19 (PRG-HQ = 1.4). Finally, the PRG for HMW PAHs was exceeded at Station DSY-2 (PRG-HQ = 9.7) and although not sufficiently proximal to a risk-ranked location, it is presumed to be intermediate to high risk.

As can be seen in Exhibit 7, good agreement exists between the magnitude of PRG exceedence and ecological risk, and a PRG-HQ = 2 appears to be the point of departure between low and intermediate to higher risks, and thus a recommended PRG equal to two times the baseline PRG was suggested to risk managers as a remediation concentration that would preferentially elevated risk areas.

Exhibit 7. Example Evaluation of Baseline PRGs.

- PRG exceedences are compared to the risk findings in order to select the RPRGs.
- The baseline and RPRGs are used as input into the Feasibility Study and resulting risk management decisions regarding the setting of Remediation Goals.

Station	Risk	HQ_{PRG} Max	
DSY-19	Low	1.4	
DSY-30	Low	1.5	2.0*
DSY-18	Intermediate	2.0	
DSY-20	Intermediate	3.1	
DSY-29	High	4.6	
DSY-2	High?	9.7	
DSY-3	High	11.0	

*In consideration of all PRG/risk comparisons, a RPRG = 2X the BPRG was adopted for the bedded sediment aquatic exposure pathway.

The recommended HMW PAH PRG value (12407 ng/g) was compared to existing sediment benchmarks as an independent check on the degree of protection that would be afforded to aquatic biota exposed to chemicals in bedded sediments. The recommended PRG concentration was found to be between the ER-M (9600 ng/g dry weight) and the State of Washington Apparent Effects Threshold - Low (AET-L; 17,000 ng/g) concentration (Barrick *et al.*, 1988). Hence, the PRG was determined to be within the range of values expected to protect aquatic biota at this site.

Total PCBs. A similar evaluation procedure to that of HMW PAHs discussed above was used to determine the recommended PRG for Total PCBs.

In contrast to HMW PAHs, the baseline PRG for Total PCBs (1638 ng/g dry wt) was exceeded only at Station DSY-27 (HQ_{PRG} = 2.02, Figure 3.3-2). The lack of PRG exceedences at all other sampled locations suggests that risks due to PCBs are not widespread at the site. Still, this station was identified as high risk in the Marine ERA and the PRG exceedence was the high observed for that location. Thus it was reasoned that PCBs could be responsible for high risks and therefore the implementation of a PRG for Total PCBs at the baseline concentration should be provided to assist remedial decisions for this location.

As with the HMW PAH PRG, the baseline Total PCB PRG concentration (1638 ng/g) was compared to existing sediment benchmarks as an independent check on the degree of protection that would be afforded to aquatic biota. While the PRG is well above the ER-M (180

ng/g dry weight), it is intermediate between the AET-low (1000 ng/g dry) and AET-high (3100 ng/g dry) benchmarks. Thus, the baseline PRG was determined to be within the range of independent estimates of PCB concentrations considered protective of aquatic biota and was recommended, without adjustment, for adoption as a clean-up concentration to address high risks at the site.

Overall Assessment. Areas targeted for potential remediation based on implementation of recommended PRGs for the bedded sediment exposure pathway are shown in Figure 3.3-3. From the comparison of PRG exceedences with observed risk at the site (Figure 3.2-2), adopting sediment concentrations of 12407 ng/g for HMW PAHs and 1638 ng/g for PCBs would ensure risk reduction at the two high-risk areas of the site. Concentrations below these thresholds that would discriminate between areas of intermediate or lower risks could not be discerned.

Resuspended Sediments. The limiting CoCs identified during PRG derivation for the resuspended sediment exposure pathway were arsenic, copper, lead, Total PCBs and o,p'-DDE.

Arsenic. Although arsenic was identified as a limiting CoC, and marginal exceedences of the elutriate-based threshold effects values were observed at two locations (DSY-38: HQ_{TEV} = 1.01; DSY-39: HQ_{TEV} = 1.88), exceedences of the corresponding PRG were not observed. This disparity is due in part to the imprecision associated with converting threshold effects values to PRGs, particularly when the extent of threshold effect value exceedence is limited. Both stations DSY-38 and DSY-39 were assigned low risks in the ERA, and were located well offshore of higher risk stations. Thus, the lack of proximity between the location of potential exceedences and the overall low risks indicated that arsenic was a poor candidate for PRG selection. Hence arsenic was not recommended for adoption as an aquatic PRG for purposes of determination of remedial action for bedded sediments.

Copper. The baseline PRG concentration of 74 µg/g dry wt was calculated following Step 6, and the spatial implementation of this baseline PRG is found in Figure 3.3-4.

While several stations had sediment concentrations above the copper PRG, a number of these locations, including the two high risk stations (Stations DSY-27 and DSY-29) had non-detectable elutriate concentrations of copper as measured in the ERA. In fact, the retention of copper as a limiting CoC was based on a single exceedence of the no effect concentration measured in the elutriate sample (HQ_{TEV} = 1.76 at Station DSY-31; Table 2.1-5). Again, as with arsenic, this disparity is due in part to the imprecision associated with converting threshold effects values to PRGs, particularly when the range of PRG exceedences is small (*i.e.*, 1.1 - 3.5). Thus, based on the above information, it was recommended that copper not be adopted as an aquatic PRG for purposes of determination of remedial action for resuspended sediments.

Lead. The spatial implementation for the baseline PRG for lead (84 µg/g dry weight) indicated exceedence at nine stations (Figure 3.3-5). For three of the stations (DSY-29, DSY-32 and DSY-36), the exceedence of the PRG for lead was the highest for that location. In comparing PRG exceedences with observed risk at the site, Stations DSY-29 (PRG-HQ = 2.4) was high risk, while DSY-32 (PRG-HQ = 1.6) and DSY-36 (PRG-HQ = 1.0) were low risk. Because there were no intermediate risk stations with sediment concentrations above the PRG, the point of departure between low and higher risk areas could not be easily discerned.

Thus in consideration of the observed variation in correlation between the baseline PRG exceedence and measured risk, a PRG concentration of about two-fold above the baseline PRG was selected to discriminate between low and higher risk areas. The resulting PRG concentration (168 µg/g) was recommended as an aquatic PRG for determination of remedial action for resuspended sediments (Table 3.1-2).

o,p'-DDE. The pesticide o,p'-DDE was retained as a limiting CoC because the sum threshold effect value hazard quotients at Station DSY-29 exceeded unity (Table 2.1-5). As explained in Section 2, this step was performed to permit a more thorough evaluation of all available sediment data (particularly non-ERA sediment data). Also, the corresponding threshold effect value was selected based on the background concentration (Table 2.1-4).

A single exceedence of the PRG value for o,p'-DDE was observed for Station DSY-27 (HQ_{PRG} = 7.2). Although this station is one of two high-risk areas identified in the case study ERA (Figure 3.2-2), the corresponding exceedence of the threshold effect value for this station (HQ_{TEV} = 0.83) was below the presumed threshold for aquatic risks. Also, no exceedence of the PRG was observed for Station 29, the location where this CoC was identified as a limiting CoC as described above. Given that this procedure is intended to facilitate the detection of any locations where this CoC might be a primary risk driver, and that no other location contained sediment concentrations above the PRG, it was recommended that o,p'-DDE not be retained as a PRG (Table 3.1-1).

Total PCBs. The baseline PRG for Total PCBs (530 ng/g dry weight) was exceeded at Stations DSY-27 (PRG-HQ = 6.8), DSY-3 (PRG-HQ = 1.5), DSY-11 (PRG-HQ = 1.4) and DSY-29 (PRG-HQ = 1.1), of which DSY-11 was the outright highest exceedence for that location (Figure 3.3-6). If a PRG for o,p'-DDE is not recommended as discussed above, then the exceedence observed for Total PCBs at Station DSY-27 also becomes the highest for that location.

Sediment concentrations at high risk Station DSY-27 were more than six-fold (PRG-HQ = 6.8) above the baseline PRG, while Station DSY-11, collocated with intermediate risk Station DSY-31 had a PRG exceedence only slightly higher than the baseline PRG (HQ_{PRG} = 1.3). Copper also had a similar exceedence of its PRG at this location (PRG-HQ= 1.2) hence there is uncertainty as to whether the L-CoC at this location that is causing toxicity is actually Total PCBs. Given this uncertainty, a PRG threshold equal to two times the baseline PRG (1060 ng/g) was conservatively recommended as the concentration that would reliably address high risks due to PCB exposure from resuspended sediments.

Overall Assessment. The recommended PRGs for lead (168 µg/g) and Total PCBs (1060 ng/g) were adopted for the resuspended sediment exposure pathway since good correspondence was observed between areas exceeding PRGs and areas of high risk (Figure 3.3-7). Unlike bedded sediments, however, it was difficult to independently assess the level of protection afforded by the derived PRG values since benchmarks for resuspended sediments are not available. In addition, it is unclear whether the high-risk areas identified in the ERA were due to exposures from bedded or resuspended sediments, or both. Areas exceeding resuspension PRGs were a subset of the total area above the bedded PRGs. This suggests that

implementing the bedded sediment PRGs would rectify resuspension risks as presently delineated.

3.3.3. Case Study Avian Predator PRG Assessment

For the avian predator exposure pathway, two metals (silver and zinc) and Total PCBs were identified as limiting CoCs (Table 3.1-1).

Metals. Zinc was above the baseline PRG at five stations having HQ_{PRG} values ranging from = 1.46 to 4.58 (Figure 3.3-8). Among these, four stations had HQ_{PRG} values > 2 (DSY-2, DSY-3, DSY-11, and DSY-27). Baseline PRGs were not exceeded for silver.

In the case study ERA, intermediate risks to avian predators were assigned to Stations DSY-28, DSY-29 and DSY-36, while low risks were apparent elsewhere, including reference locations (Table 1.3-1). There was some agreement between locations of zinc PRG exceedences and elevated risk, particularly for areas in the region of Stations DSY-2/DSY-28 and DSY-3/DSY-29. However, the corresponding zinc HQ_{TEV} values for shellfish tissue were uniformly less than unity across the site, and zinc had been retained as a limiting CoC only to permit a more thorough evaluation against all the sediment data. The fact that only minor PRG exceedences were observed and also the conservative exposure assumptions in the ERA (*e.g.*, the avian predator would spend entire life at the single location) lead to the conclusion that zinc was unlikely to pose an unacceptable risk to avian receptors. Thus, despite the fact that the PRG for zinc was exceeded it was recommended that a PRG for zinc not be adopted.

Total PCBs. Baseline PRGs for Total PCBs were exceeded at three stations (Figure 3.3-9), with HQ_{PRG} values ranging from 1.2 to 5.9. Among these locations, only Station DSY-27 exhibited a $HQ_{PRG} > 2$. Again, because of the conservative exposure assumptions and limited spatial extent of PRG exceedence, risk to avian receptors due to PCB exposure was deemed unlikely. Thus, the selection of a PRG to protect avian predators was not recommended for this CoC.

3.3.4. Case Study Human Health PRG Assessment.

The limiting CoCs identified for protection of risks to recreational fishermen were arsenic and benzo(a)pyrene. The baseline PRGs were calculated to represent concentrations causing carcinogenic effects at a rate of one in one million people (1×10^{-6}). However, the human health exposure scenario was deemed overly conservative (a fisherman was not likely to derive all seafood exclusively from the case study area for 30+ years from a single location, nor could the single location support such intensive pressure from a large number of fishermen). Instead, a more plausible (yet still conservative) assumption was adopted in that a shellfisherman might rely on the case study area for up to 10% of the total dietary requirement. Thus, a value of ten times (10x) the HQ_{PRG} threshold was adopted as a more realistic point of comparison for possible adverse health effects due to shellfish consumption. With this assumption in mind, the PRGs were evaluated at baseline PRG and 10x the baseline PRG thresholds.

Arsenic. Arsenic was identified as a limiting CoC for protection of risks to human health exposure from consumption of shellfish. While arsenic concentrations marginally exceeded the threshold effects value ($HQ_{TEV} < 2$), the corresponding HQ_{PRG} values were all less than unity.

This discrepancy was attributed to uncertainty in the Bioaccumulation Factor for arsenic used to calculate the sediment PRG from the tissue-based threshold effects value. However, any overlooked risk because of the BAF limitation was outweighed by the true risk to arsenic being overestimated by an order of magnitude since studies have shown that the toxic fraction (*i.e.,* the organic component) is typically about 10% of the total arsenic content (U.S.FDA, 1993). Further, a review of the literature regarding the methodology used to derive the toxicity reference value (extrapolated from mice) revealed that the route of exposure evaluated was arsenic in drinking water. Since arsenic was administered in soluble form, it is likely to be far more bioavailable than arsenic bound to sediment particles. Finally, arsenic risks are unlikely to be significant, as all areas had sediment concentrations below the $HQ_{PRG} = 10$ threshold.

Based on the above data, it was recommended that a total arsenic value not be selected as a final PRG, but measurement for organic arsenic concentrations be performed at least once to confirm that bioavailable concentrations are below toxic levels. Revision of the PRG list to include organic arsenic could occur depending on the results.

Benzo(a)pyrene. Benzo(a)pyrene was also identified as a limiting CoC for protection of risks from consumption of shellfish. The baseline PRG concentration (53.6 ng/g dry wt) was exceeded at 34 of 41 stations (Figure 3.3-10). The stations with highest PRG exceedences (HQ > 10) were confined to the nearshore areas. However, it was determined that much of the area exceeding the PRG (HQ=10) threshold are not fishable due to industrial/military activity in the case study area (approximately all areas between and eastward of the piers and dock areas shown in Figure 3.3-10). The area represented by polygons around Stations DSY-18 and DSY-30 was deemed to be fishable, and PRGs could be implemented in some manner to guard against adverse risk from shellfish consumption. Thus, it was recommended that the 10 x baseline PRG concentration be adopted as the recommended PRG (536 ng/g dry weight). It was also recommended that a more detailed delineation of the affected area be conducted in order to weigh the advantages of risk reduction against the disruptive nature of remediation.

For the two CoCs for which baseline PRGs were developed, only benzo(a)pyrene was retained as a recommended PRG (Figure 3.3-11). Arsenic was excluded due to low overall unity and limited exposure. Based on the case study data, it was deemed unlikely that the shellfishing population is substantially at risk since fishable areas are below recommended PRGs (*i.e.*, sediment concentrations which would result in bioaccumulation to levels where possible adverse health effects due to shellfish ingestion might be possible). The need for monitoring this CoC in order to confirm that harvested shellfish remain below toxic levels is an issue to be addressed in the Long Term Monitoring program for the site.

3.4. OVERALL SUMMARY

In consideration of the four exposure pathways identified as posing potential risks to ecological and human health receptors, baseline PRGs were developed for nine CoC including five metals (arsenic, copper, lead, silver and zinc), two PAHs (benzo(a)pyrene and HMW PAHs), Total PCBs, and the pesticide o,p'-DDE (Table 3.1-2). The PRG implementation results show that risks to aquatic receptors due to chemical exposure from bedded sediments would addressed by adoption of a RPRG for HMW PAHs at 12407 ng/g, while risks due to resuspended

sediment chemical exposure would be addressed by selection of a RPRG for lead (168 µg/g) and Total PCBs (1060 ng/g). It is noted that an RPRG for Total PCBs in bedded sediment would also be required if the lower PRG for Total PCBs (1638 ng/g) resuspended sediments was not adopted.

In contrast to the aquatic biota, the selection of a PRG for avian predator exposure was not recommended due to highly conservative exposure assumptions used in the ERA and the observed lack of corresponding exceedences of Threshold Effects Values in prey tissue. Finally, human health risks from consumption of shellfish would be addressed by implementation of a PRG for benzo(a)pyrene (535 ng/g).

4. CONCLUSIONS/RECOMMENDATIONS

The PRG development strategy presented in this report was developed in a manner consistent with typical site ARARs, as well as the findings of the risk assessments with respect to risk reduction. Confirmation of the validity of the process is demonstrated by the fact that the magnitudes of the PRGs are generally comparable to independent correlative benchmarks such as ER-L/ER-M and AET values. This reduces uncertainty with respect to residual risk associated with the CoCs once the PRGs are implemented.

The recommended PRG concentrations identified in the case study for aquatic, avian and human health exposure pathways were submitted for consideration as Final Remediation Goals in the Feasibility Study for the site after thorough Navy and regulatory review. Agreement among the lead agency (Navy), Natural Resource Trustees and the public is currently being sought. Remediation to baseline PRG concentrations does not, in the majority of locations, provide an optimal balance between the extent of risk reduction achieved and potential environmental impacts that would occur on adjacent areas during the remediation process. Hence, the Recommended PRGs were put forward for the site to address areas of highest risk. Still, these values were based solely on interpretation of the observed distribution and severity of estimated risks; other considerations presented in the FS report regarding cost, engineering constraints and non-chemical-specific ARARs may still lead to modification of the Preliminary Remediation Goals to be adopted by risk managers.

Furthermore, depending on the nature of the remedial action, a PRG list based on a combined pathway analysis may be suitable. It is acknowledged that the spatial resolution of the case study analysis depends on the density of stations within the study area. In the case study, and at other sites as well, some areas that might require remedial action may be depicted larger than necessary. Thus, confirmation sampling during the pre-design investigation is required in order to reduce uncertainty and better define the boundaries of the areas to be remediated.

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FIGURES

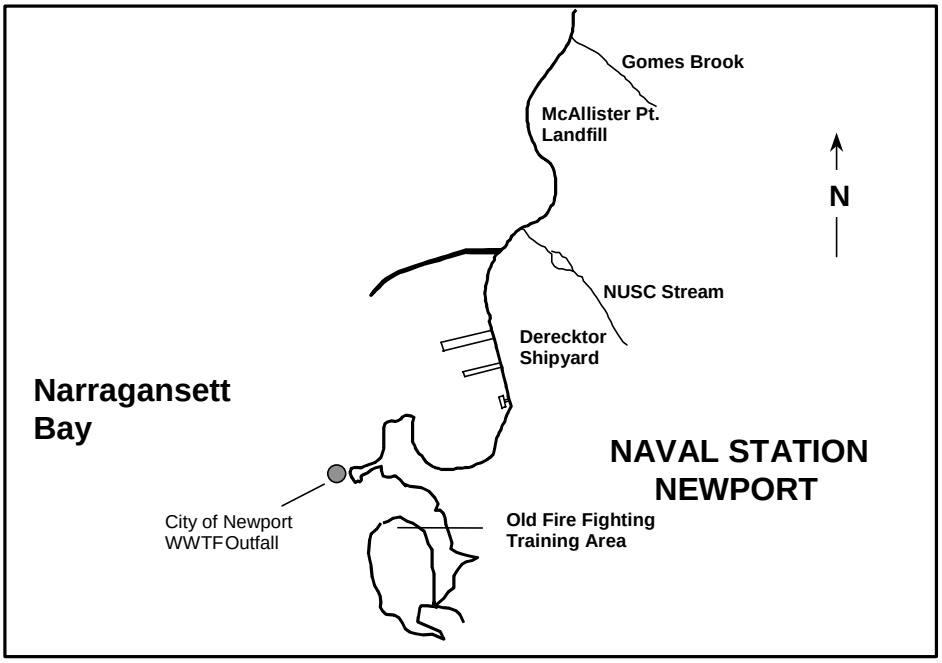


Figure 1.3-1. Derecktor Shipyard ERA case study area.

Figure 2.1-2. Equilibrium partitioning relationships for CoCs among environmental media.

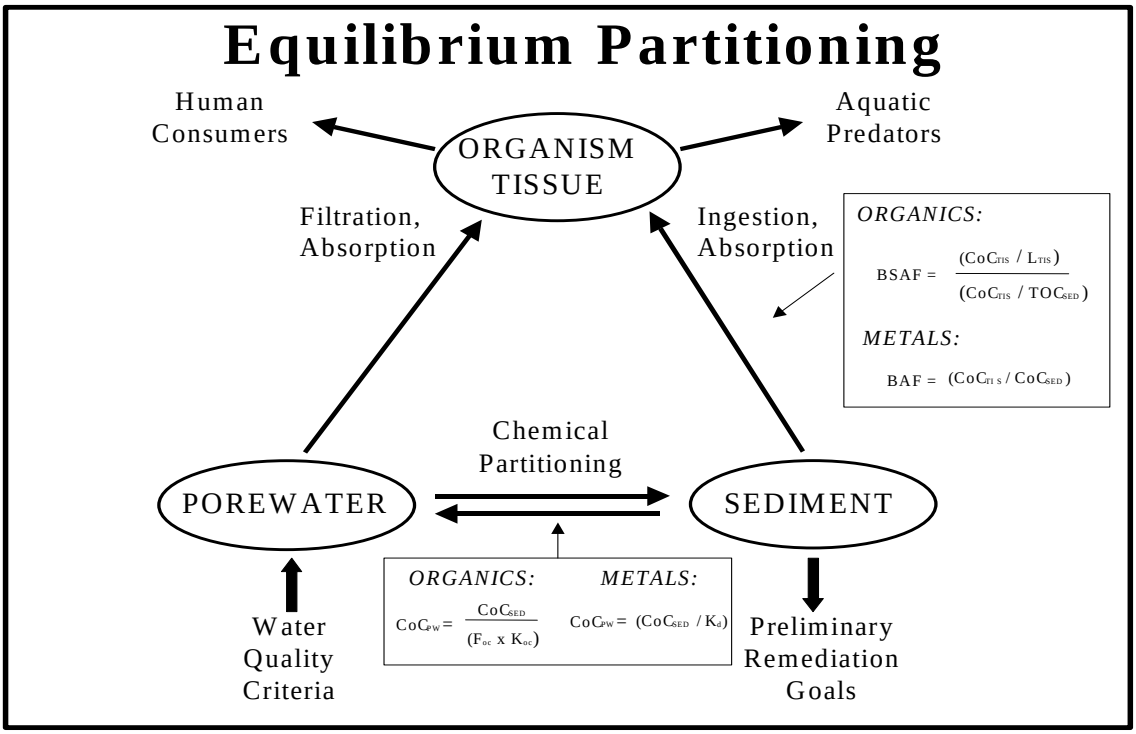
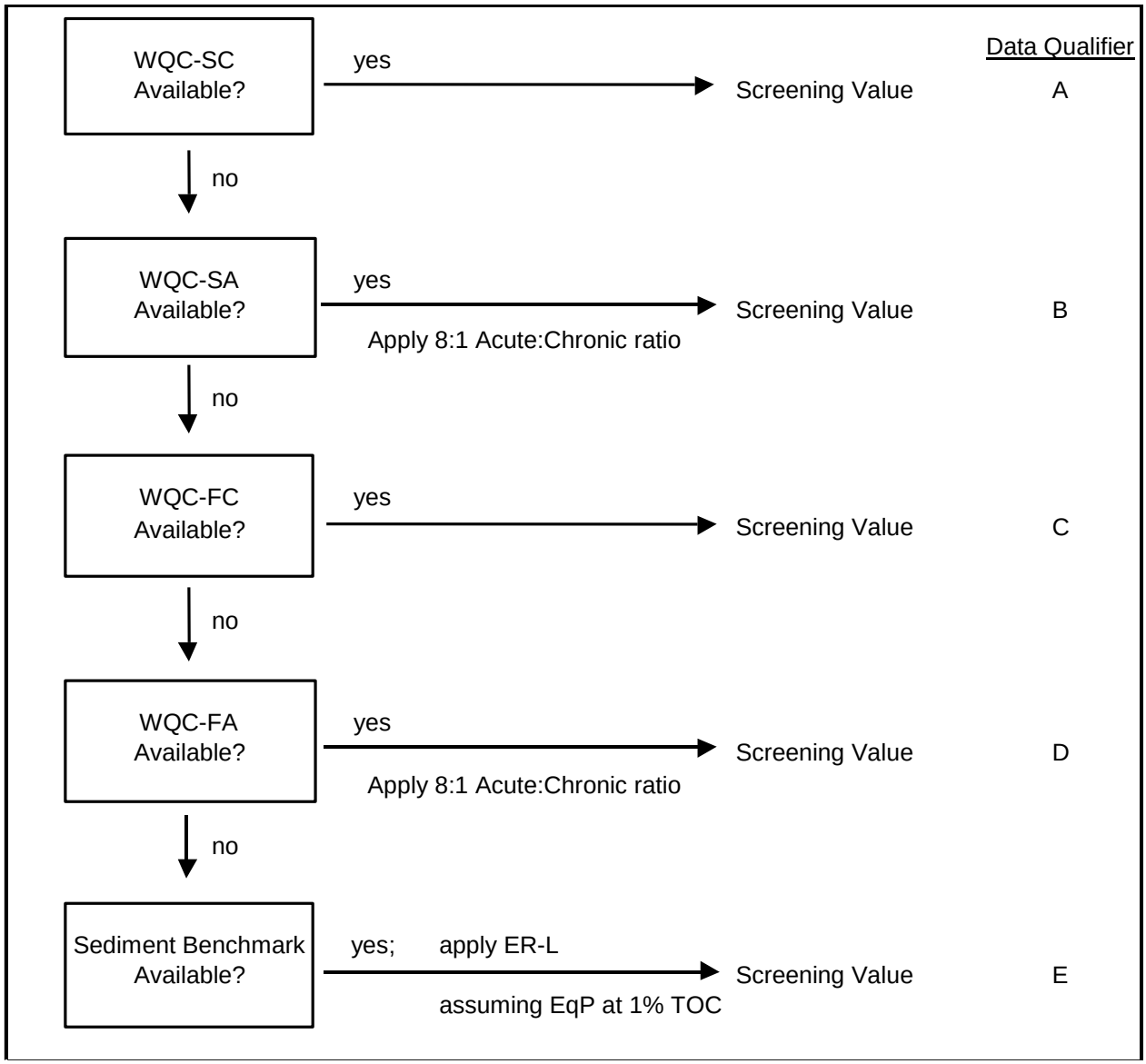


Figure 2.1-1. Water Quality Screening Value selection process.



WQC-FA = Water Quality Criteria = Freshwater Acute Value
WQC-FC = Water Quality Criteria = Freshwater Chronic Value
WQC-SA = Water Quality Criteria = Saltwater Acute Value
WQC-SC = Water Quality Criteria = Saltwater Chronic Value
WQSV = Water Quality Screening Value
Data qualifier "A" is applied to benchmarks derived directly from existing WQC-SC values.
Data qualifier " B" is applied to contaminants possessing WQC-saltwater acute values (WQC-SA). The equivalent WQC-SC value was derived by dividing the WQC-SA value by 8. The conversion factor derived from the mean overall acute:chronic ratio for paired chemical data contained in the U.S.EPA AQUIRE database (Shepard, 1995).
Data Qualifier "C" is applied when Freshwater chronic data (WQC-FC) are used directly as screening values.
Data Qualifier "D" is applied freshwater acute (FA) values were converted to chronic values by dividing by 8.
Data Qualifier "E" was applied when NOAA Effects Range-Low (ER-L) (Long *et al.* , 1995) concentrations were selected and translated into porewater equivalent concentrations using the EqP model assuming 1% sediment TOC.

Figure 3.2-1. Thiessen polygons for PRG implementation for the Derecktor Shipyard/ Coddington Cove study area.

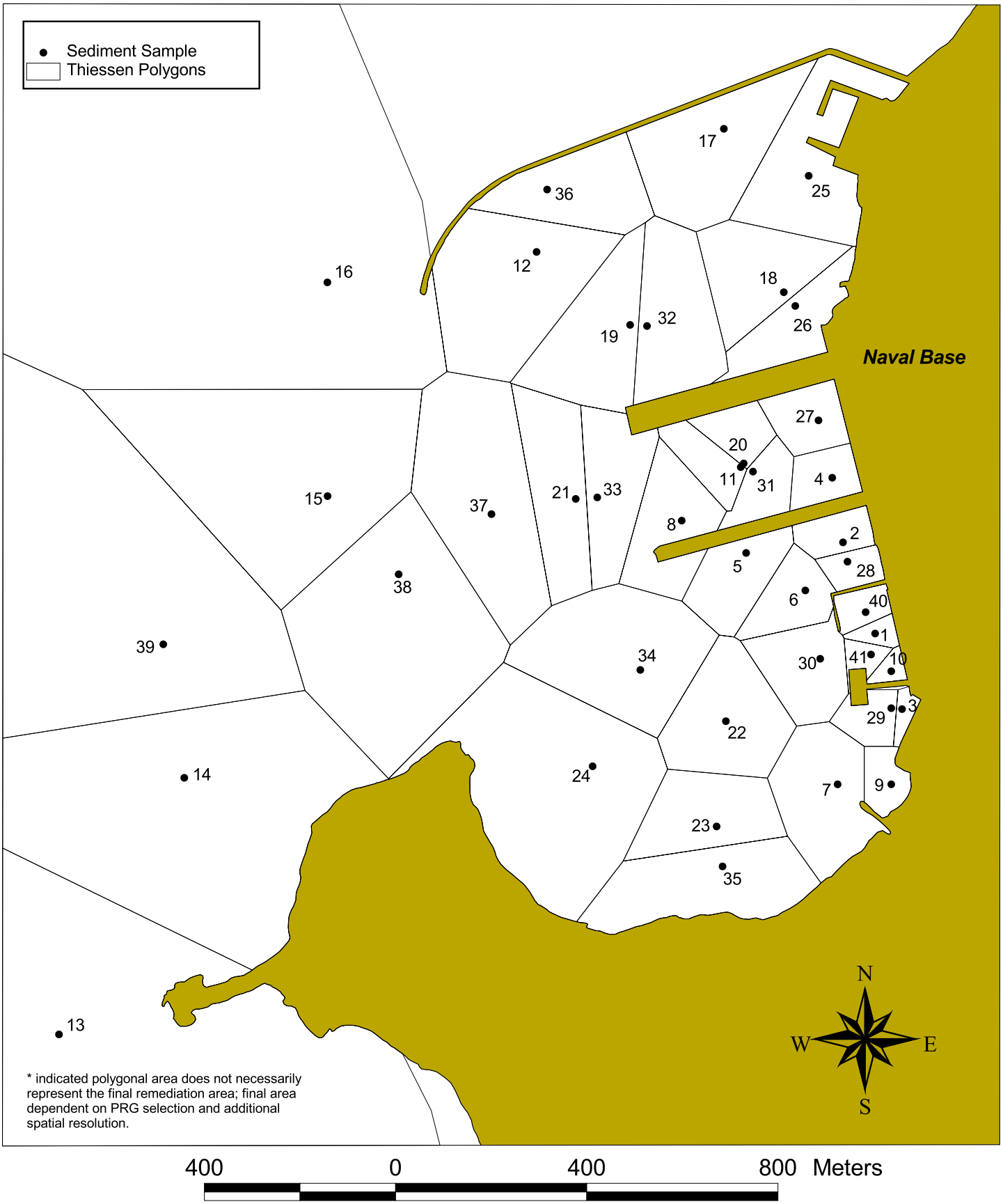


Figure 3.2-2. Risk probability for the Derecktor Shipyard/Coddington Cove study area.

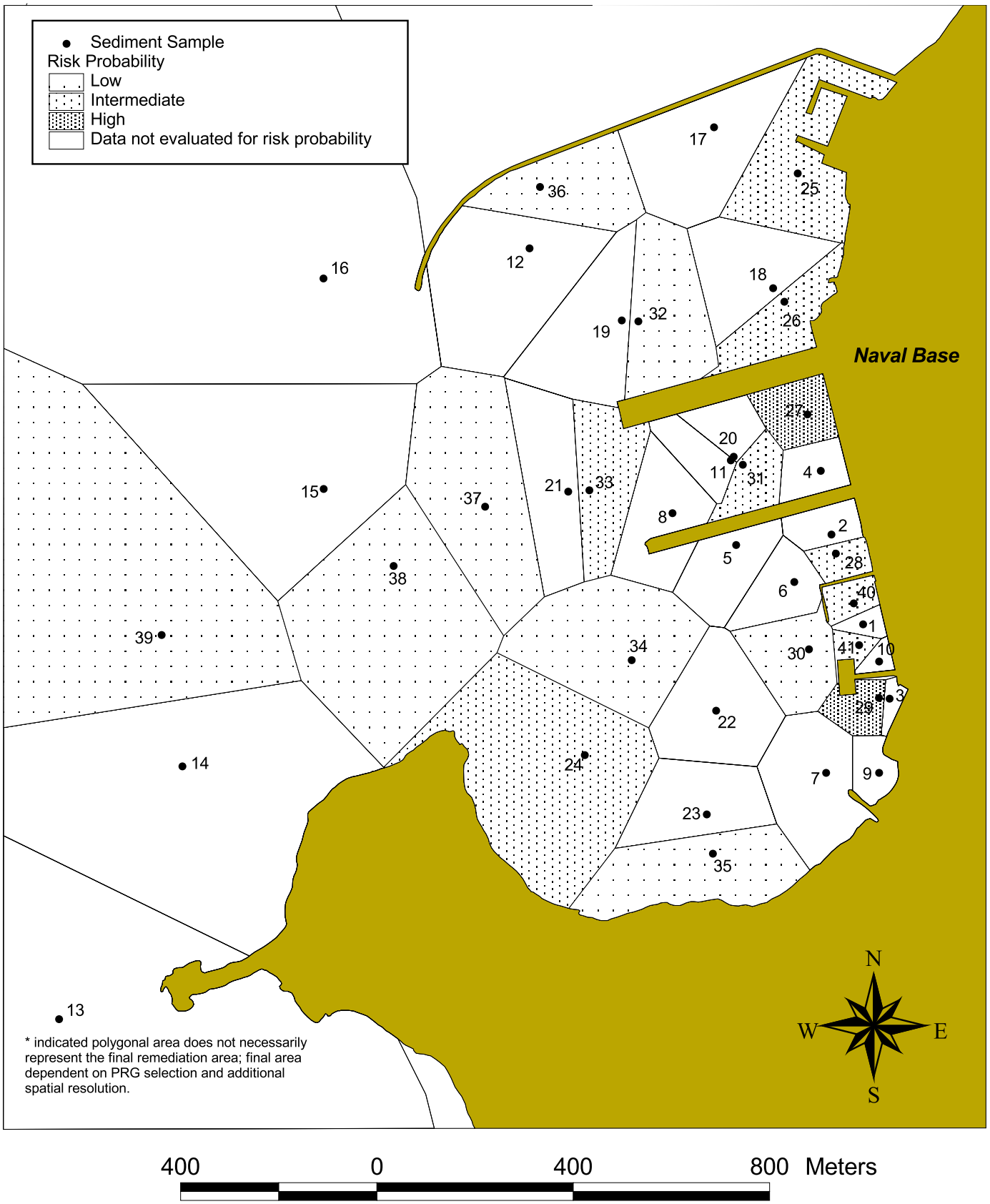


Figure 3.3-1. Summary of CoCs exceeding PRGs for protection of aquatic biota by location for sediments in the Derecktor Shipyard/Coddington Cove study area:*

Bedded Sediment Exposure Pathway for HMW PAHs

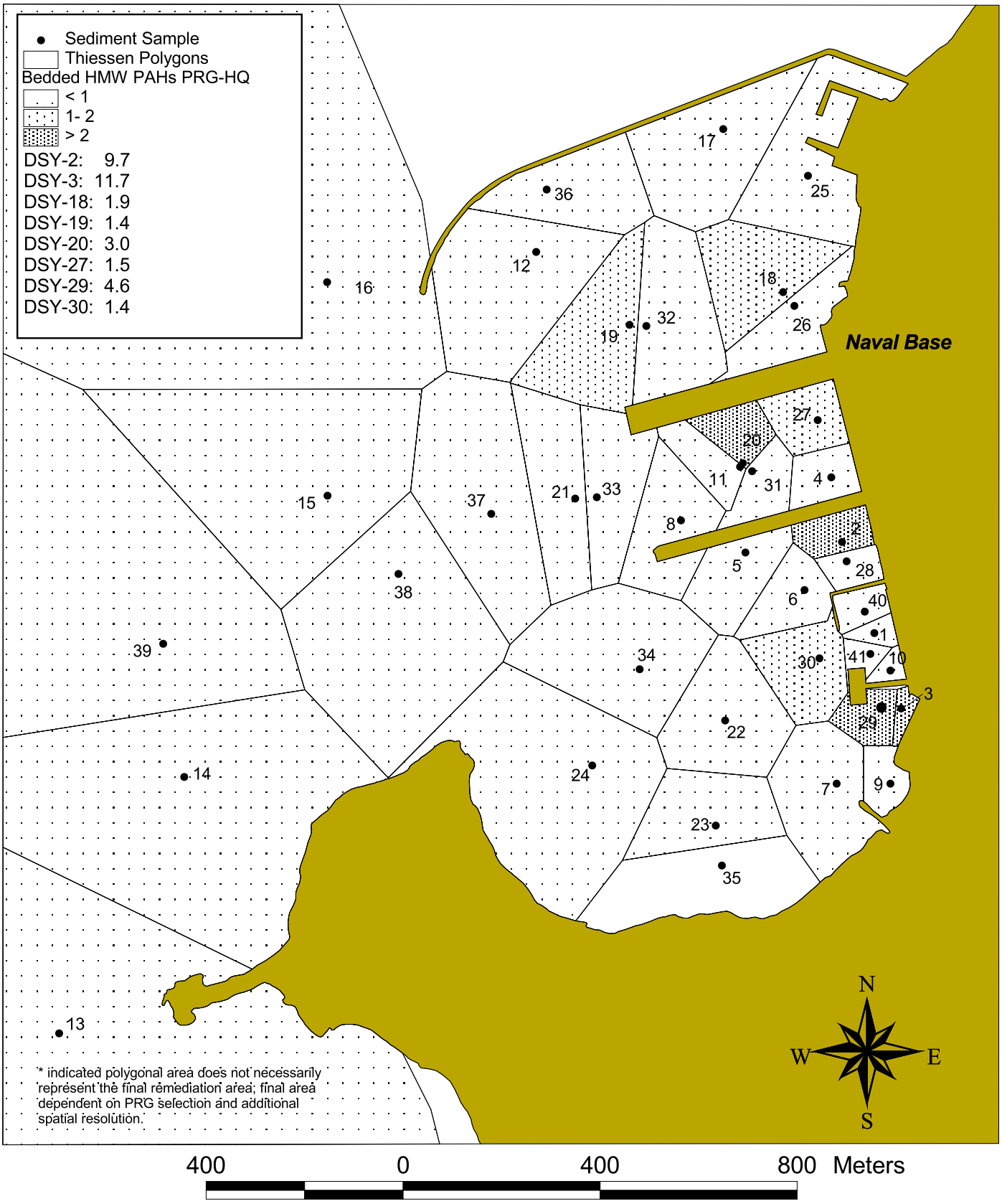


Figure 3.3-2. Summary of CoCs exceeding PRGs for protection of aquatic biota by location for sediments from the Derecktor Shipyard/Coddington Cove study area:*

Bedded Sediment Exposure Pathway for Total PCBs

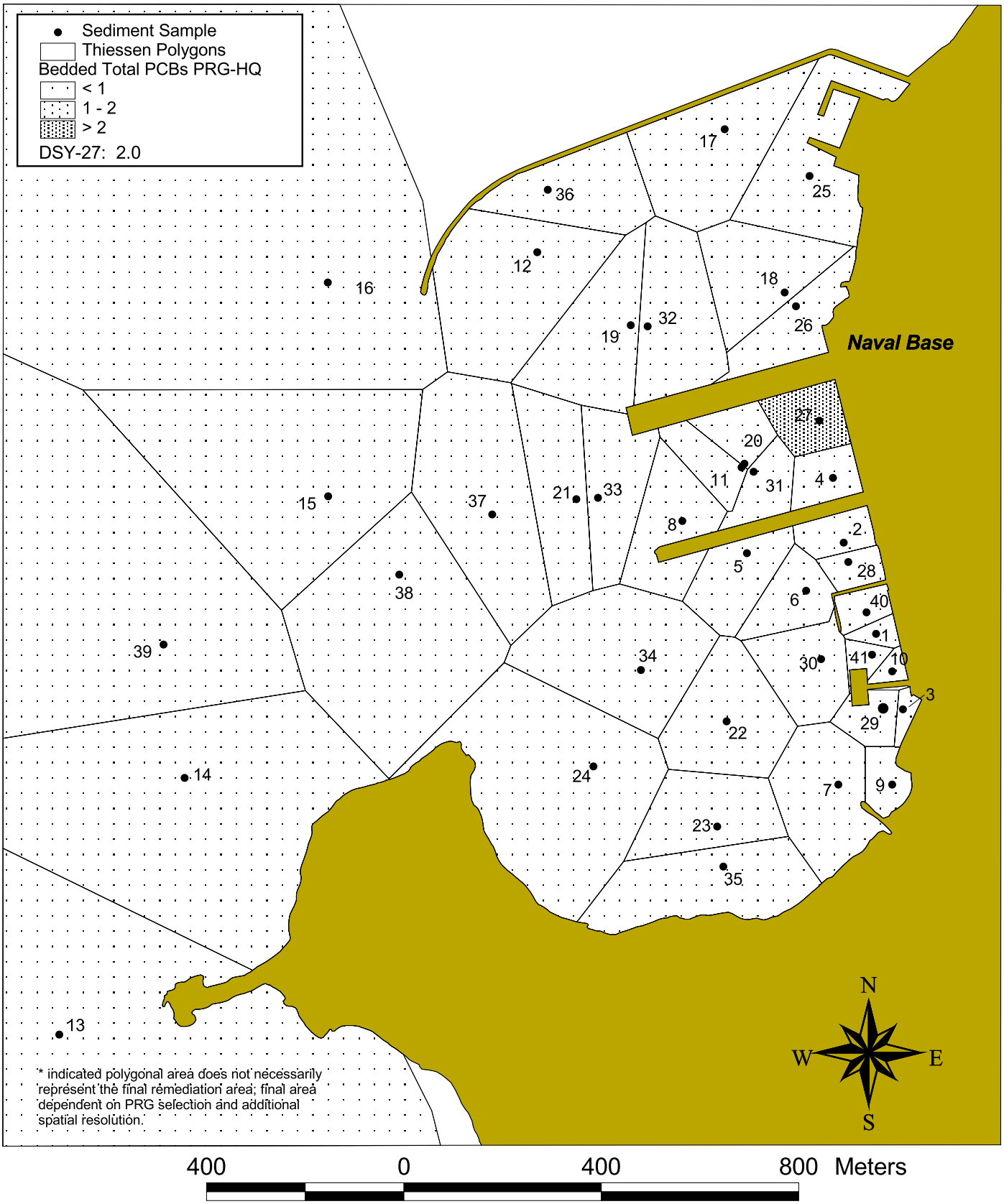


Figure 3.3-3. Summary of CoCs exceeding aquatic PRGs by location for sediments from the Derecktor Shipyard/Coddington Cove study area:*

Recommended PRG Implementation for the Bedded Sediment Exposure Pathway

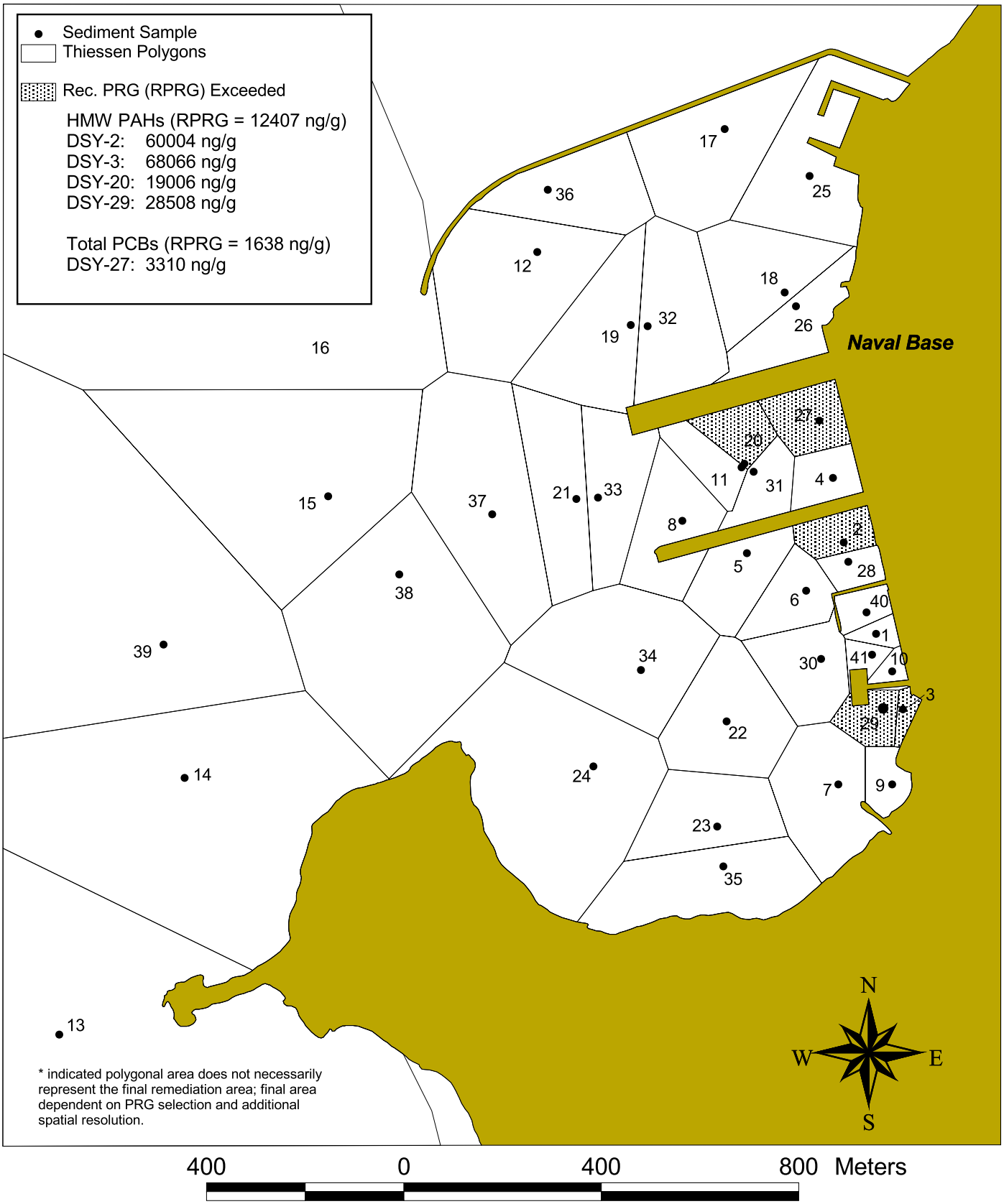


Figure 3.3-4. Summary of CoCs exceeding PRGs for protection of aquatic biota by location for sediments from the Derecktor Shipyard/Coddington Cove study area:*

Resuspended Sediment Exposure Pathway for Copper

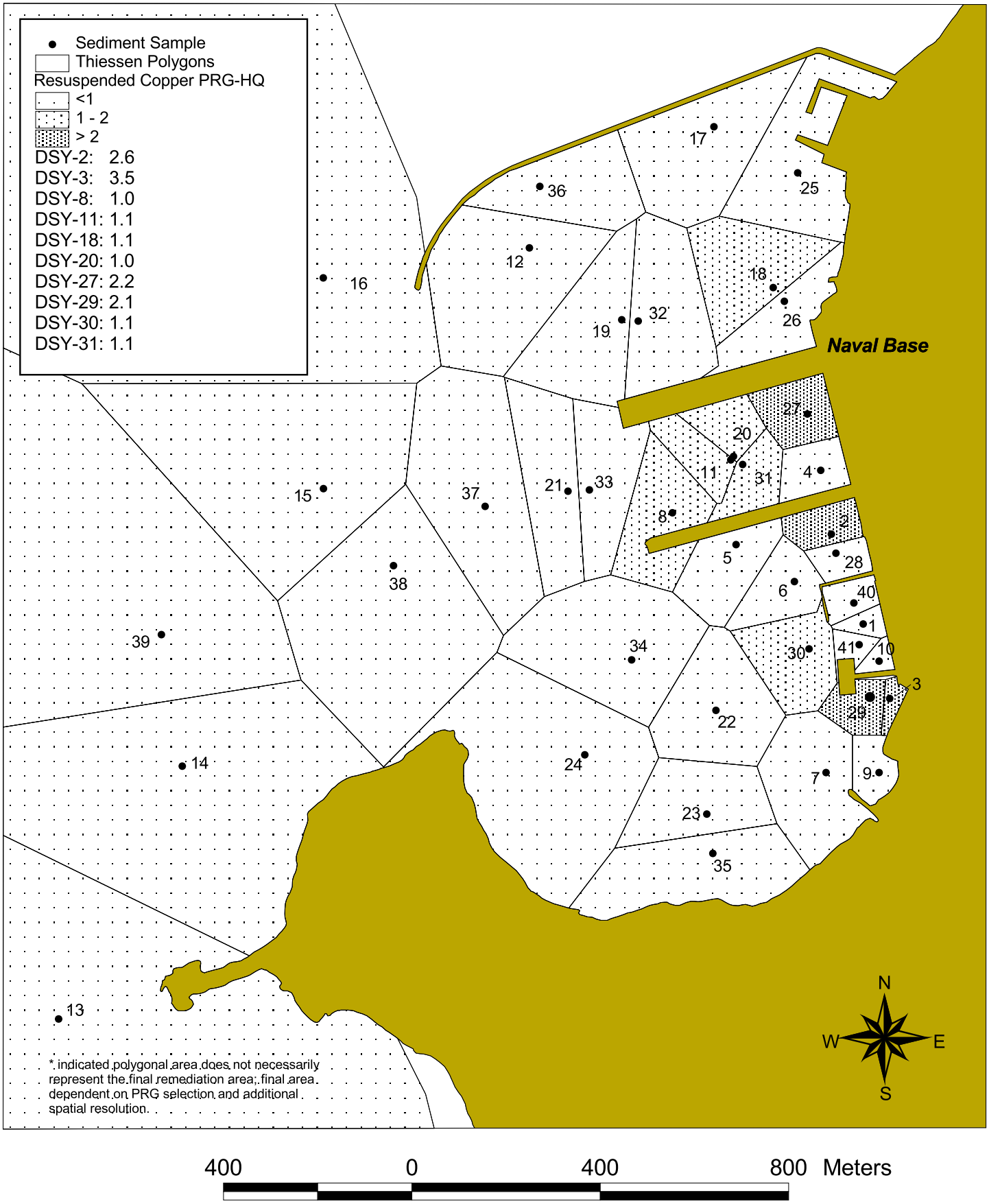


Figure 3.3-5. Summary of CoCs exceeding PRGs for protection of aquatic biota by location for sediments from the Derecktor Shipyard/Coddington Cove study area:*

Resuspended Sediment Exposure Pathway for Lead

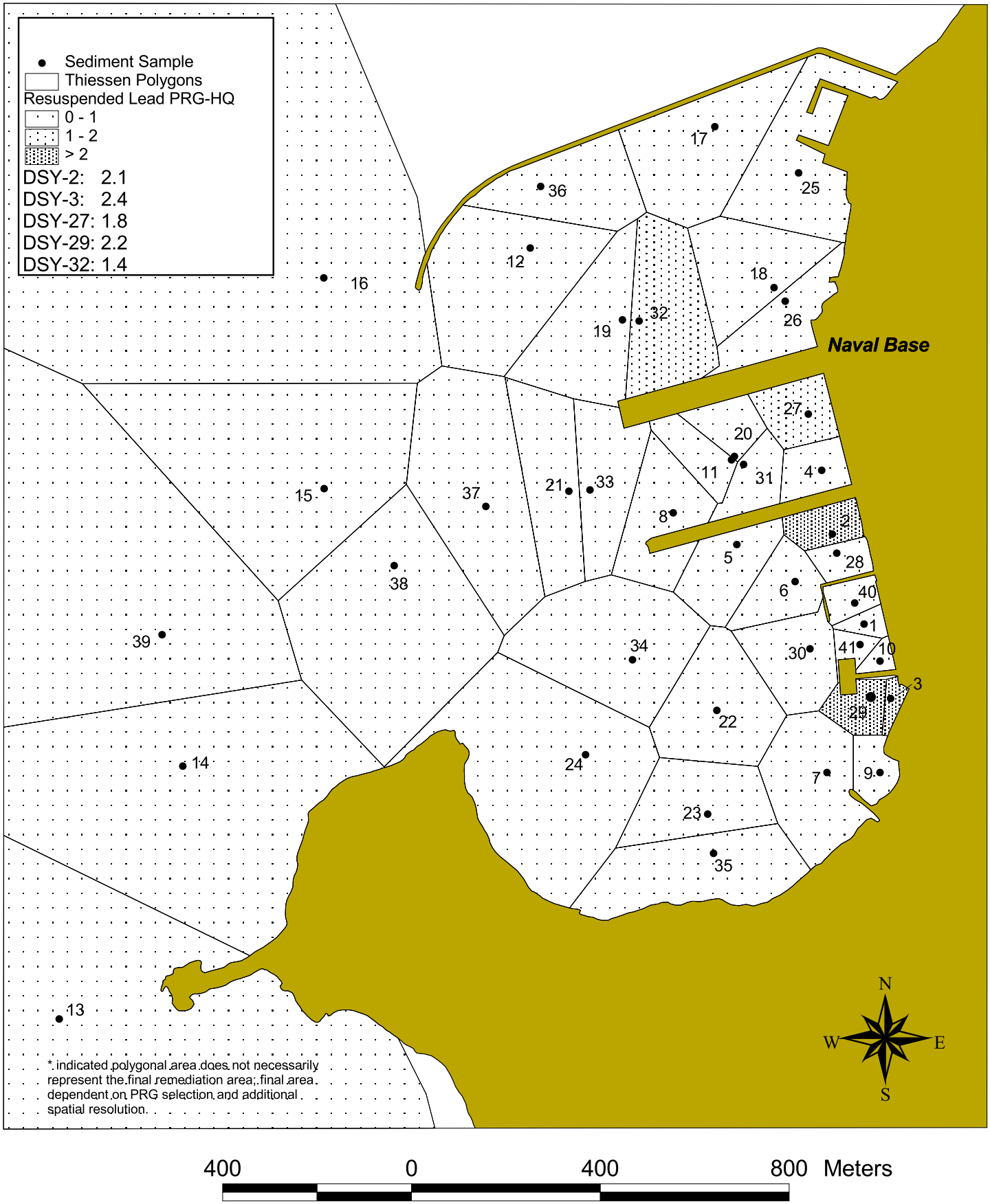


Figure 3.3-6. Summary of CoCs exceeding PRGs for protection of aquatic biota by location for sediments from the Derecktor Shipyard/Coddington Cove study area:*

Resuspended Sediment Exposure Pathway for Total PCBs

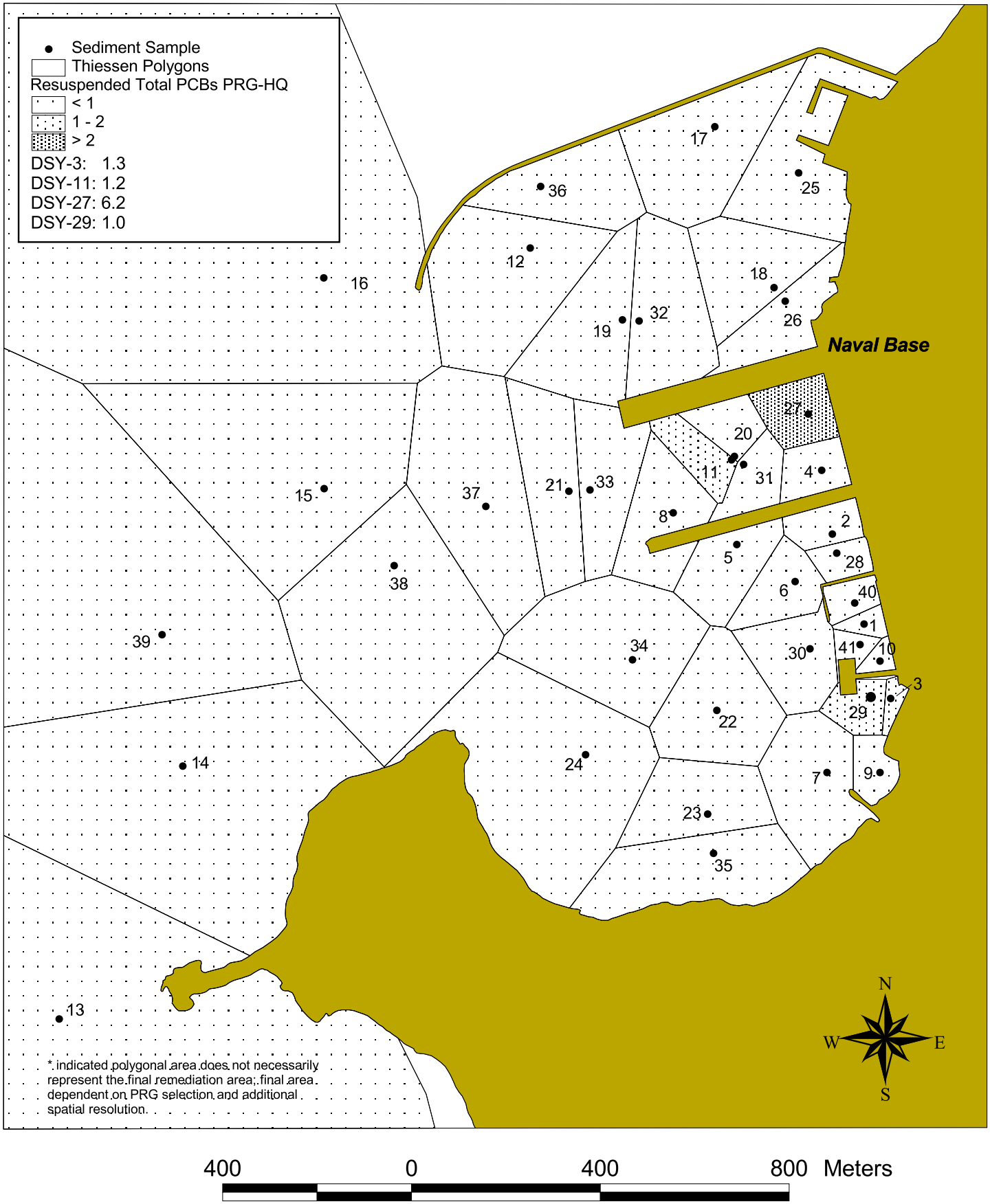


Figure 3.3-7. Summary of CoCs exceeding aquatic PRGs by location for sediments from the Derecktor Shipyard/Coddington Cove study area:*

Recommended PRG Implementation for the Resuspended Sediment Exposure Pathway

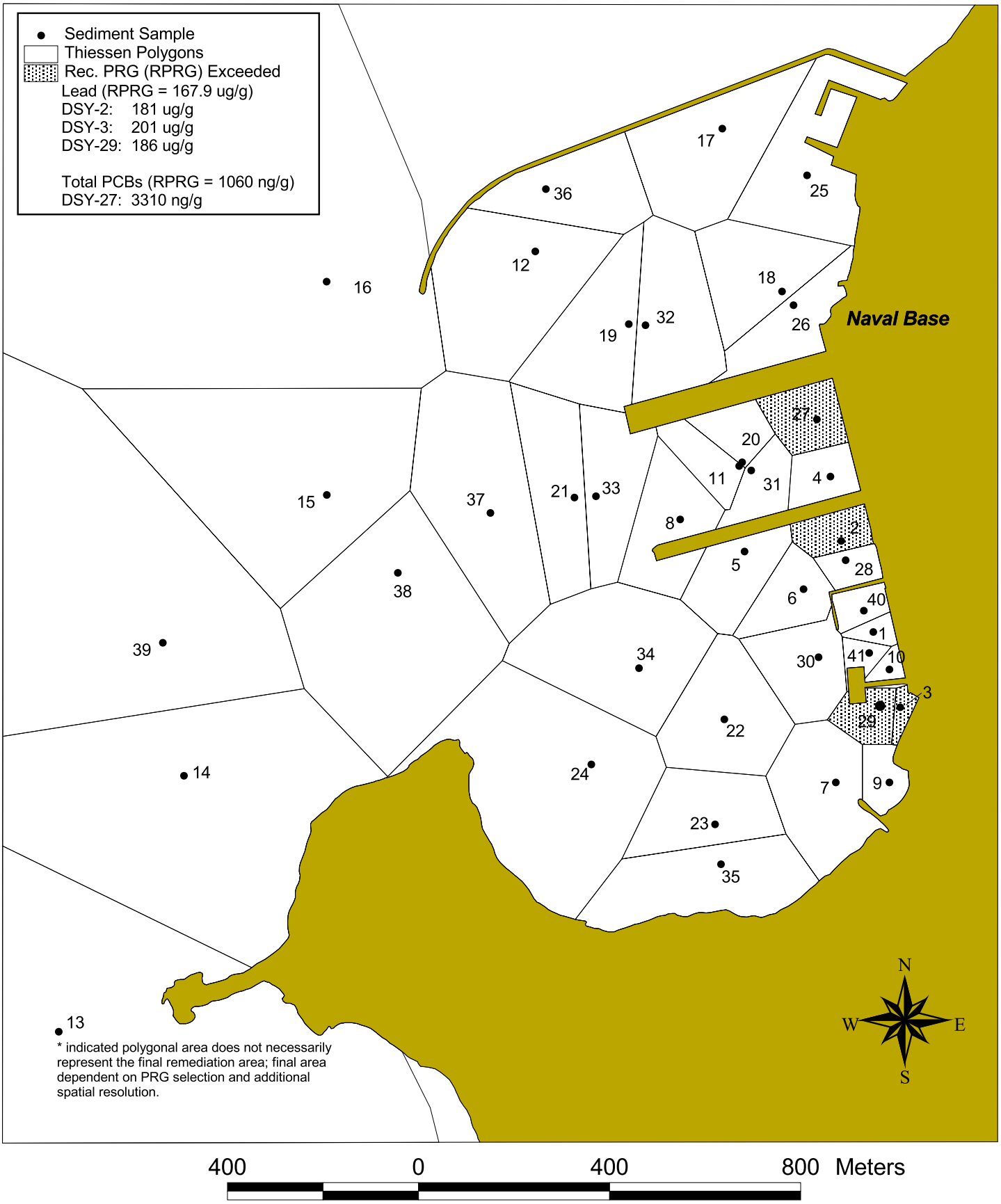


Figure 3.3-8. Summary of CoCs exceeding PRGs for protection of avian predators by location for sediments from the Derecktor Shipyard/Coddington Cove study area:*

Avian Predator Exposure Pathway for Zinc

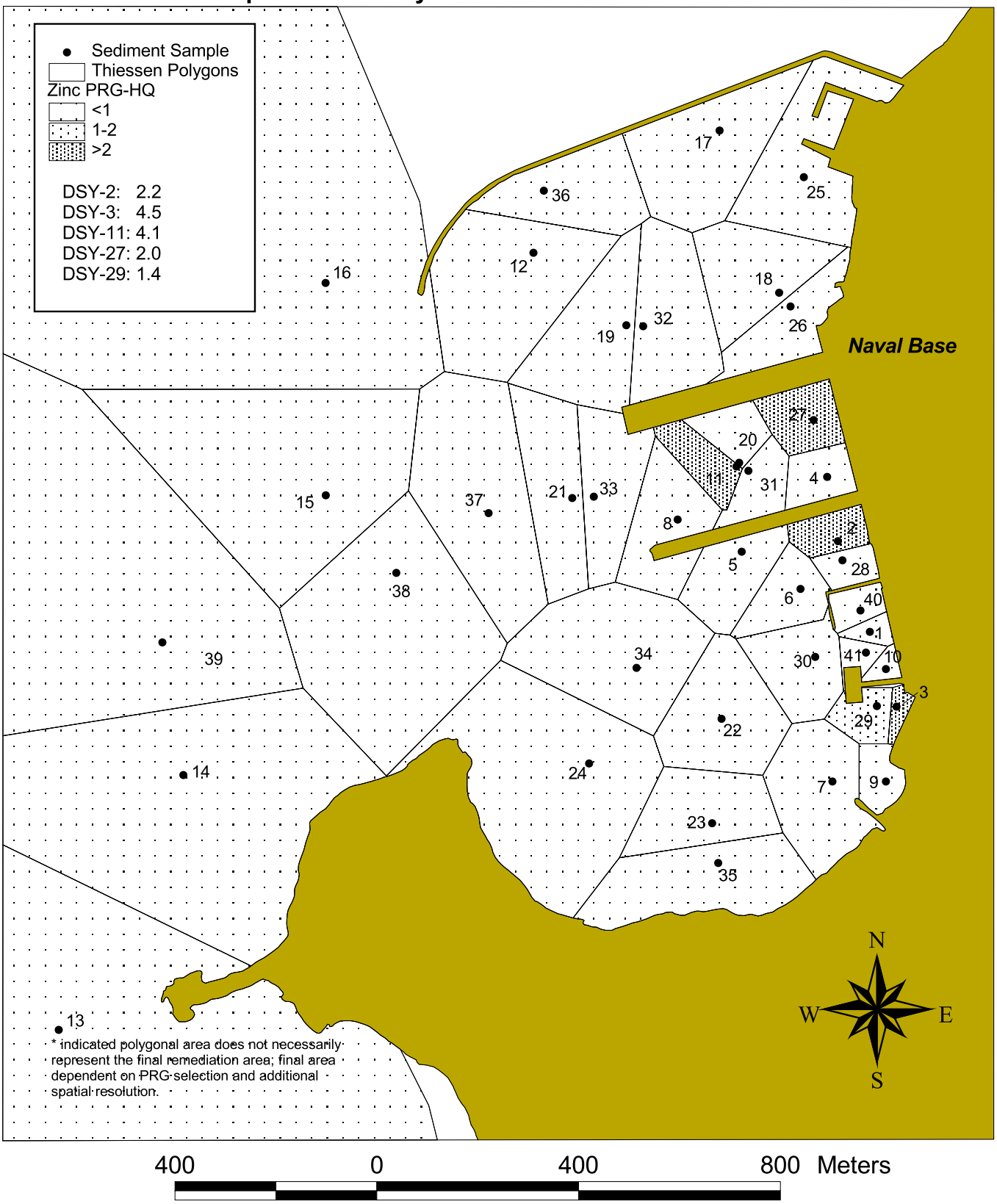


Figure 3.3-9. Summary of CoCs exceeding PRGs for protection of avian predators by location for sediments from the Derecktor Shipyard/Coddington Cove study area:*

Avian Predator Exposure Pathway for Total PCBs

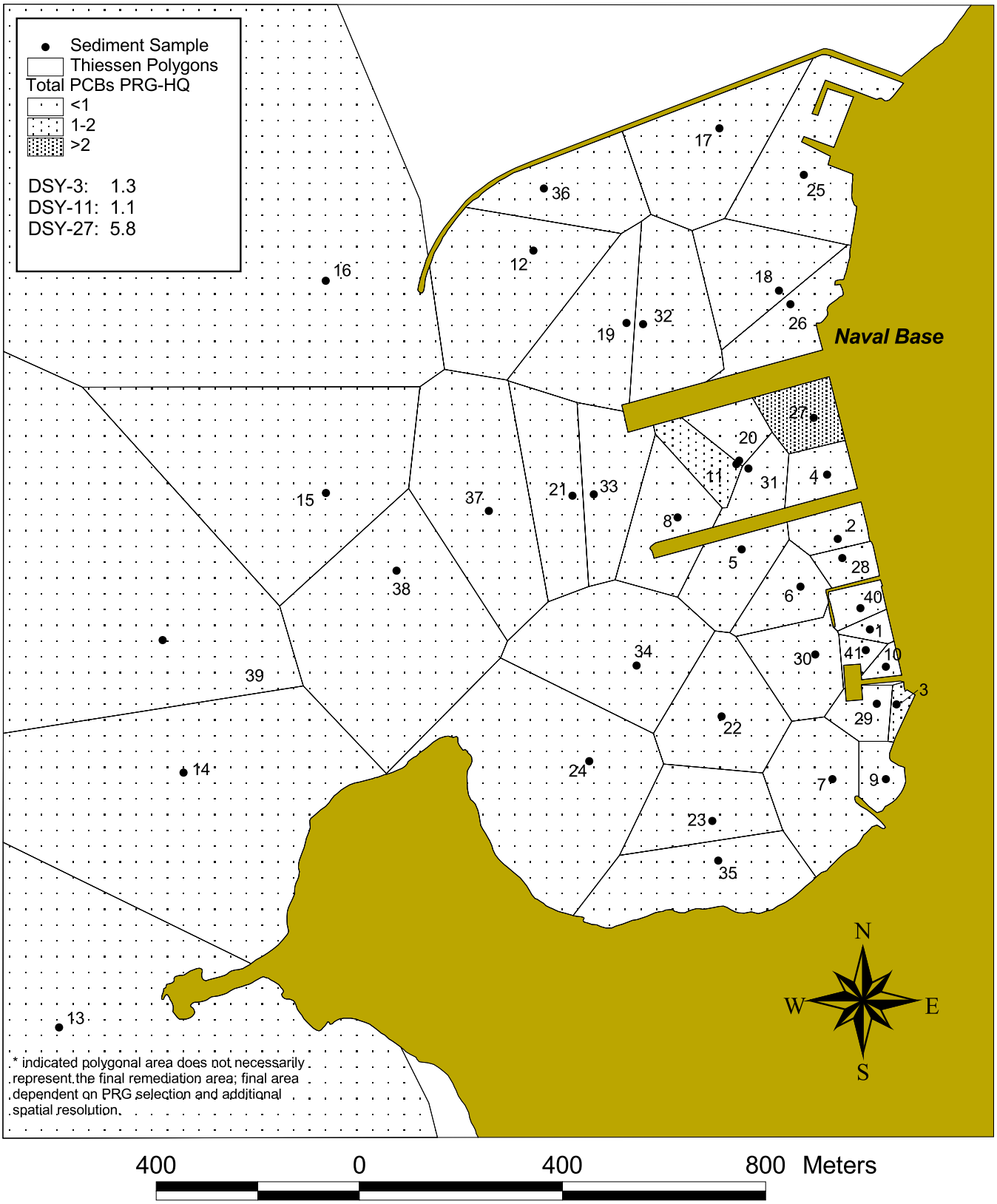


Figure 3.3-10. Summary of CoCs exceeding PRGs for protection of human health by location for sediments from the Derecktor Shipyard/Coddington Cove study area:*

Human Health Exposure Pathway for Benzo(a)pyrene

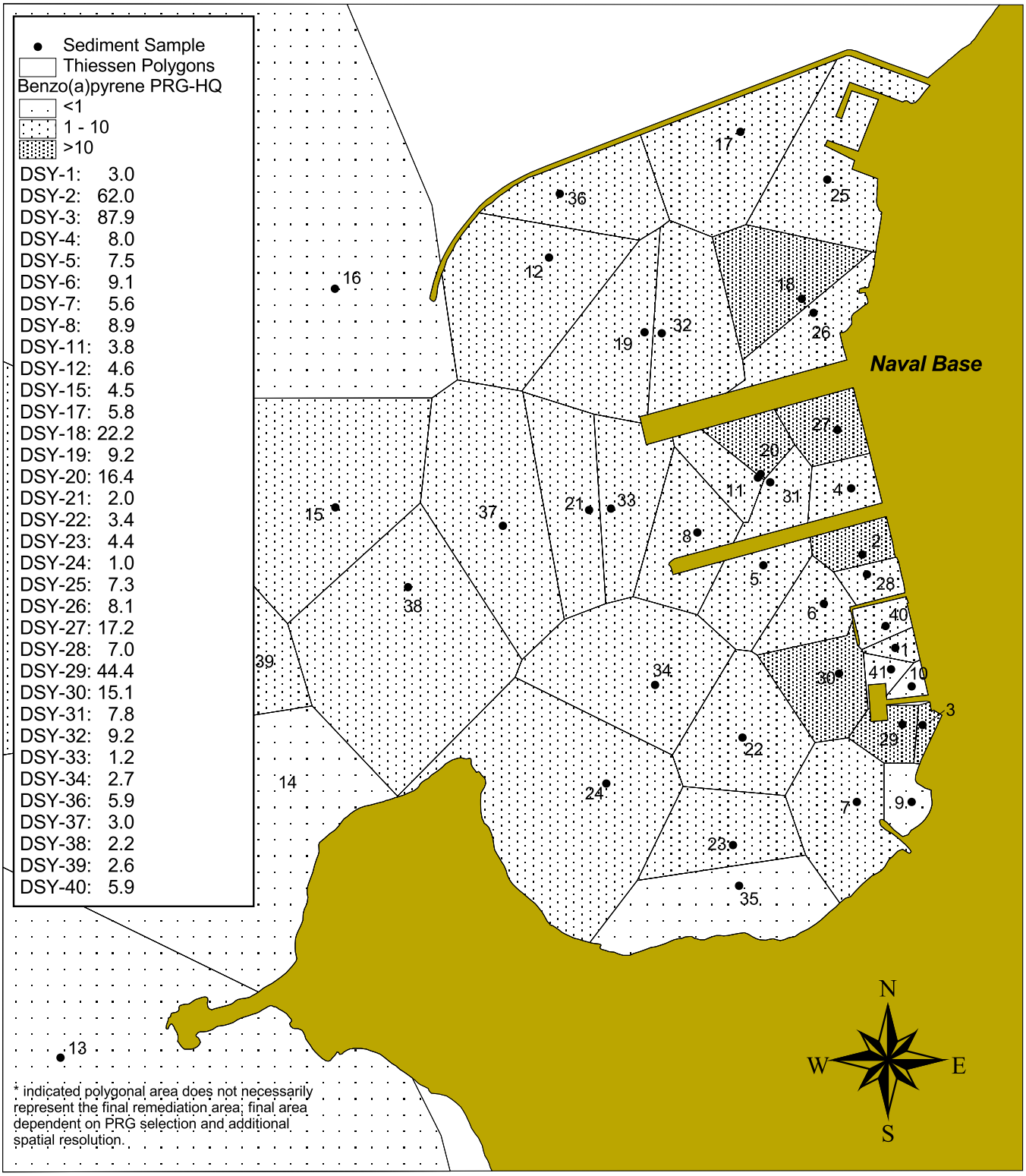
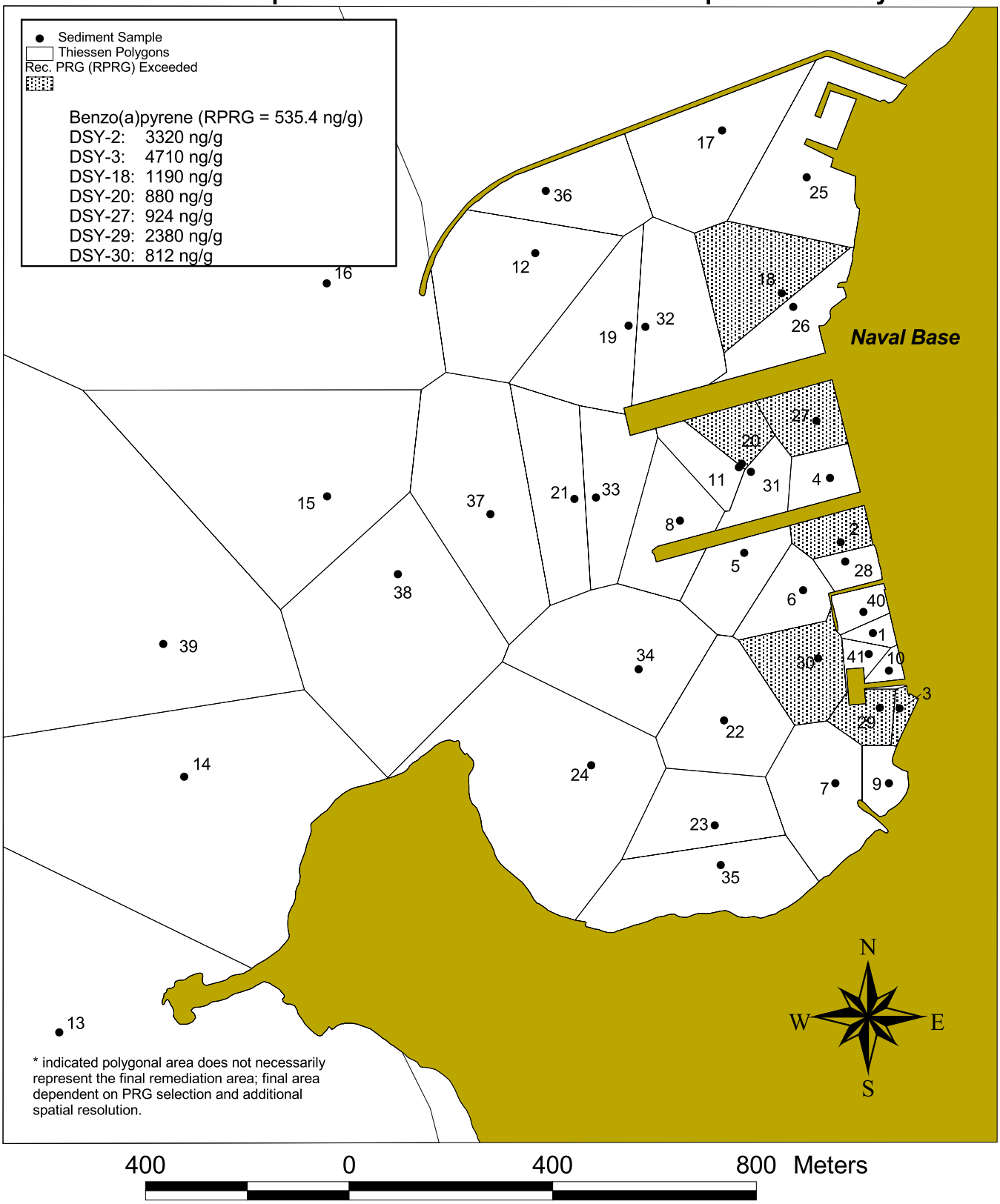


Figure 3.3-11. Summary of CoCs exceeding human health PRGs by location for sediments from the Derecktor Shipyard/Coddington Cove study area:*

Recommended PRG Implementation for the Human Health Exposure Pathway



TABLES

Table 1.2-1. Potential Remedial Action Objectives (RAOs) for the case study area.

Media/Receptor	Remedial Action Objectives (RAOs)
Aquatic Organisms	· Prevent exposure of aquatic organisms to bedded (in place) sediments with CoC concentrations exceeding the recommended PRGs. · Prevent exposure of aquatic organisms to sediments with CoC concentrations exceeding the recommended PRGs and that are present within areas where resuspension could occur.
Avian Predators	· Prevent exposure of avian predators to shellfish that are impacted by sediments with CoC concentrations exceeding the selected PRGs and are within areas where shellfish predation could regularly occur.
Human Health	· Prevent human ingestion of shellfish that are impacted by sediments with CoC concentrations exceeding the selected PRGs, and are within areas where shellfishing could regularly occur.

Table 1.2-2. Chemical-specific ARARs for the case study area.

MEDIA	REQUIREMENT	STATUS	SYNOPSIS	APPLICABILITY TO SITE CONDITIONS
Groundwater (Federal)	Federal Resource Conservation and Recovery Act (RCRA), Subpart F (40 CFR 264.94), Ground-Water Protection Standards and Alternate Concentration Levels.	To Be Considered	Allows for the development of Ambient Concentration Limit (ACL) for facilities which treat, store, or dispose of hazardous wastes when the characteristics of the ground water (e.g. high salinity) limit the application of Maximum Concentration Limits (MCLs) or health-based criteria. Exposure-based ACL may be developed which take into account potentially adverse effects on groundwater quality and hydraulically connected surface water quality.	Although currently undeveloped, ACL for groundwater may be relevant and appropriate to the development of site-specific Preliminary Remediation Goals (PRGs).
	Federal Clean Water Act (CWA) (33 USC 1251-1376); Clean Water Act, Water Quality Criteria, Section 404 (40 CFR 230)	To Be Considered	Non-enforceable guidelines established for the protection of human health and/or aquatic organisms. These guidelines are used by states to set water quality standards for surface water.	AWQC, with modification, may be relevant and appropriate for the development of PRGs for groundwater which enters a surface water.
	Federal Safe Drinking Water Act	Relevant and Appropriate	Establishes drinking water MCLs and health-based criteria.	Appropriate for the development of PRGs for remedial actions involving the discharge of treated groundwater.
Surface Water (Federal)	Federal Clean Water Act (CWA) (33 USC 1251-1376); Clean Water Act, Water Quality Criteria, Section 404 (40 CFR 230)	Relevant and Appropriate or Applicable	Non-enforceable guidelines established for the protection of human health and/or aquatic organisms. These guidelines are used by states to set water quality standards for surface water.	AWQC are relevant and appropriate to the development of PRGs for surface water. AWQC will also be applicable to remedial alternatives which involve discharges to surface water.
Soil/Sediment (Federal)	Toxicity Characteristic (40 CFR 261-24)	To Be Determined	Establishes maximum concentrations of CoC for the TCLP test method described in 40 CFR 261, Appendix II.	Applicable where wastes produced during remedial action require handling as a hazardous waste based upon results of Toxic Concentration Leachate Procedure (TCLP) analysis.
	Land Disposal Restrictions (40 CFR 268)	To Be Determined	Establishes maximum concentrations of CoCs on the basis of which hazardous wastes area restricted from land disposal.	Applicable to remedial alternatives which specify the land disposal of hazardous wastes.
	Toxic Substances Control Act (TSCA) (40 CFR 761.125)	Relevant and Appropriate	Establishes PCB cleanup levels for soils and solid surfaces.	Applicable to spills of materials containing PCBs at concentrations of 50 mg/kg or greater that occurred after May 4 1987. Although shipyard operations ceased in 1972, this regulation may still be relevant and appropriate for the development of the PRG.
	EPA Proposed Sediment Quality Criteria (Fed. Reg. Vol. 59, No. 11, 18 January 1994)	To Be Considered	Establishes proposed levels of five priority pollutants in fresh and saltwaters for the protection of benthic organisms.	To be considered for the development of PRGs.
	EPA Interim Sediment Criteria Values for Non-Polar Hydrophobic Organic Contaminants (EPA SCD#17 May 1988)	To Be Considered	Screening values for contaminants in sediments.	To be considered for the development of PRGs. EPA's proposed criteria are contained in the 1994 document (above).
	Interim Guidance on Establishing Soil Lead Cleanup Levels at Superfund Sites (OWSER 9355, 4-02)	To Be Considered	Sets as an interim soil cleanup level for lead at 500 to 1,000 mg/kg.	To be considered for the development of PRGs.
Groundwater (State)	Rules and Regulations for Groundwater Quality Criteria (CRIR No. 12-100-006)	To Be Considered	Establishes water classifications and water quality criteria. Also establishes acute and chronic water quality criteria for the protection of aquatic life.	Class GA Water Quality Standard (WQS), with modification, may be relevant and appropriate to the development of PRGs for groundwater based upon the potential discharge following treatment to fishable surface water.
	Groundwater Protection Act of 1985 (RIGL 46-13.1)	Applicable	Establishes the policy for maintaining and restoring groundwater quality and presents groundwater classifications.	Applicable to Class GB groundwater within the state of Rhode Island.
Surface Water (State)	Rules and Regulations for Groundwater Quality Criteria (CRIR No. 12-100-006)	Relevant and Appropriate or Applicable	Establishes water classifications and water quality criteria. Also establishes acute and chronic water quality criteria for the protection of aquatic life.	WQS are relevant and appropriate to the development of PRGs for surface water. WQS will also be applicable for remedial alternatives which involve discharges to surface water.
	Water Quality Regulations for Water Pollution Control	Applicable	Establishes water quality criteria and water classifications.	Applicable to Class SA surface water for the development of PRGs.
Soils (State)	Rules and Regulations for Lead Poisoning Prevention	Applicable	RIDEM, in conjunction with RIDOH, established a permissible level of lead in soil at 500 mg/kg for surface soils and 1,000 mg/kg for subsurface soils. A "lead-free" level in soil was defined as 150 mg/kg.	Applicable to the development of soil PRGs.
	RI Hazardous Waste Management Act of 1987 (RIGL 23-19.1 et seq.)	Relevant and Appropriate or Applicable	Defines Type 6 - Extremely hazardous waste at including wastes which contain PCB at a concentration of 50 mg/kg or greater or showing 10 $\mu g/100\ cm^2$ or greater as measured by a standard wipe test.	Relevant and appropriate for the development of soil PRGs. Applicable for remedial actions which involve handling hazardous wastes.
	Rules and Regulations for Solid Waste Management	Relevant and Appropriate or Applicable	Defines solid waste as including any soil, debris, or other material with a concentration of PCBs of 10 ppm or greater as measured by a standard wipe test.	Relevant and appropriate for the development of soil PRGs. Applicable for remedial action which involve handling solid wastes.

Table 1.3-1. Overall summary of exposure and effects-based weights of evidence and characterization of risk for the marine ecological risk assessment of the case study area.

	WEIGHTS OF EVIDENCE										
	EXPOSURE					EFFECTS					
Station	Sediment Hazard Quotients[1]	Elutriate HQs[2]	SEM and AVS[3]	Tissue Conc. Ratio[4]	Rank[9]	Tissue Residue Effects[5]	Laboratory Toxicity[6]	Field Effects[7]	Avian Predators[8]	Rank[9]	Overall Risk Probability Ranking[10]
DSY-24				++	I	+		-	+	L	Intermediate
DSY-25	+	+	-	+++	I	++	+	++	+	I	Intermediate
DSY-26	+		-	+++	I	+	++	++	+	I	Intermediate
DSY-27	+++	+	+	+++	H	+++	-	++	+	H	High
DSY-28	+		+	++	L	+	++	+	++	I	Intermediate
DSY-29	+++	+	+	++	H	+++	++	+++	++	H	High
DSY-30	+		+		L		-	+		B	Low
DSY-31	+++	+	-	+	I	+	+	+	+	L	Intermediate
DSY-32	+	+	-	+	L	+	+	++	+	L	Low
DSY-33	-	+	+	+	L	++	+	++	+	I	Intermediate
DSY-34	+		-	+	L	+	-	-	+	L	Low
DSY-35	-		+	+	L	++	-	-	+	L	Low
DSY-36	+	+	-	++	L	+	-	+	++	L	Low
DSY-37	+	+	+	+	L	+	+	-	+	L	Low
DSY-38	+	+	-	+	L	++	+	+	+	L	Low
DSY-39	+	+	-	+	L	++	+	+	+	L	Low
DSY-40	+	+	-	+	L	+	-	+++	+	I	Intermediate
DSY-41	-	+	-	+	L	+	+	+++	+	I	Intermediate
JPC-1[11]	-	+	-		B	+++	-	-	+	I	Low
JPC-2[11]	-		+		B		-	-	+	B	Baseline
CHC-1[11]						+++		-		I	Intermediate

Codes: - = baseline, + = low, ++ = medium, and +++ = high.
1- Sediment Hazard Quotient Risk Ranking.
2- Elutriate Hazard Quotient Risk Ranking.
3- Simultaneously extracted metals (SEM) and acid volatile sulfides (AVS) Risk Ranking.
4- Tissue Concentration Ratios Risk Ranking.
5- Tissue-based Risk Ranking: Based on Site vs. Reference Tissue Concentration Ratio, Tissue Screening Concentration and Critical Body Residues Risk Ranking.
6- Laboratory Toxicity Risk Ranking.
7- Field Effects Ranking: Based on results of Condition Index, Benthic Community Structure, Hematopoietic neoplasia, cytochrome P450, and fecal pollution indicators.
8- Avian Predator effects ranking based on Toxicity Reference Value Hazard Quotients.
9- Overall Exposure/Effects (E/E) Ranking:
B = Baseline Risk; L = Low Risk Probability; I = Intermediate Risk Probability; H = High Risk Probability.
Rankings for stations for which only one WoE observation was available are equal to the WoE observation ranking.
B = Low (+) E/E ranking observed for only one indicator or baseline E/E ranking observed for all indicators;
L = Intermediate (++) E/E ranking observed for only one indicator or low (+) E/E ranking observed for two or more indicators;
I = High (+++) E/E ranking observed for only one indicator or intermediate (++) E/E ranking observed for two or more indicators;
H = Intermediate (++) or greater E/E ranking observed for two indicators including high (+++) E/E ranking observed for one indicator.
10- Overall Risk Ranking:
Baseline = No greater than Baseline (B) ranking for E/E WoE summaries;
Low = No greater than Low (L) ranking for E/E WoE summaries, or Intermediate (I) ranking for one WoE summary and no greater than Baseline (B) ranking for the other WoE summary;
Intermediate = No greater than Intermediate (I) ranking for E/E WoE summaries, or High (H) ranking for one WoE and no greater than Low (L) ranking for the other WoE summary;
High = High (H) ranking for one WoE summary and Intermediate (I) or greater ranking for the other WoE summary.
11- References stations.

Table 2.0-1. Generic procedure for Preliminary Remediation Goals (PRGs) development for aquatic, avian predator and human health exposure pathways in the case study area.

PROCEDURES	EXPOSURE PATHWAY		
	Aquatic	**Avian Predator**	**Human Health**
1 - Consolidation of literature-based benchmarks into a single, pathway-specific list of "no effects concentrations" and application to site data for hazard normalization.	Benchmark = WQSV; HQ_{PW} = Porewater (PW) concentration/WQSV; HQ_{ELU} = Elutriate (ELU) concentration/WQSV.	Benchmark = TRV; HQ_{TRV} = Tissue concentration of prey species/TRV.	Benchmark = minimum RME value; HQ_{RME} = Shellfish tissue concentration/RME.
2 - Review of site-specific conditions and refinement of chemical exposure assumptions	Estimate 95% Upper Confidence Limit (95% UCL) of HQs_{PW} or HQs_{ELU} associated with non-toxic samples; set NOEQ = 1 where 95% UCL < 1.	TRV based on avian predator exposure model for species living in the New England region.	RME based on human health exposure model for recreational shellfishing characteristics in the New England region.
3 - Review of ERA/HHRA CoC list and modification based on refined exposure assumptions and predicted chemical hazards	Retain analytes for which the MAX HQ_{PW} or MAX HQ_{ELU} associated with toxic samples > NOEQ.	Retain analytes with $HQ_{TRV} > 1$.	Retain carcinogenic analytes with risk > $1x10^{-6}$; Retain non-carcinogenic analytes with HQ > 1.
4 - Refinement of pathway-specific no effects concentrations to account for background CoC levels (called threshold effects values)	Compare aquatic NOEC[1] and RSV for CoCs in porewater; select greater of two values as aquatic TEV.	Compare avian predator TRV and RSV for CoCs in prey species tissues; select greater of two values as avian predator TEV.	Compare human health RBV and RSV for CoCs in shellfish species tissues; select greater of two values as human health TEV.
5 - Assessment of CoC exceedences of threshold effects values to identify pathway-specific "Limiting" CoCs for PRG selection	Calcluate aquatic HQs_{TEV} as station-specific PW conc./TEV; identify MAX HQ_{TEV} by station; compile resulting list as "Limiting" aquatic CoCs for PRG development.	Calcluate avian predator HQs as station-specific prey tissue conc./TEV; identify MAX HQ_{TEV} by station; compile resulting list as "Limiting" avian predator CoCs for PRG development.	Calcluate human health HQs_{TEV} as station-specific shellfish tissue conc./TEV; identify MAX HQ_{TEV} by station; compile resulting list as "Limiting" human health CoCs for PRG development.
6 - Determine PRGs for "Limiting" CoCs, *i.e.*, convert TEV values in concentration-based units to be used during remediation.	For metal CoCs, use aquatic TEVs as PRGs (units = µg/L); derive organic PRGs (units = ng/g dry wt sediment) from TEV using EqP model.	Calculate PRGs (units = ng/g dry wt sediment) from avian TEVs using BAF (metals) and BSAF (organics) models.	Calculate PRGs (units = ng/g dry wt sediment) from human health TEVs using BAF (metals) and BSAF (organics) models.
7 - Evaluate practicality of pathway-specific PRGs for effective risk reduction.	Compare PRG exceedance to aquatic risk distribution.	Compare PRG exceedance to avian predator risk distribution.	Compare PRG exceedance to human health risk distribution.

[1] NOEQ x WQSV.
BAF = Bioaccumulation Factor
BSAF = Biota-Sediment Accumulation Factor
CoC = Chemical of Concern
ELU = Elutriate
WQSV = Water Quality Screening Value
HQ = Hazard Quotient
MAX = Maximum
NOEC = No Effect Concentration
NOEQ = No Effect Quotient
PRG = Prelimiary Remediation Goal
PW = Porewater
RME = Reasonable Maximum Exposure Value
RSV = Reference Screening Value
TEV = Threshold Effects Value
TRV = Toxicity Reference Value

Table 2.1-1. Water Quality Criteria for target analytes selected for aquatic PRG development and derived Water Quality Screening Values.

Chemical		EPA Water Quality Criteria (ug/L)				NOAA	WQSV (ug/L)	
Class	Analyte	WQC-FA	WQC-FC	WQC-SA	WQC-SC	ER-L	Conc.	Source
MET	Arsenic	360	190	69	36	8.2	36	A
MET	Cadmium	3.9	1.1	43	9.3	1.2	9.3	A
MET	Chromium	1700	210	1100	50	81	50	A
MET	Copper	18	12	2.9	2.9	34	2.9	A
MET	Lead	83	3.2	220	8.5	46.7	8.5	A
MET	Mercury	2.4	0.012	2.1	0.025	0.15	0.025	A
MET	Nickel	1400	160	75	8.3	20.9	8.3	A
MET	Silver	0.92	0.12	7.2	0.92	1	0.92	A
MET	Zinc	120	110	95	86	150	86	A
MET	SEM:AVS						5	F
PAH	1,6,7-Trimethylnaphthalene (L)							NA
PAH	1-Methylnaphthalene (L)							NA
PAH	1-Methylphenanthrene (L)							NA
PAH	2,6-Dimethylnaphthalene (L)							NA
PAH	2-Methylnaphthalene (L)					70	0.88	E
PAH	Acenaphthene (L)	1700	520	970	710	16	710	A
PAH	Acenaphthylene (L)					44	0.46	E
PAH	Anthracene (L)					85.3	0.29	E
PAH	Benzo(a)anthracene (H)					261	0.07	E
PAH	Benzo(a)pyrene (H)					430	0.04	E
PAH	Benzo(b)fluoranthene (H)							NA
PAH	Benzo(e)pyrene (H)							NA
PAH	Benzo(g,h,i)perylene (H)							NA
PAH	Benzo(k)fluoranthene (H)							NA
PAH	Biphenyl (L)							NA
PAH	Chrysene (H)					384	0.10	E
PAH	Dibenz(a,h)anthracene (H)					63.4	0.0017	E
PAH	Fluoranthene (H)	3980		40	16	600	16	A
PAH	Fluorene (L)					19	0.14	E
PAH	Indeno(1,2,3-cd)pyrene (H)							NA
PAH	Naphthalene (L)	2300	620	2350		160	293.75	B
PAH	Perylene (H)							NA
PAH	Phenanthrene (L)	30	6.3	7.7	4.6	240	4.60	A
PAH	Pyrene (H)					665	0.63	E
PAH	LMW PAHs					552	5.26	E
PAH	HMW PAHs					1700	0.29	E
PAH	Total PAHs					4022	5.09	E
PCB	Total PCBs	2	0.014	10	0.03	22.7	0.03	A
PST	Aldrin	3		1.3			0.16	B
PST	Hexachlorobenzene	6	3.68				3.68	C
PST	Mirex		0.001		0.001		0.001	A
PST	o,p'-DDE			0.13	0.001	2.2	0.001	A
PST	p,p'-DDE			0.13	0.001	2.2	0.001	A
TBT	Dibutyltin							NA

WQC-FA = Water Quality Criteria = Freshwater Acute Value
WQC-FC = Water Quality Criteria = Freshwater Chronic Value
WQC-SA = Water Quality Criteria = Saltwater Acute Value
WQC-SC = Water Quality Criteria = Saltwater Chronic Value
WQSV = Water Quality Screening Value
WQSV CODES:
NA= Benchmark not available to derive Screening Value
A- WQC-SC VALUE
B- 8:1 ACUTE/CHRONIC RATIO APPLIED TO WQ-SA VALUE (Shepard, 1995); * = Acute value based on LOAEL
C- WQC-FC VALUE
D- 8:1 ACUTE/CHRONIC RATIO APPLIED TO WQ-FA VALUE (Shepard, 1995).
E- EQUILIBRIUM PARTITIONING OF ER-L SEDIMENT BENCHMARK INTO POREWATER AT 1% TOC
F- U.S. EPA, 1997b
LMW PAH = ten 2-ring & 3-ring PAHs (NOAA, 1991)
HMW-PAH = eight 4-ring and 5-ring PAHs (NOAA, 1991)
HMW PAH Kow = median of analyte specific Kows
Total PAH = sum of LMW and HMW PAHs (NOAA, 1991)

Table 2.1-2. Summary of partitioning coefficients used in calculations of organic contaminant concentrations in porewaters by equilibrium partitioning.

Class	Analyte	CAS No.	Full Analyte Name	LogKow	Source[1]	LogKoc[2]	Koc
MET	As	7440382	Arsenic	NA			
	Cd	7440439	Cadmium	NA			
	Cr	7440473	Chromium	NA			
	Cu	7440508	Copper	NA			
	Pb	7439921	Lead	NA			
	Hg	7439976	Mercury	NA			
	Ni	7440020	Nickel	NA			
	Ag	7440224	Silver	NA			
	Zn	7440666	Zinc	NA			
	SEM:AVS		SEM-AVS	NA			
PAH	T167NAP	2245387	1,6,7-Trimethylnaphthalene	4.61	b	4.53	3.40E+4
	M1NAPH	90120	1-Methylnaphthalene	3.97	b	3.90	7.99E+3
	M1PHEN	832699	1-Methylphenanthrene	5.08	b	4.99	9.86E+4
	D26NAPH	581420	2,6-Dimethylnaphthalene	4.61	b	4.53	3.40E+4
	M2NAPH	91576	2-Methylnaphthalene	3.97	b	3.90	7.99E+3
	ACENAPH	83329	Acenaphthene	3.92	a	3.85	7.14E+3
	ACENAPL	208968	Acenaphthylene	4.05	b	3.98	9.58E+3
	ANTHRAC	120127	Anthracene	4.55	a	4.47	2.97E+4
	BENAAN	56553	Benzo(a)anthracene	5.70	a	5.60	4.01E+5
	BENAPYR	50328	Benzo(a)pyrene	6.11	a	6.01	1.01E+6
	BENBFLU	205992	Benzo(b)fluoranthene	6.20	a	6.09	1.24E+6
	BENEPYR	192972	Benzo(e)pyrene	6.11	b	6.01	1.01E+6
	BGHIPER	191242	Benzo(g,h,i)perylene	6.70	a	6.59	3.86E+6
	BENKFLU	207089	Benzo(k)fluoranthene	6.20	a	6.09	1.24E+6
	BIPHEN	92524	Biphenyl	3.96	a	3.89	7.82E+3
	CHRYSEN	218019	Chrysene	5.70	a	5.60	4.01E+5
	DBAHANT	53703	Dibenz(a,h)anthracene	6.69	a	6.58	3.77E+6
	FLUORAN	206440	Fluoranthene	5.12	a	5.03	1.08E+5
	FLUOREN	86737	Fluorene	4.21	a	4.14	1.38E+4
	I123CDP	193395	Indeno(1,2,3-cd)pyrene	6.65	a	6.54	3.45E+6
	NAPH	91203	Naphthalene	3.36	a	3.30	2.01E+3
	PERYL	198550	Perylene	6.05	b	5.95	8.86E+5
	PHENAN	85018	Phenanthrene	4.55	a	4.47	2.97E+4
	PYRENE	129000	Pyrene	5.11	a	5.02	1.06E+5
	LMWPAH	NA	Low Molecular Weight PAHs[3]	4.09	c	4.02	1.05E+4
	HMWPAH	NA	High Molecular Weight PAHs[3]	5.88	c	5.78	5.96E+5
	TOTPAH	NA	Total PAHs[3]	4.98	c	4.90	7.91E+4
PCB	PCB101	37680732	101 (2 2'3 5 5')	6.38	b	6.27	1.87E+6
	PCB105	32598144	105 (2 3 3'4 4')	6.65	b	6.54	3.45E+6
	PCB118	31508006	118 (2 3'4 4'5)	6.74	b	6.63	4.22E+6
	PCB128	39380073	128 (2 2'3 3'4 4')	6.74	b	6.63	4.22E+6
	PCB138	35065282	138 (2 2'3 4 4'5)	6.83	b	6.71	5.18E+6
	PCB153	35065271	153 (2 2'4 4'5 5')	6.92	b	6.80	6.35E+6
	PCB170	35065306	170 (2 2'3 3'4 4'5)	7.27	b	7.15	1.40E+7
	PCB18	37680652	18 (2 2'5)	5.24	b	5.15	1.42E+5
	PCB180	35065293	180 (2 2'3 4 4'5 5')	7.36	b	7.24	1.72E+7
	PCB187	52663680	187 (2 2'3 4'5 5'6)	7.17	b	7.05	1.12E+7
	PCB195	52663782	195 (2 2'3 3'4 4'5 6)	7.56	b	7.43	2.70E+7
	PCB206	40186729	206 (2 2'3 3'4 4'5 5'6)	8.09	b	7.95	8.97E+7
	PCB209	2051243	209 (2 2'3 3'4 4'5 5'6 6')	8.18	b	8.04	1.10E+8
	PCB28	7012375	28 (2 4 4')	5.67	b	5.57	3.75E+5
	PCB44	41464395	44 (2 2'3 5')	5.75	b	5.65	4.49E+5
	PCB52	35693993	52 (2 2'5 5)	5.84	b	5.74	5.51E+5
	PCB66	32598100	66 (2 3'4 4')	6.20	b	6.09	1.24E+6
	PCB8	34883437	8 (2 4)	5.07	b	4.98	9.64E+4
	TOTPCB	NA	Total PCBs[4]	6.54	b	6.43	2.69E+6
PST	ALDRIN	309002	Aldrin	6.50	a	6.39	2.45E+6
	HCB	118741	Hexachlorobenzene	5.89	a	5.79	6.17E+5
	MIREX	2385855	Mirex	6.89	a	6.77	5.93E+6
	DDE_OP	3424826	o,p'-DDE	6.76	a	6.65	4.42E+6
	DDE_PP	72559	p,p'-DDE	6.76	a	6.65	4.42E+6

1 - Literature source of LogKow values:

a - Karickhoff and Long, 1995.

b - Karickhoff *et al*., 1989.

c - Calculated value

2 - $\log_{10}(Koc) = 0.00028 + 0.983*\log_{10}(Kow)$; Karickhoff *et al.*, 1989.

3 - LMW PAH = ten 2-ring & 3-ring PAHs; HMW-PAH = eight 4-ring and 5-ring PAHs; Total PAH = sum of LMW and HMW PAHs (NOAA, 1991)LMW PAH, HMW PAH Kow = median of analyte specific Kow, Total PAH Kow = mean of LMW, HMW PAH Kow

4 - Sum of Congeners X 2

NA= not applicable, Kow = Octanol-water partitioning coefficient; Koc = organic carbon partition coefficient

Table 2.1-3. Distribution of toxic and non-toxic aquatic Hazard Qoutients and derivation of No Effect Quotients for the aquatic receptors exposed to analytes via bedded and resuspended sediment aquatic exposure pathways for the case study area.

A. BEDDED SEDIMENT		Amphipod Survival						
		Non-Toxic Samples			Toxic Samples			
Class	Analyte[1]	N	95% UCL HQ_{PW}[2]	Test NOEQ[3]	N	MAX HQ_{PW}[2]	MAX HQ_{PW} > NOEQ?	PATH NOEQ[4]
MET	Arsenic	0			0		NO	
MET	Cadmium	0			0		NO	
MET	Chromium	0			0		NO	
MET	Copper	0			0		NO	
MET	Lead	0			0		NO	
MET	Mercury	0			0		NO	
MET	Nickel	0			0		NO	
MET	Silver	0			0		NO	
MET	Zinc	0			0		NO	
MET	SEM:AVS[1]	15	-0.67	5.00	2	-11.10	NO	
PAH	1,6,7-Trimethylnaphthalene (L)	0			0		NO	
PAH	1-Methylnaphthalene (L)	0			0		NO	
PAH	1-Methylphenanthrene (L)	0			0		NO	
PAH	2,6-Dimethylnaphthalene (L)	0			0		NO	
PAH	2-Methylnaphthalene (L)	15	0.16	1.00	2	0.16	NO	
PAH	Acenaphthene (L)	15	3.60E-04	1.00	2	1.27E-04	NO	
PAH	Acenaphthylene (L)	15	0.84	1.00	2	0.87	NO	
PAH	Anthracene (L)	15	1.38	1.38	2	1.21	NO	
PAH	Benzo(a)anthracene (H)	15	0.73	1.00	2	0.83	NO	
PAH	Benzo(a)pyrene (H)	15	0.45	1.00	2	0.58	NO	
PAH	Benzo(b)fluoranthene (H)	0			0		NO	
PAH	Benzo(e)pyrene (H)	0			0		NO	
PAH	Benzo(g,h,i)perylene (H)	0			0		NO	
PAH	Benzo(k)fluoranthene (H)	0			0		NO	
PAH	Biphenyl (L)	0			0		NO	
PAH	Chrysene (H)	15	0.59	1.00	2	0.64	NO	
PAH	Dibenz(a,h)anthracene (H)	15	0.45	1.00	2	0.56	NO	
PAH	Fluoranthene (H)	15	0.02	1.00	2	0.01	NO	
PAH	Fluorene (L)	15	1.23	1.23	2	0.87	NO	
PAH	Indeno(1,2,3-cd)pyrene (H)	0			0		NO	
PAH	Naphthalene (L)	15	1.22E-03	1.00	2	9.01E-04	NO	
PAH	Perylene (H)	0			0		NO	
PAH	Phenanthrene (L)	15	0.67	1.00	2	0.38	NO	
PAH	Pyrene (H)	15	0.64	1.00	2	0.79	NO	
PAH	LMW PAHs	15	3.38	3.38	2	2.58	NO	
PAH	HMW PAHs	15	2.87	2.87	2	3.41	YES	2.87
PAH	Total PAHs	15	6.22	6.22	2	5.99	NO	
PCB	Total PCBs	15	0.11	1.00	2	1.78	YES	1.00
PST	Aldrin	15	1.91E-05	1.00	2	6.72E-06	NO	
PST	Hexachlorobenzene	15	2.83E-06	1.00	2	1.46E-06	NO	
PST	Mirex	15	6.14E-03	1.00	2	0.01	NO	
PST	o,p'-DDE	15	0.02	1.00	2	0.40	NO	
PST	p,p'-DDE	15	0.02	1.00	2	0.04	NO	

1 - SEM-AVS expressed as µmol/g dry wt. sediment (benchmark from U.S.EPA, 1996).

2 - HQ_{PW}=Porewater Hazard Quotient.

3 - NOEQ = No Effect Quotient = greater of 95% Upper Confidence Limit (UCL) HQ or 1.

4 - If MAX HQ_{PW}>test NOEQ, Pathway NOEQ = test NOEQ

Table 2.1-3. Continued.

B. RESUSPENDED SEDIMENT		Sea Urchin Fertilization							Sea Urchin Larval Development							
		Non-Toxic Samples			Toxic Samples				Non-Toxic Samples			Toxic Samples				
Class	Analyte[1]	N	95% UCL HQ	NOEQ[2]	N	MAX HQ	MAX HQ > NOEQ?	FERT[3A] NOEQ	N	95% UCL HQ	NOEQ[2]	N	MAX HQ	MAX HQ > NOEQ?	DEV[3B] NOEQ	PATH[4] NOEQ
MET	Arsenic	9	1.29	1.29	0		NO		2	1.12	1.12	7.00	2.11	YES	1.12	1.12
MET	Cadmium	9	0.01	1.00	0		NO		2	0.01	1.00	7.00	0.01	NO		
MET	Chromium	9	8.00E-03	1.00	0		NO		2	8.00E-03	1.00	7.00	8.00E-03	NO		
MET	Copper	9	0.91	1.00	0		NO		2	0.43	1.00	7.00	1.76	YES	1.00	1.00
MET	Lead	9	1.54	1.54	0		NO		2	1.06	1.06	7.00	1.87	YES	1.06	1.06
MET	Mercury	9	4.00	4.00	0		NO		2	4.00	4.00	7.00	4.00	NO		
MET	Nickel	9	0.48	1.00	0		NO		2	0.48	1.00	7.00	0.48	NO		
MET	Silver	9	0.27	1.00	0		NO		2	0.27	1.00	7.00	0.27	NO		
MET	Zinc	9	0.05	1.00	0		NO		2	0.05	1.00	7.00	0.05	NO		
PAH	1,6,7-Trimethylnaphthalene (L)	0			0		NO		0			0		NO		
PAH	1-Methylnaphthalene (L)	0			0		NO		0			0		NO		
PAH	1-Methylphenanthrene (L)	0			0		NO		0			0		NO		
PAH	2,6-Dimethylnaphthalene (L)	0			0		NO		0			0		NO		
PAH	2-Methylnaphthalene (L)	9	9.47E-03	1.00	0		NO		2	6.57E-03	1.00	7.00	0.01	NO		
PAH	Acenaphthene (L)	11	1.37E-05	1.00	0		NO		3	3.45E-06	1.00	8.00	3.90E-05	NO		
PAH	Acenaphthylene (L)	11	0.01	1.00	0		NO		3	0.01	1.00	8.00	0.03	NO		
PAH	Anthracene (L)	10	0.07	1.00	0		NO		2	0.05	1.00	8.00	0.16	NO		
PAH	Benzo(a)anthracene (H)	10	0.40	1.00	0		NO		2	0.19	1.00	8.00	0.71	NO		
PAH	Benzo(a)pyrene (H)	11	0.46	1.00	0		NO		3	0.28	1.00	8.00	0.97	NO		
PAH	Benzo(b)fluoranthene (H)	0			0		NO		0			0		NO		
PAH	Benzo(e)pyrene (H)	0			0		NO		0			0		NO		
PAH	Benzo(g,h,i)perylene (H)	0			0		NO		0			0		NO		
PAH	Benzo(k)fluoranthene (H)	0			0		NO		0			0		NO		
PAH	Biphenyl (L)	0			0		NO		0			0		NO		
PAH	Chrysene (H)	9	0.16	1.00	0		NO		2	0.06	1.00	7.00	0.33	NO		
PAH	Dibenz(a,h)anthracene (H)	11	5.53	5.53	0		NO		3	6.84	6.84	8.00	6.84	NO		
PAH	Fluoranthene (H)	11	3.33E-03	1.00	0		NO		3	1.19E-03	1.00	8.00	7.36E-03	NO		
PAH	Fluorene (L)	11	0.07	1.00	0		NO		3	0.04	1.00	8.00	0.18	NO		
PAH	Indeno(1,2,3-cd)pyrene (H)	0			0		NO		0			0		NO		
PAH	Naphthalene (L)	9	4.09E-05	1.00	0		NO		2	3.26E-05	1.00	7.00	4.97E-05	NO		
PAH	Perylene (H)	0			0		NO		0			0		NO		
PAH	Phenanthrene (L)	11	3.47E-03	1.00	0		NO		3	2.45E-03	1.00	8.00	6.24E-03	NO		
PAH	Pyrene (H)	11	0.31	1.00	0		NO		3	0.44	1.00	8.00	0.49	NO		
PAH	LMW PAHs	11	0.14	1.00	0		NO		3	0.07	1.00	8.00	0.36	NO		
PAH	HMW PAHs	11	6.41	6.41	0		NO		3	7.32	7.32	8.00	8.37	YES	7.32	7.32
PAH	Total PAHs	11	6.51	6.51	0		NO		3	7.37	7.37	8.00	8.45	YES	7.37	7.37
PCB	Total PCBs	11	1.86	1.86	0		NO		3	2.31	2.31	8.00	2.59	YES	2.31	2.31
PST	Aldrin	11	0.01	1.00	0		NO		3	9.82E-03	1.00	8.00	9.82E-03	NO		
PST	Hexachlorobenzene	11	1.25E-04	1.00	0		NO		3	2.45E-04	1.00	8.00	2.45E-04	NO		
PST	Mirex	11	0.49	1.00	0		NO		3	0.60	1.00	8.00	0.71	NO		
PST	o,p'-DDE	11	2.99	2.99	0		NO		3	2.80	2.80	8.00	3.52	YES	2.80	2.80
PST	p,p'-DDE	11	0.59	1.00	0		NO		3	0.59	1.00	8.00	0.71	NO		

HQ_{ELU}=Elutriate Hazard Quotient.

1 - SEM concentration used; AVS assumed = 0 in suspended sediment.

2 - NOEQ = No Effect Quotient = greater of 95% Upper Confidence Limit (UCL) HQ or 1.

3A - If MAX HQ> NOEQ, FERT NOEQ = NOEQ

3B - If MAX DEV HQ > NOEQ, DEV NOEQ = NOEQ

4 - Pathway NOEQ = minimum of TEST-specific NOEQs

Table 2.1-4. Derivation of Threshold Efect Values for aquatic exposure pathways in the case study area.

Exposure Pathway	Class	CoC[1]	Aquatic NOEQ[2]	WQSV[3] (µg/L)	NOEC[4] (µg/L)	Aquatic RSV[5] (µg/L)	Aquatic TEV[6] (µg/L)
PW	PAH	HMW PAHs	2.87	0.29	0.82	0.18	0.82
PW	PCB	Total PCBs	1.00	0.03	0.03	1.96E-04	0.03
ELU	MET	Arsenic	1.12	36.00	40.40	18.30	40.40
ELU	MET	Copper	1.00	2.90	2.90	1.25	2.90
ELU	MET	Lead	1.06	8.50	9.00	13.20	13.20
ELU	PAH	HMW PAHs	7.32	0.29	2.09	0.21	2.09
ELU	PAH	Total PAHs	7.37	5.09	37.46	0.24	37.46
ELU	PCB	Total PCBs	2.31	0.03	0.07	0.05	0.07
ELU	PST	o,p'-DDE	2.80	0.0010	0.0028	0.0036	0.0036

NOEQ = No Effect Quotient
WQSV = Water Quality Screening Value
NOEC = No Effect Concentration
RSV = Reference Screening Value (regional background).
TEV = Threshold Effect Value
1 - List includes analytes for which Aquatic NOEQs were developed; see Table 2.1-3.
2 - Aquatic NOEQ = minimum of exposure pathway-specific NOEQs taken from Table 2.1-3.
3 - WQSV values are from Table 2.1-3.
4 - NOEC = Aquatic NOEQ x WQSV.
5 - Aquatic RSV reference data compiled by SAIC.
6 - Aquatic TEV is the greater of the NOEC or RSV.

Table 2.1-5. Summary of bedded and resuspended pathway Hazard Quotients and identification of "Limiting" CoCs for the aquatic exposure pathway in the case study area.

Class	CoC	Aquatic Pathway	DSY-25	DSY-26	DSY-27	DSY-28	DSY-29	DSY-30	DSY-31	DSY-32	DSY-33	DSY-34	DSY-35	DSY-36	DSY-37	DSY-38	DSY-39	DSY-40	DSY-41
PAH	HMW PAHs	PW	0.95	1.50	1.13	0.42	2.44	1.20	0.49	0.61	0.18	0.21	0.03	0.36	0.40	0.22	0.25	1.80	0.15
PCB	Total PCBs	PW	0.15	0.16	1.78	0.06	0.08	0.15	0.13	0.12	0.05	0.04	0.04	0.06	0.08	0.04	0.04	0.08	0.06
	SUM HQ_{TEV}		1.09	1.65	2.91	0.48	2.52	1.35	0.62	0.74	0.23	0.25	0.07	0.42	0.49	0.26	0.30	1.88	0.21
	MAX HQ_{TEV}		0.95	1.50	1.78		2.44	1.20										1.80	
	Limiting CoC		HMW PAHs	HMW PAHs	Total PCBs		HMW PAHs	HMW PAHs										HMW PAHs	
MET	Arsenic	ELU	0.31						0.67	0.61	0.06			1.00	0.49	1.01	1.88	0.74	
MET	Copper	ELU	0.43						1.76	0.43	0.43			0.43	0.43	0.43	0.43	0.43	
MET	Lead	ELU	0.71						0.64	0.71	1.20			0.63	0.98	0.81	1.11	0.68	
PAH	HMW PAHs	ELU	0.21		0.11		0.24		0.06	0.06	0.05			0.03	0.03	0.02	0.03	0.16	
PAH	Total PAHs	ELU	0.02		0.01		0.01		0.00	0.00	0.00			0.00	0.00	0.00	0.00	0.01	
PCB	Total PCBs	ELU	1.12		1.00		0.78		0.85	0.56	0.38			0.38	0.54	0.28	0.37	0.53	
PST	o,p'-DDE	ELU	0.98		0.78		0.83		0.89	0.53	0.37			0.57	0.94	0.40	0.62	0.69	
	SUM HQ_{TEV}		3.76	0.00	1.89	0.00	1.85	0.00	4.88	2.91	2.50	0.00	0.00	3.04	3.40	2.95	4.44	3.23	0.00
	MAX HQ_{TEV}		1.12		1.00		0.83		1.76	0.71	1.20			1.00	0.98	1.01	1.88	0.74	
	Limiting CoC		Total PCBs		Total PCBs		o,p'-DDE		Copper	Lead	Lead			Arsenic	Lead	Arsenic	Arsenic	Arsenic	

HQ_{TEV} = CoC concentration/TEV; see Table 2.1-4.

SUM HQ_{TEV} = sum HQ for all CoCs for which TEVs were developed and having similar mode of toxic action (narcosis).

PW = Bedded sediment exposure pathway, CoCs measured in sediment porewater.

ELU = Resuspended sediment exposure pathway, CoCs measured in sediment elutriates.

Table 2.2-1. Documentation of Toxicity Reference Values used for calculation of risks for the avian predator exposure pathway in the case study area.

		RoC		TEST SPECIES DATA							RECEPTOR DATA			
Chemical Class	Target Analyte	Common name	BW (kg)	Test Species	BW (Kg)	Endpoint	Endpoint Value	Reference	Safety Factor	Test Species NOEC	RoC NOEC	Food (Prey) FCR	Food (Prey) *f*	RoC TRV
MET	Arsenic	Gull	1.00	Mallard duck	1.000	Chronic NOEC	5.14	Sample *et al.*, 1996	1	5.14	5.14	0.06	0.06	80.2
MET	Cadmium	Gull	1.00	Mallard duck	1.000	Chronic NOEC	1.15	Sample *et al.*, 1996	1	1.15	1.15	0.06	0.06	18.0
MET	Chromium	Gull	1.00	Black duck	1.250	Chronic NOEC	1.00	Sample *et al.*, 1996	1	1.00	1.08	0.06	0.06	16.8
MET	Copper	Gull	1.00	Chicken, 1-70 days old	0.534	Chronic NOEC	47.0	Sample *et al.*, 1996	1	47.0	38.1	0.06	0.06	595.2
MET	Lead	Gull	1.00	American kestrel	0.130	Chronic NOEC	3.85	Sample *et al.*, 1996	1	3.85	1.95	0.06	0.06	30.4
MET	Mercury	Gull	1.00	Japanese Quail	0.150	Chronic NOEC	0.45	Sample *et al.*, 1996	1	0.45	0.24	0.06	0.06	3.7
MET	Silver	Gull	1.00	Mallard duck (juvenile)	0.600	4 wk. NOEC	8.30	Van Vleet, 1982	10	0.83	0.70	0.06	0.06	10.9
MET	Zinc	Gull	1.00	White Leghorn Hens	1.935	Chronic NOEC	14.5	Sample *et al.*, 1996	1	14.5	18.1	0.06	0.06	282.0
PCB	Total PCBs (c)	Gull	1.00	Ring-necked pheasant	1.000	Chronic NOEC	0.18	Sample *et al.*, 1996	1	0.18	0.18	0.06	0.06	2.8

TRV = Toxicity Reference Value
RoC = Receptor of Concern
BW = Body Weight
NOEC = No Effect Concentration
Endpoint Value = mg CoC/kg-BW/day
Safety Factor = conversion factor for non-chronic NOAEL data
Test Species NOEC = No Effect Concentration (mg CoC/kg-RoC/day) for test species
Wildlife NOEC = test species NOEC x (BW test species/BW of RoC)
FCR = Food Consumption Rate (kg prey/day)
f = FCR/BW

Table 2.2-2. Derivation of Threshold Effects Values for the avian predator exposure pathway in the case study area.

Exposure Pathway[1]	Class	CoC	Avian Predator TRV[2] (mg/kg prey dry tiss. wt.)	Avian Predator RSV[3] (mg/kg prey dry tiss. wt.)	Avian Predator TEV[4] (mg/kg prey dry tiss. wt.)
AVIAN	MET	Arsenic	80.16	8.17	80.16
AVIAN	MET	Cadmium	17.95	0.60	17.95
AVIAN	MET	Chromium	16.82	1.99	16.82
AVIAN	MET	Copper	595.2	20.81	595.2
AVIAN	MET	Lead	30.44	0.72	30.44
AVIAN	MET	Mercury	3.73	0.13	3.73
AVIAN	MET	Silver	10.93	1.28	10.93
AVIAN	MET	Zinc	282.0	101.2	282.0
AVIAN	PCB	Total PCBs	2.81	0.58	2.81

TRV = Toxicity Reference Value
RSV = Reference Screening Value
TEV = Threshold Effects Value
1 - List includes analytes identified as CoCs in the ERA investigation.
2 - Avian Predator TRVs summarized in Table 2.2-1.
3 - 95% upper confidence limit of reference tissue data from regional ERA investigations.
4 - TEV selected as greater of the TRV or RSV.

Table 2.2-3. Summary of Threshold Effects Value Hazard Quotients and identification of "Limiting" CoCs for the avian predator exposure pathway in the case study area[1]

Class	CoC	Aquatic Pathway[3]	DSY-24-IBM	DSY-25-IBM	DSY-25-LOB	DSY-26-CN	DSY-26-DM	DSY-26-IBM	DSY-27-IBM	DSY-27-LOB	DSY-28-CN	DSY-28-DM	DSY-28-IBM	DSY-28-LOB	DSY-29-CN	DSY-29-DM	DSY-29-LOB	DSY-31-DM	DSY-31-PM	DSY-32-PM	DSY-33-DM	DSY-33-LOB	DSY-33-PM
MET	Arsenic	AVIAN	0.13	0.16	0.36	0.05	0.07	0.10	0.08	0.21	0.06	0.21	0.03		0.10	0.11	0.36	0.06	0.12	0.06	0.16	0.28	0.07
MET	Cadmium	AVIAN	0.10	0.07	0.02	0.05	0.05	0.04	0.04	0.01	0.06	0.04	0.03		0.06	0.05	0.03	0.03	0.04	0.03	0.05	9E-03	0.04
MET	Chromium	AVIAN	0.19	0.18	0.10	0.08	0.13	0.15	0.17	0.13	0.06	0.14	0.15		0.07	0.12	0.10	0.14	0.12	0.10	0.15	0.12	0.11
MET	Copper	AVIAN	7E-03	0.02	0.25	0.03	0.02	0.01	0.03	0.28	0.03	0.02	2E-03		0.04	0.01	0.17	0.01	0.02	0.02	0.01	0.10	0.01
MET	Lead	AVIAN	0.19	1E-05	5E-03	0.03	0.04	1E-05	0.10	2E-03	1E-05	1E-05	1E-05		0.03	0.03	7E-03	0.04	0.06	0.09	0.05	0.02	1E-05
MET	Mercury	AVIAN	0.07	0.05	0.07	0.03	0.04	0.03	0.04	0.12	0.03	0.04	0.04		0.04	0.05	0.08	0.04	0.04	0.04	0.03	0.06	0.03
MET	Silver	AVIAN	9E-06	9E-06	0.50	0.05	9E-06	9E-06	9E-06	0.63	0.03	0.11	9E-06		9E-06	9E-06	0.53	9E-06	9E-06	0.13	9E-06	0.31	9E-06
MET	Zinc	AVIAN	0.27	0.40	0.31	0.15	0.36	0.32	0.50	0.40	0.09	0.34	0.43		0.10	0.27	0.46	0.58	0.31	0.40	0.43	0.37	0.36
PCB	Total PCBs	AVIAN	0.22	0.19	0.17	0.78	0.63	0.30	0.41	0.27	1.38	0.25	0.29	0.31	1.13	0.31	0.13	0.28	0.34	0.13	0.23	0.17	0.19
	SUM TEV-HQ[2]		1.18	1.06	1.78	1.24	1.36	0.95	1.37	2.05	1.73	1.14	0.97	0.31	1.57	0.94	1.86	1.19	1.04	1.00	1.11	1.44	0.81
	MAX TEV-HQ[4]		0.27	0.40	0.50	0.78	0.63		0.50	0.63	1.38	0.34			1.13		0.53	0.58	0.34	0.40	0.43	0.37	
	MAX CoC[4]		Zinc	Zinc	Silver	Total PCBs	Total PCBs		Zinc	Silver	Total PCBs	Zinc			Total PCBs		Silver	Zinc	Total PCBs	Zinc	Zinc	Zinc	
	Limiting CoC[5]		Zinc		Silver	Total PCBs				Silver	Total PCBs				Total PCBs			Zinc		Zinc	Zinc		

1- HQ_{TEV} = CoC concentration/TEV; see Table 2.2-2.
2- SUM HQ_{TEV} = sum HQs_{TEV} for all CoCs for which TEVs were developed.
3- AVIAN = Avian predator exposure pathway, CoCs measured in prey tissue.
4- MAX CoC associated with maximum observed HQ_{TEV} by sample.
5- Limiting CoC associated with maximum observed HQ_{TEV} by station.
IBM = indigenous blue mussel
LOB = lobster
CN = cunner
DM = deployed blue mussel
PM = *Pitar morrhuana*
MM = *Mercenaria mercenaria*

Table 2.2-3, con't.

Class	CoC	Aquatic Pathway[3]	DSY-34-PM	DSY-35-IBM	DSY-35-LOB	DSY-35-MM	DSY-35-PM	DSY-36-CN	DSY-36-IBM	DSY-36-LOB	DSY-36-PM	DSY-37-PM	DSY-38-DM	DSY-38-LOB	DSY-38-PM	DSY-39-DM	DSY-39-LOB	DSY-40-DM	DSY-40-IBM	DSY-41-MM	DSY-41-PM
MET	Arsenic	AVIAN	0.11	0.08	0.20	0.08	0.11	0.04	0.08	0.25	0.09	0.09	0.08	0.33	0.08	0.11	0.23	0.07	0.07	0.09	0.03
MET	Cadmium	AVIAN	0.04	0.04	0.03	0.04	0.04	0.05	0.02	2E-05	0.05	0.04	0.03	0.03	0.03	0.02	0.02	0.03	0.04	0.05	0.04
MET	Chromium	AVIAN	0.13	0.13	0.13	0.14	0.12	0.05	0.17	0.12	0.15	0.10	0.16	0.12	0.11	0.18	0.10	0.16	0.13	0.10	0.12
MET	Copper	AVIAN	0.02	0.01	0.22	0.01	0.01	0.03	0.01	0.08	0.02	0.02	0.02	0.28	0.02	0.01	0.33	0.01	0.01	0.02	0.02
MET	Lead	AVIAN	0.05	0.06	0.02	1E-05	0.05	0.03	1E-05	0.01	0.07	0.10	0.06	9E-03	1E-05	1E-05	6E-03	0.10	0.08	0.05	1E-05
MET	Mercury	AVIAN	0.03	0.04	0.07	0.03	0.03	0.04	0.05	0.09	0.04	0.04	0.03	0.09	0.04	0.04	0.11	0.03	0.04	0.03	0.04
MET	Silver	AVIAN	9E-06	9E-06	0.60	9E-06	9E-06	9E-06	9E-06	0.26	9E-06	0.06	0.16	0.56	9E-06	0.09	0.08	9E-06	9E-06	0.12	9E-06
MET	Zinc	AVIAN	0.33	0.46	0.39	0.39	0.44	0.19	0.30	0.41	0.43	0.38	0.32	0.61	0.47	0.46	0.46	0.41	0.37	0.23	0.30
PCB	Total PCBs	AVIAN	0.11	0.25	0.11	0.06	0.17	1.11	0.32	0.30	0.18	0.15	0.20	0.10		0.16	0.23	0.20	0.32	0.09	0.10
	SUM TEV-HQ[2]		0.82	1.07	1.78	0.75	0.97	1.56	0.95	1.52	1.02	0.97	1.05	2.11	0.75	1.08	1.55	1.02	1.07	0.79	0.64
	MAX TEV-HQ[4]			0.46	0.60			1.11		0.41	0.43		0.32	0.61		0.46	0.46	0.41	0.37		
	MAX CoC[4]			Zinc	Silver			Total PCBs		Zinc	Zinc		Zinc	Zinc		Zinc	Zinc	Zinc	Zinc		
	Limiting CoC[5]				Silver			Total PCBs						Zinc		Zinc		Zinc			

HQ_{TEV} = CoC concentration/TEV; see Table 2.2-2.
SUM HQ_{TEV} = sum HQs_{TEV} for all CoCs for which TEVs were developed.
AVIAN = Avian predator exposure pathway, CoCs measured in prey tissue.
MAX CoC associated with maximum observed HQ_{TEV} by sample.
Limiting CoC associated with maximum observed HQ_{TEV} by station.
IBM = indigenous blue mussel
LOB = lobster
CN = cunner
DM = deployed blue mussel
PM = *Pitar morrhuana*
MM = *Mercenaria mercenaria*

Table 2.3-1. Summary of Exposure Parameter Values used in estimating exposures via shellfish consumption by recreational fishermen.

Parameter	Value to Estimate RME	Rationale	Reference
Global variables			
Body Weight (kg)			
- Shellfishing	59	Weighted averages of children and adults assuming 9 and 30 year exposures.	USEPA 1994
Exposure Duration (yr)			
- Shellfishing and Residential (yr)	30	Median and upper-bound time at one residence, adults.	USEPA 1994
Averaging Time (days)			
- Cancer risks	25,550	Based on 70 year life expectancy.	USEPA 1989b
- Noncancer risks			
Shellfishing and Residential	10,950	Based on exposure duration.	USEPA 1989b
Relative Absorption Factors			
- Ingestion of shellfish			USEPA 1989b
VOCs	1		
PAHs	1		
PCBs	1		
Pesticides	0.3 or 1	For CoCs with high and low sorption to soil, respectively.	
Inorganics	1		
Lead	0.3 or 0.5	For adults and children, respectively.	
Consumption of Locally-Caught Shellfish Scenario			
Exposure Frequency (day/yr)	350	Default value to use with daily average consumption rates.	USEPA 1994
Ingestion Rate (g/day)	15.6	Values for New England total clam and oyster consumption.	USEPA 1994; Rupp *et al* . (1980)
Fraction of Ingested Shellfish Caught Locally	1	Conservative assumption in absence of site-specific data.	BPJ

RME: Reasonable Maximum Exposure
AE: Average Exposure
BPJ: Best professional judgment

Table 2.3-2. Summary of Risk Based Values used for the calculation of risks for the human health exposure pathway in the case study area.

Class	Analyte[2]	**RME Benchmark[1] (mg CoC/kg wet tissue wt.)**		**Risk-based Value[4] (mg CoC/kg dry tissue wt.)**
		Cancer Risk[3]	Non-cancer Risk[3,5]	
MET	Arsenic	7.3E-03	1.40	0.05
PAH	Benzo(a)anthracene (H)	1.5E-02		0.11
PAH	Benzo(a)pyrene (H)	1.5E-03		0.01
PAH	Dibenz(a,h)anthracene (H)	1.5E-03		0.01
PAH	Indeno(1,2,3-cd)pyrene (H)	1.5E-02		0.11
PCB	Total PCBs	0.18		1.30

RME = Reasonable Maximum Exposure

RBV = Risk Based Value

1 - Benchmarks calculated for CoCs with > 1×10^{-6} cancer risk or HQ>1.0 non-cancer risk for HH ERA under RME exposure scenario.

2 - CoCs as identified in HHRA investigation

3 - CoCs and associated risks as identified in HHRA investigation

4 - Minimum of risk-based RME values for carcinogenic and non-carcinogenic CoCs; dry tissue wt. = 14% of wet tissue wt. assumed.

5 - Missing values indicates this CoC is not a non-carcinogenic CoC.

Table 2.3-3. Derivation of Threshold Effects Values for the human health exposure pathway in the case study area.

Exposure Pathway	Class	CoC[1]	Human Health RBV[2] (mg/kg dry tiss. wt)	Human Health RSV[3] (mg/kg dry tiss. wt)	Human Health TEV[4] (mg/kg dry tiss. wt)
HH	MET	Arsenic	0.05	10.65	10.65
HH	PAH	Benzo(a)anthracene (H)	0.11	0.01	0.11
HH	PAH	Benzo(a)pyrene (H)	0.01	0.005	0.011
HH	PAH	Dibenz(a,h)anthracene (H)	0.01	0.0006	0.011
HH	PAH	Indeno(1,2,3-cd)pyrene (H)	0.11	0.003	0.11
HH	PCB	Total PCBs	1.30	0.32	1.30

RBV = Risk Based Value
RSV = Reference Screening Value
TEV = Threshold Effects Value
1 - List includes analytes for which Human Health RBVs were developed; see Table 2.3-2.
2 - Human Health RBVs summarized in Table 2.3-2.
3 - RSV background data compiled for regional HHRA investigations
4 - TEV selected as greater of the RBV and RSV.

Table 3.1-1. Summary of baseline Preliminary Remediation Goals (PRGs) for aquatic, avian predator, and human health exposure pathways for the case study area.

		Baseline Preliminary Remediation Goal[1]			
Class	**Analyte[2]**	**Aquatic-Bedded[3]**	**Aquatic-Resuspended[3]**	**Avian Predator[3]**	**Human Health[3]**
MET	Arsenic		24.3		12.2
MET	Copper		66.7		
MET	Lead		78.7		
MET	Silver			18213	
MET	Zinc			269	
PAH	Benzo(a)pyrene				53.5
PAH	HMW PAHs	6203			
PCB	Total PCBs	1638	517	561.9	
PST	o,p'-DDE		9.0		

1 - Pathway-specific PRGs expressed in sediment concentration units for use during remediation:
PAHs, PCBs, pesticides: units = ng/g dry weight sediment; metals: units = µg/g dry weight sediment.
2 - List includes only limiting CoCs identified for Aquatic, Avain and Human Health Pathways.
3- See text Section 3.1 for derivation method.

Table 3.1-2. Summary of baseline and Recommended Preliminary Remediation Goals (PRGs) for aquatic, avian predator, and human health exposure for the case study area expressed as sediment concentration-based values.

		Preliminary Remediation Goal[1]							
		Aquatic-Bedded[3]		**Aquatic-Resuspended[3]**		**Avian Predator[3]**		**Human Health[3]**	
Class	**Analyte[2]**	BPRG	RPRG	BPRG	RPRG	BPRG	RPRG	BPRG	RPRG
MET	Arsenic			24.3	NR			12.2	NR
MET	Copper			66.7	NR				
MET	Lead			78.7	157.3				
MET	Silver					18213	NR		
MET	Zinc					269	NR		
PAH	Benzo(a)pyrene							53.5	535.4
PAH	HMW PAHs	6203	12407						
PCB	Total PCBs	1638	1638	517	1033	561.9	NR		
PST	o,p'-DDE			9.0	NR				

BPRG - Baseline (HQ=1) Preliminary Remediation Goal

RPRG - Recommended Preliminary Remediation Goal

NR - Not Recommended

1 - Pathway-specific PRGs expressed in sediment concentration units for use during remediation:

PAHs, PCBs, pesticides: units = ng/g dry weight sediment; metals: units = µg/g dry weight sediment.

2 - List includes only limiting CoCs identified for Aquatic, Avain and Human Health Pathways.

3- See text Section 3.1 for derivation method.